META-ANALYSIS

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Systematic Review and Meta-Analysis: Predictors of Adult Psychiatric Outcomes of Childhood Attention-Deficit/Hyperactivity Disorder

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Objective: Psychiatric disorders are highly prevalent in adults with childhood-onset attention-deficit/hyperactivity disorder (ADHD). Currently, little is known about childhood predictors for these outcomes.

Method: PubMed, PsychInfo, Web of Science, and EMBASE were searched until June 2024. Eligible studies investigated childhood predictors of persistent ADHD, substance use disorders (SUDs), conduct disorder, antisocial personality disorder, major depressive disorder (MDD), and/or anxiety disorders in adults diagnosed with childhood ADHD (PROSPERO #CRD42022320887). Meta-analytic models were tested when N $\geq$3 for a predictor with similar effect measures, otherwise predictors were discussed narratively when N $\geq$2. The Newcastle–Ottawa Scale (NOS) was used to assess study quality.

Results: The selected 36 studies included 119 predictors, with 10 predictors eligible for meta-analyses. History of stimulant treatment (odds ratio [OR] = 1.88, 95% CI = 1.28-2.75, p = .001) was associated with increased, and higher childhood IQ with decreased (OR = 0.99, 95% CI = 0.98-1.00, p =.039), risk of ADHD persistence in adulthood. ADHD persistence was associated with increased risk of SUDs (OR = 2.12, 95% CI = 1.53-3.17, p =.004) and MDD (OR = 3.19, 95% CI = 1.71-5.95, p <.001). Narratively reviewed predictors of fair/good quality studies showed potential predictors for ADHD persistence (ie, ADHD combined type, hyperactive/impulsive symptoms, anxiety disorders, externalizing problems, social dysfunctioning, and socioeconomic status).

Conclusion: We confirmed earlier reported childhood predictors (ie, stimulant treatment history, ADHD persistence) and identified potential new predictors (ie, childhood anxiety disorders, social problems, socioeconomic status) for psychiatric outcomes of ADHD. However, the available literature is hampered by methodological shortcomings. Future studies should focus on studying combined effects of potential predictors.

Plain language summary: This study systematically evaluated previous research on risk factors for adult psychiatric outcomes of children diagnosed with ADHD. Results showed that those adults who were treated with stimulant medication were twice as likely to still have ADHD in adulthood. Those adults who still had ADHD were at greater risk for also having a substance use disorder and depressive disorder.

Study registration information: Predictors of Long-term Psychiatric Outcomes of Childhood ADHD in Adulthood: a Systematic Review and Meta-Analysis; https://www.crd.york.ac.uk/PROSPERO/view/CRD42022320887

Key words: ADHD; mental disorders; comorbidity; risk factors; cohort studies

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Attention-deficit/hyperactivity disorder (ADHD) is a predominantly childhood-onset neurodevelopmental disorder characterized by a persistent pattern of hyperactive-impulsive and/or inattentive behaviors.[1] The developmental trajectory of ADHD is highly variable, ranging from full remittance to full persistence. Approximately 75% of individuals with ADHD experience remittance of their ADHD diagnosis at a certain point, but impairing symptoms of ADHD persist in the majority of the cases.[2,3] Meta-analyzed point estimations of ADHD persistence rates range between 3.3% and 25.7%, depending on the type of sample (clinical vs population-based), diagnostic classification system and diagnostic tools used, sex distribution of the sample, and geographic region.[4,5] However, in cohort studies with clinical samples, the ADHD persistence rates reported are often higher, up to 77%.[3] Although the long-term outcome of childhood ADHD is heterogeneous, many individuals show impairments due to their ADHD symptoms and the development of comorbid problems.[6]

Childhood ADHD is an early warning sign for later psychopathology, both for adults with persistent ADHD and for those with remitted childhood ADHD.[7,8] Childhood ADHD increases the risk for developing psychiatric outcomes by 2.5 times.[9] Besides ADHD persistence, the most common psychiatric adult outcomes of childhood ADHD include substance use disorders (SUDs; estimated prevalence 21%-36%), mood disorders (eg, major

depressive disorder [MDD]; estimated prevalence 21-22%), anxiety disorders (eg, generalized anxiety disorder [GAD]; estimated prevalence 23%-25%), and disorders involving antisocial behavior (eg, conduct disorder [CD] and antisocial personality disorder [ASPD], estimated prevalence 27%-34%).[1,10] These outcomes have a negative impact on global and daily functioning,[9,11] including an increased risk for long-term work disability, delinquency, negative life events, suicidality, and premature death.[12-16] Identifying predictors of prevalent psychiatric outcomes in adults with childhood ADHD is important,[11] as this may open up possibilities for early intervention, enhance outcomes, and reduce health care costs.[17]

A substantial number of studies have addressed predictors for adult psychiatric outcomes of childhood ADHD. The only available review aggregated 16 studies on predictors for ADHD (symptom) persistence and reported higher ADHD severity, receiving pharmacological treatment for ADHD, comorbid CD, and comorbid MDD to be predictive of ADHD persistence into adulthood.[18] There is currently no review or meta-analysis on predictors other than ADHD (symptom persistence) on adult psychiatric outcomes of childhood ADHD.[1,18]

The current systematic review and meta-analysis aimed to aggregate existing longitudinal studies focusing on childhood predictors for the development of the most common psychiatric outcomes in adults diagnosed with childhood ADHD, including ADHD persistence, SUDs, CD, ASPD, MDD, and anxiety disorders. We critically appraised the current state of evidence, and formulated recommendations for future research.

METHOD

Design

The systematic review and meta-analysis was conducted in agreement with Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) criteria (PRISMA checklists are outlined in Table S1 and Table S2, available online).[19] The study protocol was registered with the International Prospective Register of Systematic Reviews (PROSPERO; #CRD42022320887).

Study Selection

Studies were selected if they met the following criteria: (1) contained a sample of adults with a childhood ADHD diagnosis (according to *DSM-III*, *ICD-9*, or later versions), where childhood ADHD should have been diagnosed before the age of 18 years, or retrospectively by reliable clinical interviews; (2) investigated childhood (mean age <18 years) predictors of most prevalent (estimated prevalence ≥20%) adult (mean age ≥18 years) *DSM*- or *ICD*-based psychiatric outcomes (ie, persistent ADHD, SUDs, CD, ASPD, MDD, and/or anxiety disorders [GAD, agoraphobia, social phobia, panic disorder, and specific phobia]); (3) used a retrospective or longitudinal design, where the sample was measured at least at 2 time points, of which at least 1 measurement took place at a mean age of <18 years and at least 1 measurement at a mean age of ≥18 years; and (4) was published in a peer-reviewed journal (last search May 2024).

PubMed, PsychInfo, Web of Science, and EMBASE (including MEDLINE) were searched for relevant studies, combining search terms of ADHD, predictors, and adult psychiatric outcomes (full search strategy in Supplement 1). Reference lists were searched for additional studies. The Web app Rayyan was used to detect duplicates.[20] Two raters (NP, and either DJ, ML, SN, or JO) independently judged articles for eligibility based on titles and abstracts. Thereafter, full-text articles were checked by 2 raters for eligibility. If studies reported on overlapping samples, a hierarchical selection process was used to select one study. Preferably, the study was selected that reported on the largest number of predictors. If multiple studies had the same number of predictors, we the study with the longest follow-up interval (ie, the oldest age at assessment) was selected.

Data Extraction

The first 60% of the data extraction was done in duplicate (NP, DJ), and the remainder was done by one of the authors (NP). Extracted data included total sample size, sex distribution, age at last follow-up, ADHD subgroups and accompanying group size, attrition rates, statistical approach, reported effect sizes of the statistical model, risk factors studied, statistical controls, and psychiatric outcomes. Findings of studies that investigated predictors that assessed highly similar constructs were merged: findings were pooled for the presence/absence of the following: (1) oppositional defiant disorder (ODD) and CD, (2) parental psychiatric problems, (3) maternal psychiatric problems, (4) paternal psychiatric problems, (5) educational difficulties, and (6) family stress factors (Table S3, available online; Table S3 also includes all operationalizations of predictors found in at least 2 studies). Likewise, several outcome measures were merged, including outcomes of (1) SUDs and (2) anxiety disorders (ie, panic disorder, social anxiety disorder, separation anxiety disorder, specific phobia, selective mutism, GAD, and agoraphobia).

Study Quality

Two raters independently assessed study quality using the Newcastle–Ottawa Quality Assessment Scale (NOS) for cohort studies (Supplement 2, available online).[21]

Inconsistencies in ratings were solved by consensus and, if needed, by discussion with a third rater. Studies were classified as poor, fair, or good quality corresponding to the Agency for Health Research and Quality standard.[22]

Statistical Analyses

Multivariate meta-analysis was used to account for studies that included several effect sizes of the same predictor of the studied psychiatric outcomes. This analysis used 3 levels, which included the following: (1) primary study summary statistics (ie, all relevant effect sizes provided by 1 study); (2) nested within study (ie, 1 effect size per study); and (3) nested within 1 effect size (ie, meta-analysis of 1 effect size across all studies) to control for several effect sizes within 1 study. Robust variance estimation was used to provide valid standard errors of the point estimates and valid significance tests.[23] All multivariate meta-analyses used a random-effects model to calculate aggregated point estimates using R.[24] Where possible, effect sizes were selected that adjusted for the largest number of possible confounding variables of a study, irrespective of the type of confounder (eg, age, sex, ADHD severity) to better approximate the true effect of the specific predictor. The Higgins I^2 index was measured and the Cochran Q test was conducted to indicate heterogeneity among studies. Only cases of potential high heterogeneity were reported in the text.

If a meta-analysis was not feasible (ie, when there were fewer than 3 studies looking at a specific predictor or when effect sizes were not comparable), a narrative review was provided for those predictors of a particular psychiatric outcome included in at least 2 studies. The narrative systematic review reported terms of evidence as follows: (1) evidence for an association, if all studies found an association; (2) some evidence for an association, if a study/some studies found an association and a study/some studies did not; (3) evidence for no association, if all studies found no association. Figure 1 provides a flowchart of decisions made for our analyses.

For the narrative review, multiple effect sizes of the same predictor of the psychiatric outcomes studied were first averaged within studies to have each study contributing a single effect size per predictor to the narrative review, where possible using odds ratios (ORs) or hazard ratios (HRs). When merging was not possible (eg, for studies that did not report effect sizes), we assumed the following: evidence for no association, if all effect sizes or the majority of the effect sizes were nonsignificant; evidence for an association, if all effect sizes or the majority of the effect sizes were significant; some evidence for an association, if the effect sizes were equally divided between significant and nonsignificant.

Sensitivity analyses for the multivariate meta-analyses and narrative review were performed excluding poor-quality studies.

RESULTS

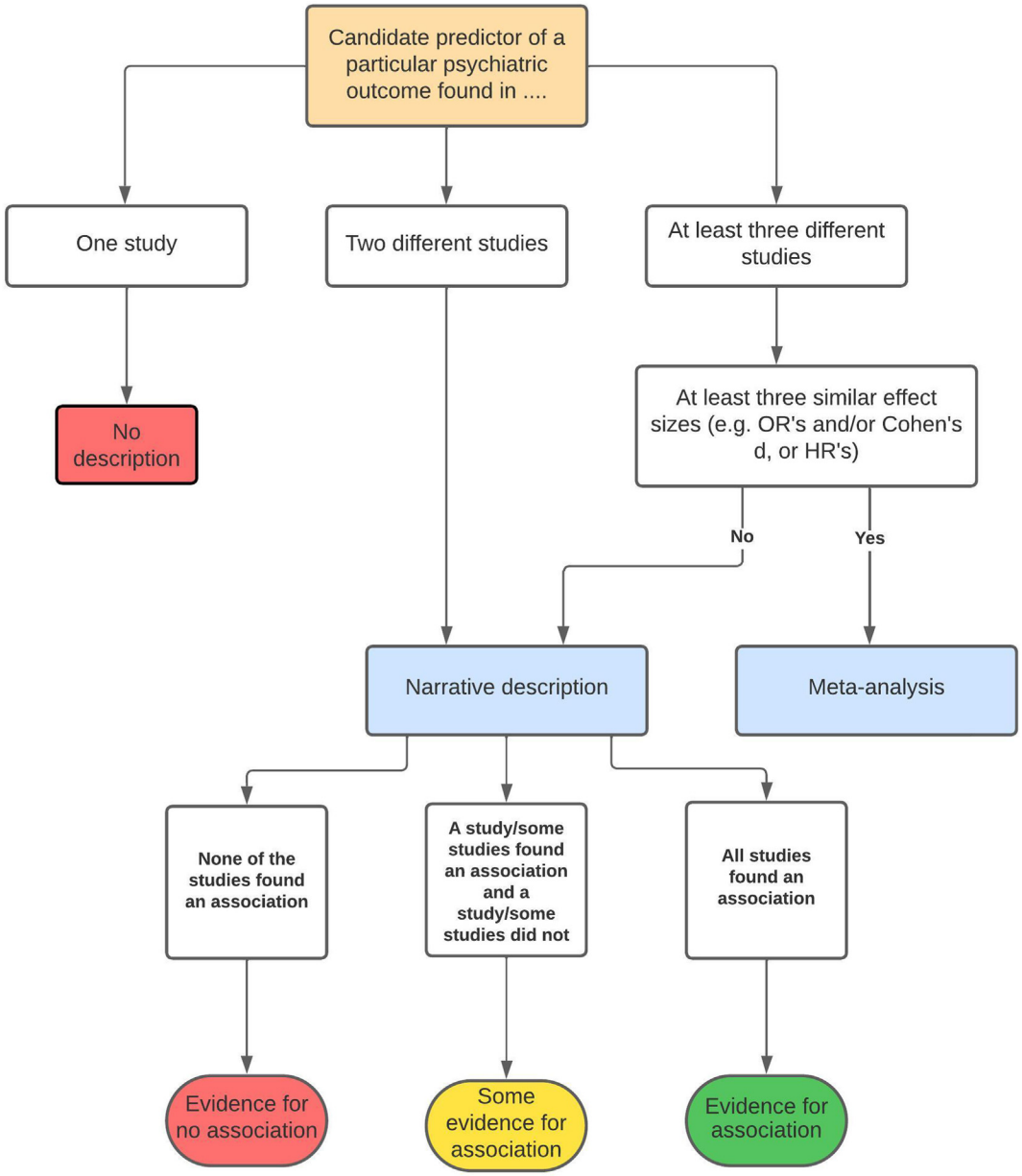
The systematic search revealed a total of 2,729 unique studies (Figure S1, available online). Of these, 2,558 studies were excluded at the title/abstract level and 133 after full-text assessment (Supplement 3). In total, 34 prospective and 2 retrospective longitudinal studies in adults with childhood ADHD were included, with 17 studies looking into ADHD persistence, 19 studies into SUDs, 4 studies into CD, 5 studies into ASPD, 8 studies into MDD, and 4 studies into anxiety disorders (Table 1[25-59]; Tables S4-S9, available online). Studies reported on several predictors for each outcome (Tables S10 and S11, available online). The available data allowed multivariate meta-analyses of 10 predictors of ADHD persistence, SUDs, MDD, and anxiety disorders (Table 2). A narrative review was provided for a subsequent 31 predictors that were studied at least twice. Of 36 studies, 16 were of fair or good study quality (Tables S12-S14, available online). Notably, 53% of studies did not control for any confounding variables.

Predictors of ADHD Persistence

Five predictors of ADHD persistence were included in the meta-analyses (Table 2). A childhood history of stimulant treatment (OR = 1.88, 95% CI 1.28-2.75, $p = .001$) was associated with an increased risk for a persistent course of ADHD,[29,32,36,37] and a higher childhood Full Scale IQ (OR = 0.99, 95% CI = 0.98-1.00, $p = .039$) was associated with a decreased risk.[7,33,37,53] Severity of childhood ADHD-related impairment was not associated with adulthood ADHD persistence (OR = 2.12, 95% CI = 0.99-4.54, $p = .054$),[29,37,44] and findings showed potentially high heterogeneity among studies (Q[2] = 10.44, $p = .005$, $I^2 = 76.9\%$). Furthermore, childhood ODD/CD (OR = 1.03, 95% CI = 0.87-1.24, $p = .564$),[7,30,32,37,44] and male sex (OR = 0.93, 95% CI = 0.71-1.23 $p = .621$)[7,32,36,37,53] were not associated with ADHD persistence. Sensitivity analyses excluding poor quality studies for predictors of adulthood ADHD persistence did not change the results for ADHD-related impairment,[29,37,44] stimulant treatment history,[29,36,37] and male sex,[36,37,53] with the single exception that the association for childhood Full-Scale IQ became nonsignificant (Table S13, available online).[33,37,53]

An additional 25 predictors for ADHD persistence were included in the narrative review (Table 2). There

FIGURE 1 Flowchart of Decisions Made for Narrative Description and Meta-Analysis



Note: *First, the meta-analytic and narrative review was described including all studies (poor/fair/good study quality). Consecutively, findings of only fair and good quality studies were discussed. HR = hazard ratio; OR = odds ratio. Please note color figures are available online.*

was some evidence for the following predictors being associated with an increased risk of ADHD persistence: ADHD combined type (compared to ADHD inattentive type),[37,44] total ADHD symptoms,[7,29,30,46,47,50,54] hyperactive/impulsive symptoms,[7,29,32,33,36,37,46] inattention symptoms,[7,29,32,33,36,37,46] anxiety disorders,[29,30,44] externalizing problems,[7,29,30,32,33,36,37,46,50,54] social dysfunctioning,[29,30,33] thought problems,[29,30] unspecified ADHD treatment,[44,46] parental psychopathology,[7,53] maternal psychopathology,[7,29,44] and low socioeconomic status.[7,29,32,33,36,53,54] Some evidence for an association with a protective effect was found for performance IQ.[7,33] Evidence for no association was found for ADHD hyperactive-impulsive type,[37,44] mood disorders,[29,30,44] internalizing problems,[7,29,30,33] childhood traumatic life events,[7,44] verbal IQ,[7,33] executive functioning,[7,50] genetic biomarkers,[28,47] birth weight,[7,36] paternal psychopathology,[29,44] educational difficulties,[29,30,46] family stress factors,[29,30,32,44,53] and age.[32,47,54]

TABLE 1 Overview of All Included Studies (N = 36 Studies)

Study	N childhood ADHD (% male)	Ethnicity/race, %	Age, y, at last follow-up; groups specified; mean, SD, range	Criteria used to diagnose ADHD in childhood	Follow-up rate, %	Mean age, y, of study sample at baseline wave and follow-up assessments	Psychiatric disorders assessed at outcome	At which assessments were psychiatric disorders measured	Method of assessment psychiatric outcomes
Agnew-Blais *et al.*, 2016[7]	247 (42)	Unknown, representative of all socioeconomic conditions of the UK[f]	18, 0, 0	*DSM-IV* diagnosis based on criteria reported by mothers and teachers	93	5, 7, 10, 12, 18	ADHD, any SUD, CD, any anxiety disorder, MDD	ADHD measured at all assessments; Others at T4	DIS
Biederman *et al.*, 2008[25a]	112 (100)	White	21, unknown	Diagnosis by psychiatrist/ pediatrician, ADHD screening telephone questionnaire and K-SADS-E diagnostic interview	80	11, 12, 15, 21	any SUD, CD, ASPD, MDD	All	K-SADS-E
Biederman *et al.*, 2008[26a]	112 (100)	White	No stimulant treatment: 23, 4, unknown Stimulant treatment: 21, 3, unknown	Diagnosis by psychiatrist/ pediatrician, ADHD screening telephone questionnaire and K-SADS-E diagnostic interview	80	No stimulant history: 12, unknown, 23 Stimulant group: 10, unknown, 21	Any SUD	All	K-SADS-E
Biederman *et al.*, 2009[27a]	217 (100)	White	No stimulant treatment: 23, 3.7, 15-30 Stimulant treatment: 21, 3, 15-30	Diagnosis by psychiatrist/ pediatrician, ADHD screening telephone questionnaire and K-SADS-E diagnostic interview	80	11, 12, 15, 21	CD, MDD	All	K-SADS-E
Biederman *et al.*, 2009[28a]	563 (59)	White	20, 14, unknown	Diagnosis by psychiatrist/ pediatrician, ADHD screening telephone questionnaire and K-SADS-E diagnostic interview	Unknown	Boys family study: 11, 15, 21 Girls family study: 11, 16 Genetic linkage study: retrospective	ADHD	All	K-SADS-E/SCID

(*continued*)

TABLE 1 Continued

Study	N childhood ADHD (% male)	Ethnicity/race, %	Age, y, at last follow-up; groups specified; mean, SD, range	Criteria used to diagnose ADHD in childhood	Follow-up rate, %	Mean age, y, of study sample at baseline wave and follow-up assessments	Psychiatric disorders assessed at outcome	At which assessments were psychiatric disorders measured	Method of assessment psychiatric outcomes
Biederman *et al.*, 2011[29a]	217 (100)	White	22, 4, 15-31	Diagnosis by psychiatrist/ pediatrician, ADHD screening telephone questionnaire and K-SADS-E diagnostic interview	80	11, 12, 15, 22	ADHD	All	K-SADS-E, SCID
Biederman *et al.*, 2012[30a]	96 (0)	Predominantly White[f]	22, 3, 15-30	Diagnosis by psychiatrist/ pediatrician, ADHD screening telephone questionnaire and K-SADS-E diagnostic interview	69	11, 16, 22	ADHD, any SUD, MDD	All	K-SADS-E
Biederman *et al.*, 2012[31a]	213 (55)	Predominantly White[f]	Nonsmoking 22, 3, unknown Smoking 23, 3, unknown	Diagnosis by psychiatrist/ pediatrician, ADHD screening telephone questionnaire and K-SADS-E diagnostic interview	Unknown	Male participants: 11, 12, 15, 21 Female participants: 11, 16, 22	Any SUD	All	K-SADS-E, SCID
Breyer *et al.*, 2014[32c]	150 (81)	Predominantly White[f]	Remitted ADHD 22, 1, unknown Persistent ADHD 22, 1, unknown	Parent assessed DICA	89	9, 11, 13, 18, 20, 22	ADHD, any SUD	All	Adolescent Diagnostic Interview
Cheung *et al.*, 2015[33]	110 (86)	White	18, 3, 11-26	PACS	63	12, 18	ADHD	All	DIVA
Clarke *et al.*, 2011[34]	38 (100)	Australian[f]	22, unknown	Clinical assessment by pediatrician and psychologist, using diagnostic criteria of *DSM-IV*	95	10, 22	ADHD	All	CAARS

(*continued*)

TABLE 1 Continued

Study	N childhood ADHD (% male)	Ethnicity/race, %	Age, y, at last follow-up; groups specified; mean, SD, range	Criteria used to diagnose ADHD in childhood	Follow-up rate, %	Mean age, y, of study sample at baseline wave and follow-up assessments	Psychiatric disorders assessed at outcome	At which assessments were psychiatric disorders measured	Method of assessment psychiatric outcomes
Dalsgaard *et al.*, 2014[35]	208 (88)	Danish[f]	31, 7, unknown	Reassessment of ADHD registry records according to *DSM-IV/ICD-10* ADHD criteria	100	8, 31	Any SUD	All	DPCR data (ICD codes)
Elkins *et al.*, 2020[36]	807 (45)	Predominantly White[f]	24, 1, unknown	Primary caregiver and child reports on the DICA-R, modified for *DSM-IV*	81	11, 14, 17, 24	ADHD	Persistence based on symptoms age 17 and/or 24 y. Any SUD at age 24 (any period between 18 and 24 y)	DICA-R, Substance Abuse Module from CIDI
Gao *et al.*, 2015[37e]	399 (82)	Han Chinese	19, 1, 18-24	Chinese translation of CDIS	72	12, 18	ADHD	All	CAADI
Halperin *et al.*, 2011[38b]	90 (87)	Unknown, childhood sample: African American 41, Hispanic 26, Mixed or other Ethnicity 11, White 22	18, 2, 16–26	Parent-rated CBCL and DISC, teacher IOWA Conners	59	9, 18	ADHD, any SUD	ADHD: both, any SUD at follow-up	K-SADS-PL
Harty *et al.*, 2013[39b]	97 (unknown, childhood sample: 89)	Unknown, childhood sample: African American 28, Hispanic 31, Mixed or other Ethnicity10, White 25	18, 2	Parent-rated CBCL and DISC, teacher IOWA Conners	57	9, 18	Any SUD	Any SUD at follow-up	K-SADS-PL
Hechtman *et al.*, 2016[40d]	476 (unknown)	Unknown, childhood sample: African American 20, Hispanic 8, White 61	25, unknown, 19-28	DISC	82	8, 9, 10, 10, 11, 14, 16, 18, 20, 22, 25	Any SUD	At 20, 22, and/or 25 y	DISC
Huss *et al.*, 2008[41]	215 (91)	Unknown	22, 5, 15-unkown	Clinical diagnosis based on *ICD-9/DSM-III-R* or *ICD-10/DSM-IV*	N/A	N/A (retrospective)	Any SUD	Present	CIDI

(*continued*)

TABLE 1 Continued

Study	N childhood ADHD (% male)	Ethnicity/race, %	Age, y, at last follow-up; groups specified; mean, SD, range	Criteria used to diagnose ADHD in childhood	Follow-up rate, %	Mean age, y, of study sample at baseline wave and follow-up assessments	Psychiatric disorders assessed at outcome	At which assessments were psychiatric disorders measured	Method of assessment psychiatric outcomes
Ilbegi *et al.*, 2018[42]	189 (57)	White	21, unknown	CPRS-R:L, CTRS-R:L	Unknown	11, unknown, 21	Any SUD	All	Drug Abuse Screening Test—20; Fagerström Test for Nicotine Dependence; Short Michigan Alcohol Screening Test. Best-estimate diagnosis of SUD was considered present if either alcohol or drug use disorder according to the *DSM-IV* criteria was present
Ivanov *et al.*, 2018[43b]	40 (93)	African American 23, Hispanic 43, Mixed or other Ethnicity 8, White 28	25, 2, unknown	Parent-rated CBCL and DISC, teacher IOWA Conners	36	9, 18, 24	ASPD	All	SCID-II
Kessler *et al.*, 2005[44]	3,197 (59)	Black 10, Hispanic 11, Mixed or other Ethnicity 4, White 75	Unknown, 18-44	NCR-S assessment based on DIS for *DSM-IV*	N/A	N/A (retrospective)	ADHD	Retrospectively and present	Clinician-assessed
Lambert *et al.*, 1998[45]	214 (85)	Unknown, childhood sample: representative of East Bay Region of San Francisco metropolitan area, with members of minority ethnic groups 23[f]	Unknown	CAAS	81	Evaluated prospectively from kindergarten to fifth grade to end of high school, and as young adults	Any SUD	Adult follow-up	QDIS-III-R, CSBS

(*continued*)

TABLE 1 Continued

Study	N childhood ADHD (% male)	Ethnicity/race, %	Age, y, at last follow-up; groups specified; mean, SD, range	Criteria used to diagnose ADHD in childhood	Follow-up rate, %	Mean age, y, of study sample at baseline wave and follow-up assessments	Psychiatric disorders assessed at outcome	At which assessments were psychiatric disorders measured	Method of assessment psychiatric outcomes
Lecendreux *et al.*, 2019[46]	492 (50)	Representative of the French population[f]	18, unknown	K-SADS-E updated for *DSM-5* diagnosis	49	9, 13, 18	ADHD	All	K-SADS-E
Li *et al.*, 2013[47e]	193 (82)	Han Chinese	19, 1, unknown	CDIS	75	12, 19	ADHD	All	CAADI
Miller *et al.*, 2008[48b]	96 (87)	African American 23, Hispanic 33, Mixed or other Ethnicity 20 White 24	18, 2, unknown	Parent-rated CBCL and DISC, teacher IOWA Conners	Unknown	9, 18	ASPD	CD, OD at baseline, ASPD at follow-up	SCID-II
Molina *et al.*, 2023[49d]	579 (80)	Unknown, childhood sample: African American 20, Hispanic 8, White 61	25, 1, unknown	DISC	81	11, 12, 15, 17, 19, 21, 23, 25	Any SUD	Unknown	DISC
Øie *et al.*, 2023[50]	19 (100)	White	37, 2, unknown	Semi-structured clinical interviews and standardized rating scale with mental health professionals (*DSM-III-R*)	95	14, 27, 37	ADHD	All	MINI
Qian *et al.*, 2016[51e]	68 (82)	Han Chinese	Unknown, 18-24	*DSM-IV* criteria based on semi-structured parent and child interviews	76	13, unknown	ADHD	All	CAADI
Rasmussen *et al.*, 2000[52]	50 (76)	Swedish[f]	22, 0, unknown	ADHD *DSM-IV* checklist (parent interview)	89	6, 22	ASPD, MDD	All	Child and adolescent psychiatrists according to operationalized criteria

(*continued*)

TABLE 1 Continued

Study	N childhood ADHD (% male)	Ethnicity/race, %	Age, y, at last follow-up; groups specified; mean, SD, range	Criteria used to diagnose ADHD in childhood	Follow-up rate, %	Mean age, y, of study sample at baseline wave and follow-up assessments	Psychiatric disorders assessed at outcome	At which assessments were psychiatric disorders measured	Method of assessment psychiatric outcomes
Roy *et al.*, 2016[53d]	453 (78)	African American 19, Asian 1, Black Hispanic 1, Mixed or other Ethnicity 8, non-Black Hispanic 7 White 63	25, unknown	DISC	78	8, 21, 23, 25	ADHD	All	CAARS (*DSM-5* symptoms)
Shaw et al., 2013[54]	92 (58)	Unknown	24, 4, unknown	DICA	55	11, 24	ADHD, any anxiety disorder, MDD	All	CAARS, SCID
Steinhausen *et al.*, 2014[55]	2,0742 (75)	Danish[f]	Mean age at time of first diagnosis SUD 25	DPCR *ICD* codes	N/A	Mean age at ADHD diagnosis 15, mean age at time of first diagnosis SUD 25	Any SUD	All	DPCR data (*ICD* codes)
Tsai *et al.*, 2019[56]	214 (66)	Taiwanese	25, 5, 18-36	K-SADS-E	Unknown	12, 24	CD, MDD, any SUD, any anxiety disorder	Both or retrospectively	K-SADS-E
Wilens *et al.*, 2011[57a]	257 (52)	White	20, 4, unknown	Diagnosis by psychiatrist/ pediatrician, ADHD screening telephone questionnaire, and K-SADS-E diagnostic interview	Female participants: 69 Male participants: 80	Male participants: 11, 12, 15, 21 Female participants: 11, 16, 22	Any SUD	All	K-SADS-E
Winters *et al.*, 2011[58,e]	149 (81)	Predomonantly White[f]	22, 1, 22-24	DICA-R, modified to include all *DSM-III-R* symptoms	73	7-9, 11-15, 12-16, 18, 20, 22	Any SUD	Any SUD T4-T6	Updated structure of Adolescent Diagnostic Interview

(*continued*)

TABLE 1 Continued

Study	N childhood ADHD (% male)	Ethnicity/race, %	Age, y, at last follow-up; groups specified; mean, SD, range	Criteria used to diagnose ADHD in childhood	Follow-up rate, %	Mean age, y, of study sample at baseline wave and follow-up assessments	Psychiatric disorders assessed at outcome	At which assessments were psychiatric disorders measured	Method of assessment psychiatric outcomes
Yoshimasu *et al.*, 2018[59]	232 (72)	White	30, unknown	School/medical records included *DSM-IV* criteria for ADHD, positive ADHD questionnaire results, and/or clinical diagnosis of ADHD documented	66	Birth cohort, 30	Any SUD, ASPD, MDD, any anxiety disorder	All	MINI

Note: *ADHD = attention-deficit/hyperactive disorder; ASPD = antisocial personality disorder; BASC = Behavior Assessment System for Children; BRIEF = Behavior Rating Inventory of Executive Function; CAADI = Chinese version of the Conner's Adult ADHD Diagnostic Interview; CAADID = Conners Adult ADHD Diagnostic Interview for DSM-IV; CAARS = Conners Adult ADHD Rating Scales; CAAS = Children's Attention and Adjustment Survey; CBCL = Child Behavior Checklist; CD = conduct disorder; CDIS = Clinical Diagnostic Interview Scale; CPRS = Conners Parent Rating Scale; CSBS = California Smoking Baseline Survey; CTRS = Conners Teacher Rating Scale; DCD = developmental coordination disorder; DICA = Diagnostic Interview for Children and Adolescents; DIS = Diagnostic Interview Schedule; DISC = Diagnostic Interview Schedule for Children; DIVA = Diagnostic Interview for ADHD in Adults; DPCR = Danish Psychiatric Central Register; DSM = Diagnostic and Statistical Manual of Mental Disorders; GAF = Global Assessment of Functioning; ICD = International Classification of Diseases and Related Health Problems; IMAGE = International Multicenter ADHD Genetic; IOWA Conners = Inattention/Overactivity with Aggression Rating Scale; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia; MDD = major depressive disorder; MINI = Mini-International Neuropsychiatric Interview; MNCEP = Minnesota Competence Enhancement Program; MTA = Multimodal Treatment Study of ADHD; NCS-R = National Comorbidity Survey—Replication; ODD = oppositional defiant disorder; PACS = Parental Account of Childhood Symptoms; QDIS = Quick Diagnostic Interview Schedule; SCID = Structured Clinical Interview for DSM-IV Axis I Disorders; SES = socioeconomic status; SNAP = Swanson, Nolan, and Pelham Rating Scale; SUD = substance use disorder; WAIS = Wechsler Adult Intelligence Scale; WISC = Wechsler Intelligence Scale for Children; WRAT = Wide Range Achievement Test.*

[a,b,c,d]Studies are part of the same cohort.

[e]Studies possibly have overlapping cohorts.

[f]Exact ethnic/racial composition was not reported in the original study; therefore, data on the ethnic/race composition were not available.

TABLE 2 Meta-Analytic Results of Childhood Predictors of Attention-Deficit/hyperactive disorder (ADHD) Persistence, Substance Use Disorders (SUDs), Major Depressive Disorder (MDD), and Anxiety Disorders

	K	n	OR	95% CI	*p*	I^2 (%)	Q(df), *p*
Outcome ADHD persistence							
ADHD-related impairment	3	3	2.12	0.99-4.54	.054	76.9	10.44 (2), $p = .005$
ODD/CD classification	5	9	1.03	0.87-1.24	.564	0	5.53 (2), $p = .700$
Stimulant treatment history	4	4	1.88	1.28-2.75	.001	30.4	4.89 (2), $p = .180$
Full Scale IQ	4	4	0.99	0.98-1.00	.039	7.5	7.14 (2), $p = .068$
Male sex	5	5	0.93	0.71-1.23	.621	16.1	6.35 (2), $p = .174$
Outcome SUDs							
ADHD persistence	6	11	2.21	1.53-3.17	.004	59.7	25.84 (2), $p = .004$
Stimulant treatment history	4	19	1.22	0.70-2.12	.322	18.5	8.40 (2), $p = .972$
ODD/CD classification (HR)	3	5	2.09	0.60-7.30	.104	80.7	7.07 (2), $p = .132$
Outcome MDD							
ADHD persistence	4	4	3.19	1.71-5.95	<.001	33.9	3.79 (2), $p = .285$
Outcome anxiety disorders							
ADHD persistence	3	4	3.88	0.24-62.42	.165	73.1	6.43 (2), $p = .092$

Note: *CD = conduct disorder; HR = hazard ratio; I^2 = heterogeneity index; k = number of studies; n = number of effect sizes; ODD = oppositional defiant disorder; OR = odds ratio.*

Sensitivity analyses excluding poor quality studies (10 of 17 studies) showed some evidence for the following predictors being associated with an increased risk of ADHD persistence: ADHD combined type,[37,44] hyperactive/impulsive symptoms,[29,33,36,37] anxiety disorders,[29,30] externalizing problems,[29,33,36,37,50] social dysfunctioning,[29,33] and low socioeconomic status.[29,33,36,53] There was evidence for no association for ADHD hyperactive-impulsive type,[37,44] inattention symptoms,[29,33,36,37] mood disorders,[29,44] internalizing problems,[29,33] paternal psychopathology,[29,44] maternal psychopathology,[29,44] and family stress factors.[29,44,53] Too few studies with fair/good quality were left for a narrative review of the remaining predictors.

Predictors of SUDs

Three predictors were included in the meta-analysis for SUDs in adulthood (Table 2). ADHD persistence was associated with an increased risk of SUDs in adulthood (OR = 2.20, 95% CI = 1.26-3.85, $p = .018$),[7,30,32,57,59] whereas stimulant treatment (OR = 1.41, 95% CI = 0.72-2.75, $p = .142$)[45,56,57] and ODD/CD (HR = 2.09, 95% CI = 0.60-7.30, $p = .104$)[35,55,57] in childhood were not. Findings for ADHD persistence showed moderate-to-high heterogeneity (Q[2] = 25.84, $p = .004$, I^2 = 59.7%). After exclusion of poor quality studies, too few effect sizes were available for a meta-analysis.

In addition, 3 childhood predictors were included in the narrative review (Table 3). There was some evidence for later age at initiation of stimulant treatment being associated with an increased risk of SUDs in adulthood.[26,35] For ADHD combined type[56,57] and stimulant treatment duration,[26,35,49] there was evidence for no association.

Sensitivity analyses excluding poor quality studies showed that there was evidence for no association for stimulant treatment duration.[26,49] Too few studies were left for a narrative review of the remaining predictors.

Predictors of CD

No predictors of CD were eligible for meta-analysis. Narratively, there was some evidence that childhood stimulant treatment history was associated with an increased risk of adult CD (Table 3).[27,56] Too few studies were left for narrative review after exclusion of the poor quality studies.

Predictors of ASPD

No predictors of ASPD were eligible for meta-analysis. One predictor was included in the narrative review, showing that persistent ADHD was associated with an increased risk of adult ASPD (Table 3).[38,59] However, this result was based on 2 poor quality studies.

Predictors of MDD

One predictor of MDD was included in the meta-analysis (Table 2), showing ADHD persistence to be associated with an increased risk of MDD in adulthood (OR = 3.19, 95% CI = 1.71-5.95, $p < .001$).[7,30,54,59] After exclusion of poor quality studies, too few effect sizes were available for a meta-analysis.

TABLE 3 Predictors of Adult Psychiatric Outcomes of Childhood Attention-Deficit/Hyperactive Disorder (ADHD) Studied in Longitudinal Research for Which a Narrative Review was Possible (N > 1)

	Outcomes (no. of significant findings for a predictor / no. of times predictor studied)					
Predictors	**ADHD persistence**	**SUDs**	**CD**	**ASPD**	**MDD**	**Anxiety disorders**
ADHD						
Persistent childhood ADHD		**4/7**[a,e]		**2/2**	**2/4**[a]	1/3[a]
ADHD combined type	**1/2**	0/2				
ADHD hyperactive/impulsive type	0/2					
(No. of) inattention symptoms	**1/7**[c]					
(No. of) hyperactive/impulsive symptoms	**2.5/7**[e]					
Total no. of ADHD symptoms	**3.5/7**[e]					
Severity of ADHD-related impairment	2.5/4[a,e]					
Psychopathology (classifications)						
Mood disorders	0/3[c]					
Anxiety disorders	**1/3**					
ODD/CD	1.5/8[a,d]	1/4[a]				
Behavior and emotional problems						
Internalizing problems score	0/4[c]					
Externalizing problems score	**2/10**[c,d]					
Social dysfunctioning score	**1/3**					
Thought problems score	**1/2**					
Childhood traumatic life events (yes/no)	0/2					
Treatment history						
Stimulant treatment history (yes/no)	**3/6**[a]	0/8[a]	**1/2**		**1/2**[b]	
Age at initiation of stimulant treatment		**1/2**				
Stimulant treatment duration (y)		0/3				
Unspecified treatment	**1/2**					
Cognitive measures						
Full Scale IQ score	**2/9**[a]					
Performance IQ score	**1/2**[b]					
Verbal IQ score	0/2					
Executive functioning score	0/2					
Biomarkers						
Genetic biomarker	0/2					
Perinatal factors						
Birth weight	0/2					
Parental factors	**1/2**					
Parental psychopathology (yes/no)	**1/2**					
Paternal psychopathology (yes/no)	0/2					
Maternal psychopathology (yes/no)	**1/3**					

(continued)

One additional predictor was included in the narrative review and showed some evidence for childhood stimulant treatment history being associated with a decreased risk of adult MDD (Table 3).[27,56] Excluding poor quality studies, too few studies were left for narrative review.

Predictors of Anxiety Disorders

One candidate predictor of anxiety disorders was included in the meta-analysis and showed that ADHD persistence was not associated with an increased risk of anxiety disorders in adulthood (OR = 3.88, 95% CI = 0.24-62.42,

TABLE 3 Continued

Predictors	Outcomes (no. of significant findings for a predictor / no. of times predictor studied)					
	ADHD persistence	**SUDs**	**CD**	**ASPD**	**MDD**	**Anxiety disorders**
Educational factors						
Educational difficulties (yes/no)	0/3[c]					
Family factors						
Family stress factors	0/5[c]					
Demographics						
Sex	2/7[a]					
Age	0/3					
SES	**3/7**					

Note: *Table does not show single predictors investigated by only 1 study (see Table S10, available online). Boldface type indicates that (some evidence for) a possible positive association was found, except for 2 predictors for which a (possible) negative association was found, see footnotes. ASPD = antisocial personality disorder; CD = conduct disorder; IQ = intelligence quotient; MDD = major depressive disorder; ODD = oppositional defiant disorder; SES = socioeconomic status; SUDs = substance use disorders.*

[a]*Included in our meta-analysis.*

[b]*Boldface type indicates that (some evidence for) a possible positive association was found, except for 2 predictors for which a (possible) negative association was found.*

[c]*Study was treated as reporting no association, as both/all measures of the same predictor were nonsignificant, and averaging of effect sizes was not possible.*

[d]*Study was treated as reporting an association, as both/all measures of the same predictor were significant, and averaging of effect sizes was not possible.*

[e]*Study included 2 measures of the same predictor, of which one was significant and the other was not; averaging of effect sizes was not possible.*

$p = .165$).[7,54,59] No meta-analysis was possible on the studies that remained after exclusion of poor quality studies. There were too few studies to perform a narrative review.

DISCUSSION

This meta-analysis and systematic review is the first to aggregate results on a large variety of childhood assessed predictors of adult psychiatric outcomes of childhood ADHD, including ADHD persistence, SUDs, CD, ASPD, MDD, and anxiety disorders. We identified 36 studies, including 119 unique predictors. Most outcomes were considered in few studies, and 66% of all predictors were studied only once. The available data allowed meta-analyses of 10 predictors, and 31 predictors were narratively reviewed. Only 44% of all included studies were of fair/good study quality. Our meta-analytic results demonstrated that a history of childhood stimulant treatment was associated with an increased risk for ADHD persistence into adulthood, whereas higher IQ was associated with a decreased risk. ADHD persistence was not associated with childhood assessed ADHD-related impairment, ODD/CD, or sex. Although ADHD persistence was associated with an increased risk of SUDs and MDD in adulthood, ADHD persistence was not associated with an increased risk for adulthood anxiety disorders. Adulthood SUDs were not predicted by childhood ODD/CD or stimulant treatment. Sensitivity analysis excluding poor quality studies replicated most meta-analytic findings for ADHD persistence as outcome, showing an association with stimulant treatment history, and no association with ADHD-related impairment and sex, with the single exception that childhood IQ no longer was associated with an increased risk for ADHD persistence.

Limiting the narrative review to fair/good quality studies, our narrative results revealed 7 additional potential childhood predictors (showing some evidence) of ADHD persistence, including ADHD combined type, hyperactive/impulsive symptoms, anxiety disorders, externalizing problems, social dysfunctioning, and socioeconomic status. Furthermore, fair/good studies showed no evidence that adult ADHD persistence was predicted by childhood ADHD hyperactive-impulsive type, inattention symptoms, mood disorders, internalizing problems, paternal psychopathology, maternal psychopathology, and family stress factors; and no evidence that SUDs in adulthood were predicted by childhood stimulant treatment duration. However, there was large heterogeneity in our findings across studies. Also, some studies provided several effect sizes per potential predictor, which we dealt with by averaging effect sizes to obtain a single conclusion. However, this approach was limited by the assumption of equal weight of each effect size. Therefore, it is important to interpret the narrative results with caution.

ADHD Persistence Into Adulthood

The meta-analytic results for adult ADHD persistence in relation to childhood stimulant treatment history and sex confirm the findings of an earlier meta-analysis by Caye *et al.*,[18] except that we found an association for childhood IQ and no association for childhood ODD/CD and severity of ADHD-related impairment. A possible explanation for the discrepancy in the findings on ODD/CD is that our systematic review included only clinical samples with an ADHD diagnosis and looked at a persistent diagnosis of ADHD, whereas the meta-analysis of Caye *et al.*[18] also included (subclinical) samples of hyperactive children and included persistent ADHD symptoms as outcome. It is possible that ODD/CD is predictive of ADHD symptom persistence, but not of a diagnosis of ADHD in clinical samples. Furthermore, the results regarding impairment were not significant, despite all individual studies reporting significant odds ratios. Substantial heterogeneity between effect sizes likely contributed to our finding, possibly due to different operationalizations of impairment between studies and differences between studies in the adjustment for possible confounding variables. More specifically, 1 study adjusted for age and residence,[37] whereas the other 2 studies did not provide adjusted effect sizes,[29,37] which might introduce additional heterogeneity. With regard to our surprising finding on childhood IQ, which reached significance in predicting adult persistent ADHD, the effect was very small, with the confidence interval including a rounded 1. Moreover, IQ was no longer significant after removing 1 poor quality study,[18] indicating that this finding may not have been very robust and may warrant further investigation. Furthermore, our meta-analysis showed that a childhood history of stimulant treatment was associated with a doubled risk for a persistent course of ADHD in adulthood. Although stimulant treatment has often been reported as a protective factor for a range of ADHD-related outcomes,[60] it is also related to higher symptom severity and level of impairment during time of prescription, suggesting that the most affected children may receive stimulant treatment, which potentially increases the relationship between childhood stimulant treatment and adulthood ADHD persistence.[61] Importantly, studies included in our meta-analyses on stimulant treatment did not control for severity of impairment, stressing the need to look at combined effects of predictors in future studies.

The narrative results of fair/good quality studies hint toward the relevance of childhood symptom severity (symptoms of ADHD, anxiety, and externalizing problems) in predicting ADHD persistence in adulthood, corresponding to results from a previous narrative review.[62] In addition, (low) childhood SES might be a relevant factor. In an earlier meta-analysis, SES was not associated with an increased risk for ADHD persistence[18]; however, in that study, the investigators disregarded study quality and included 1 poor quality study without an effect (out of 3 studies). Furthermore, participants with lower SES might be more often lost to follow-up,[63] and as low SES increases risk for receiving an ADHD diagnosis in childhood,[64] SES should be further studied as a potential predictor.

Our narrative review of fair/good quality studies showed no specific evidence of childhood maternal or paternal psychopathology as a predictive factor, but 1 good quality study did hint toward the predictive value of parental psychopathology as a risk factor for ADHD persistence. In addition, we found a predictive effect of maternal psychopathology driven by a poor quality study, also hinting toward potential relevance of parental psychopathology. Similarly, the earlier meta-analysis[18] remained inconclusive on the role of parental psychopathology for ADHD persistence. Parental psychopathology is an umbrella term for several psychiatric problems and is thought to act as a risk factor for an ADHD diagnosis in childhood.[65] Our results suggest that although it may influence ADHD in childhood, it may not be associated with ADHD persistence into adulthood. Potentially, only specific aspects of parental psychopathology may have an impact on persistence.[65] In contrast to our findings, Caye *et al.*[18] identified childhood comorbid MDD as a predictor for adult ADHD persistence.[18] However, the single study with an effect was excluded in our systematic review, as the sample overlapped[66] with another, more recent, included study (without an effect).[44]

To summarize, childhood stimulant treatment history was associated with an increased risk of adult ADHD persistence, whereas sex was not. The inconsistent findings of childhood ADHD–related impairment, ODD/CD, and IQ warrant further investigation. In addition, potential childhood predictors of adult ADHD persistence that should be further investigated, preferably by meta-analyses, include ADHD combined type, hyperactive/impulsive symptoms, anxiety disorders, externalizing problems, social dysfunctioning, thought problems, parental psychopathology, and socioeconomic status.

Psychiatric Outcomes Other Than ADHD Persistence in Adulthood

Our meta-analytic results showed that a persistent course of childhood ADHD was associated with an increased risk of SUDs and MDD in adulthood by more than 2 and 3 times, respectively, corroborating previous findings.[1] A persistent course of childhood ADHD was defined by a diagnosis of

ADHD in childhood that was subsequently confirmed in adulthood, reflecting not only childhood ADHD but also adult ADHD. Therefore, alternatively, SUDs and MDD may also be the result of the presence of adult ADHD. However, the underlying mechanism linking ADHD and SUDs or MDD is likely due to a shared neurobiological and familial/genetic underpinnings of the disorders, suggesting that a persistent course of childhood ADHD may indeed be related to an enhanced risk of SUDs and MDD in adulthood.[67,68] Furthermore, in agreement with a recent review, SUDs were not associated with a childhood history of stimulant treatment.[69] Finally, our results indicate that childhood ODD/CD was not associated with SUDs in adulthood. Despite the ongoing debate (see, for example, Groenman *et al.*[70]), our finding suggests that ODD/CD is not driving the risk for SUDs in individuals with ADHD. Narratively, too few fair/good qualities studies were left to identify potential predictors of SUDs, CD, ASPD, and MDD.

For adult anxiety disorder, we found no evidence that ADHD persistence acts as a risk factor. Our findings build upon suggestions based on the results of another review on the relationship between ADHD and anxiety disordeers,[71] suggesting that future studies should include other predictors (such as internalizing problems, trauma, childhood aggression, or difficulties making friends) when studying the development of anxiety disorders in individuals with ADHD.[72]

Clearly, the current meta-analyses were limited by the number of available studies and by the types of predictors and outcomes studied. We suggest that future studies into the psychiatric long-term outcomes of childhood ADHD take the following recommendations into account. First, studies should include a wide range of potential predictors, including genetic, prenatal, perinatal, neuroimaging, neurocognitive, environmental, psychological, and social factors, to gain a better understanding of the mechanisms that play a role in the development of adult psychiatric outcomes.[1,73-76] Second, existing studies rely on linear statistical models including single predictors that do not capture the complex (nonlinear) relations between predictors and adult psychiatric outcomes of ADHD. Machine learning might offer promising possibilities, as becomes evident in the wider medical field.[77,78] Third, studies should investigate other highly impairing, albeit less prevalent, outcomes, to capture all of the psychiatric outcomes of ADHD, including eating disorders or obsessive-compulsive disorder.[79,80] Fourth, studies should improve on the adjustment for confounders, as this was the primary reason for qualifying almost half of the studies as studies of poor quality. No studies controlled for CD, MDD, or anxiety disorders in childhood, whereas they did study the presence of these disorders in adults, thereby complicating identification of causal effects. Also, hardly any studies controlled for ADHD severity in childhood. Ideally, future studies should adjust for a standardized set of covariates, such as sex, age, and severity of ADHD-related impairment in childhood, and childhood presence of the psychiatric outcome at hand to be better able to approximate the true effect. Finally, the current meta-analysis was susceptible to ecological fallacy due to the availability of summary data instead of individual participant data. Therefore, we call for studies to share individual participant data to facilitate individual patient data meta analysis (IPDMA). Ideally, an IPDMA would generate larger sample sizes, increasing the power to detect predictors for psychiatric outcomes of ADHD with lower prevalences (eg, bipolar disorder, obsessive-compulsive disorder, schizophrenia), which were not included in the current study.

The current meta-analysis and narrative review studied predictors of adult psychiatric outcomes of childhood ADHD, including ADHD persistence, SUDs, CD, ASPD, MDD, and anxiety disorders. Our meta-analysis added to the existing evidence of some earlier reported predictors (ie, history of stimulant treatment as predictor for ADHD persistence), and was the first to meta-analytically identify additional predictors (ie, ADHD persistence as predictor for adult SUD and MDD). Narratively (including only fair/good quality studies), we identified potential new predictors including thought and social problems, anxiety disorders, and socioeconomic status for ADHD persistence. No other predictors of adult psychiatric outcomes were identified, possibly due to limitations in available literature (eg, clinical and methodological heterogeneity, poor study quality) and the statistical approaches used. Finally, we highlighted avenues for future research to deepen our understanding of childhood predictors of adult ADHD psychiatric outcomes.

CRediT authorship contribution statement

Noa E. van der Plas: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Siri D.S. Noordermeer:** Writing – review & editing, Supervision, Project administration, Methodology, Funding acquisition, Data curation, Conceptualization. **Jaap Oosterlaan:** Writing – review & editing, Supervision, Project administration, Methodology, Funding acquisition, Data curation, Conceptualization. **Marjolein Luman:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization.

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Data Sharing: The data dictionary will be made available to others upon request from the corresponding author.

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