



The prevalence of cannabis use disorder in attention-deficit hyperactivity disorder: A clinical epidemiological meta-analysis

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ABSTRACT

Studies have shown that individuals with attention-deficit hyperactivity disorder (ADHD) pose an increased risk for developing substance use disorders. Increased cannabis product accessibility and recent legislative changes have led to increased cannabis consumption, thereby increasing the risk of cannabis use disorder (CUD). The present meta-analysis explored the lifetime and current prevalence of CUD in ADHD. A systematic review was conducted using the following databases: *PubMed*, *PsycINFO* and *Web of Science*. A total of 14 articles were included and used to estimate the aggregate lifetime and current prevalence of CUD in ADHD alongside risk ratios comparing increased risk of CUD in ADHD versus control samples. Mixed and random-effects models indicated that lifetime and current prevalence rates of CUD in ADHD populations were 26.9% and 19.2%, respectively (although prediction intervals ranged from 12.4% to 48.8% and 5.5%–39.1%, respectively). Analysis of the risk ratios indicated that those with ADHD were at 2.85- and 2.91-times greater risk of a lifetime or current diagnosis of CUD, respectively, than those in the general population. Our findings support the need for additional research on the prevalence of CUD in those with ADHD, as well as the inclusion of CUD screening in the treatment of ADHD.

The worldwide prevalence of lifetime non-medical cannabis use exceeds 4% among individuals 15–64 years old (i.e., 209 million people; United Nations Office on Drugs and Crime [UNODC], 2022) with the highest rates found in higher-income countries (Peacock et al., 2018). From 2010 to 2020, the number of individuals that used cannabis in the past year increased by 23% (UNODC, 2022). This is believed to be attributed to increased global decriminalization and legalization of cannabis products, social acceptability, and reduced perceptions of harm (Harris-Lane et al., 2020; Romm et al., 2022). Cannabis use is highest among younger populations, becoming the substance of choice-replacing prevalent substances such as alcohol and tobacco (UNODC, 2022). Importantly, cannabis use is related to negative health outcomes, especially among younger populations who use cannabis frequently. Frequent cannabis use may lead to impaired cognitive performance and mental health (Kroon et al., 2021; Patel et al., 2020), lower educational attainment (Thompson et al., 2019), unemployment (Sorkhou et al., 2021), increased risk for the development of anxiety disorders (Kedzior and Laeber, 2014), psychotic disorders (e.g., schizophrenia; Patel et al., 2020), and cannabis use disorder (CUD; Sorkhou et al., 2021).

Individuals diagnosed with CUD must exhibit problematic cannabis use, occurring within a 12-month period, accompanied by impairment or distress, and at least two of 11 symptoms outlined by the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*; American Psychiatric Association [APA], 2013). Some of these symptoms include consuming more cannabis than intended, difficulty reducing cannabis use, and continued use of cannabis despite persistent or recurrent social or interpersonal problems. A recent systematic review and meta-analysis of people who used cannabis worldwide found a pooled prevalence estimate of 22% for CUD, with higher prevalence among those who use more frequently (daily or weekly) and are of younger age (Leung et al., 2020). Further risk factors include a family history of substance use and substance use disorders (SUDs; Epstein et al., 2020; Lipari and Van Horn, 2017), early onset of cannabis use (11–15 years-of-age; Richmond-Rakerd et al., 2017; Schlossarek et al., 2016), the use of more potent cannabis products with higher levels of Tetrahydrocannabinol (THC; Robinson et al., 2022; Rubin-Kahana et al., 2022), male sex (Jeffers et al., 2021; Rubin-Kahana et al., 2022), lower socioeconomic status (Jeffers et al., 2021; Lipari and Van Horn, 2017), an unstable or abusive

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home environment (Schäfer et al., 2017), comorbid mental health disorders including major depression, generalized anxiety disorder (Onaemo et al., 2021), posttraumatic stress disorder (Kroon et al., 2020), bipolar disorder (Bahji et al., 2021), schizophrenia or psychosis (Koskinen et al., 2010; Kroon et al., 2020), conduct disorder (CD; Defoe et al., 2018; Masroor et al., 2019), and attention deficit hyperactivity disorder (ADHD; Schlossarek et al., 2016).

ADHD is defined as a neurodevelopmental disorder that consists of patterns of inattention and/or hyperactivity-impulsivity that negatively impacts an individual's functioning and development (APA, 2013). Symptoms associated with inattention include difficulties paying attention, leading to negligent mistakes and distraction. Alternatively, hyperactivity-impulsivity symptoms include feelings of restlessness and excessive talking or fidgeting. ADHD prevalence varies across developmental levels with rates in childhood ranging from 2.2% to 14% (Faraone et al., 2021; Sayal et al., 2018) and 1.5%–6.8% in adulthood (Faraone et al., 2021; Song et al., 2021). Individuals with ADHD are more likely to engage in potentially harmful behaviours that are associated with substance use (Davis et al., 2015; Dekkers et al., 2020; Pollak et al., 2019; Wilens, 2007; Zulauf et al., 2014). Individuals diagnosed with ADHD are up to two times more likely to consume cannabis compared to individuals without ADHD (Hernandez and Levin, 2022; Lee et al., 2011). Furthermore, individuals with ADHD are more likely to use cannabis daily with one study showing 57% of adult daily cannabis users meeting diagnostic criteria for ADHD (Loflin et al., 2014).

There are several possible factors that may help to account for increased rates of cannabis consumption among individuals with ADHD. For example, a shared genetic liability between ADHD and cannabis use initiation may help to explain this association (Soler Artigas et al., 2020). Furthermore, for some individuals with ADHD cannabis may help self-medicate symptoms of inattention and hyperactivity as well as help relieve symptoms of anxiety and stress that frequently co-occur with ADHD (Mitchell et al., 2016; Swanson et al., 2017). Characteristics associated with ADHD such as impulsivity and sensation seeking also put some individuals at greater risk of engaging in a wide range of behaviours including the use of substances such as cannabis (Malmberg et al., 2010; Moggi et al., 2020). Lastly, individuals with ADHD have also been found to be at greater risk of co-occurring psychological disorders such as CD (Moggi et al., 2020) as well as co-occurring use of other substances (Fuller-Thomson et al., 2022; Martínez-Luna et al., 2021; McCabe et al., 2016). Co-occurring mental health and substance use issues may put some individuals with ADHD at even further risk of engaging in maladaptive cannabis use.

Importantly, three additional meta-analyses have examined the later development of CUD alongside other SUDs in individuals with a previous childhood diagnosis of ADHD. Groenman et al. (2017) found that childhood ADHD increased the risk (OR = 2.61) of SUDs from middle adolescence to adulthood, however, there was no significant difference in studies examining CUD in comparison to other SUDs. Second, Charach et al. (2011) found that childhood ADHD was associated with alcohol use disorder by young adulthood (OR = 1.35), as well as an increased risk (OR = 1.51) of CUD during young adulthood. However, the authors noted significant heterogeneity among studies included in the CUD-specific meta-analysis. Finally, Lee et al. (2011) compared children with and without ADHD on substance use and substance dependence outcomes in adolescence and adulthood. Children diagnosed with ADHD were more likely to have reported lifetime nicotine dependence (OR = 2.82), alcohol abuse/dependence (OR = 1.74), cannabis use (OR = 2.78), and meet CUD criteria (OR = 2.29) when compared to children without ADHD. However, the authors reported significant heterogeneity across effect sizes.

The prevalence of comorbid CUD among individuals with ADHD is variable, with current estimates ranging from 5% (Faraone et al., 2007) to 35.6% (Elkins et al., 2020), and lifetime estimates ranging from 17.1% (Anker et al., 2020) to 80% (Castaño Pérez and Sierra Hincapié, 2016). Higher rates of cannabis use initiation, frequency, and potency is

concerning (UNODC, 2022). Frequent cannabis use can have negative effects on cognitive processes in youth and emerging adults including learning, memory, attention, decision-making, and inhibitory control (Dellazizzo et al., 2022; Kroon et al., 2021; Scott et al., 2018). These effects may exacerbate challenges with sustained attention experienced by those with ADHD (Fergusson and Boden, 2008; Wallace et al., 2019) which may further impair academic, occupational, and interpersonal functioning (Harty et al., 2015; Lisdahl et al., 2016; Volkow et al., 2016). ADHD has also been associated with the development of CUD, especially among treatment-seeking populations. Notzon et al. (2020) found that between 34% and 46% of individuals seeking treatment for CUD had comorbid ADHD. Furthermore, comorbid CUD may also have treatment implications for individuals with ADHD. Patel et al. (2018) reported that among adolescent patients with ADHD, comorbid CUD increased the risk for needing acute inpatient care and prolonged inpatient stay.

With further global decriminalization and legalization of cannabis products as well as rising cannabis use, establishing CUD prevalence rates among populations at risk is important. There are several factors that may contribute to higher rates of cannabis use and CUD among ADHD populations. However, no meta-analyses have examined the prevalence of comorbid CUD in participants with a current diagnosis of ADHD. This is despite meta-analytic work that has reported that 23.1% of patients seeking treatment for SUDs also had comorbid ADHD (van Emmerik-van Oortmerssen et al., 2012).

As cannabis use continues to rise across the globe, a comprehensive and in-depth meta-analytic review and moderator analysis of existing ADHD and CUD literature is necessary to gain a more nuanced understanding of the intricate relationship between ADHD and CUD. Doing so will allow for more accurate quantifications of CUD prevalence rates among at-risk ADHD populations and will ensure a greater understanding of the extent of such risks. For the present meta-analysis, the target population was purposely inclusive to allow a range of sample types (e.g., inpatient, outpatient, community), age ranges, and genders to increase the representativeness of individuals living with ADHD. As such, the aims of the present meta-analysis are to (1) determine the lifetime and current global prevalence of CUD in individuals meeting criteria for current ADHD, (2) evaluate the relative risk of CUD in those with and without ADHD, and (3) clarify the heterogeneity of prevalence estimates through moderator analyses. In doing so, we hope to provide a benchmark to clinicians in terms of the risk of comorbid CUD in their patients with ADHD to help advocate for increased substance use disorder screening (as substance use symptoms may be misattributed to ADHD).

1. Method

1.1. Procedure

A systematic literature search was conducted on September 26, 2021, using *PubMed*, *PsycINFO* and *Web of Science*, and resulted in a total of 1279 articles. A research librarian at Memorial University of Newfoundland's Queen Elizabeth II Library was consulted for this study to ensure that the natural keywords and controlled terms utilized for ADHD and CUD accurately reflected the literature of interest. The Boolean search phrases utilized for each database have been summarized in the supplementary material (eTable 1). Titles and abstracts were screened to identify articles that met the inclusion criteria. Articles of interest underwent full-text review to determine their eligibility (see Fig. 1).

1.2. Inclusion criteria

Participant samples were required to have a diagnosis of ADHD as defined by the criteria within the validated International Classification of Diseases tenth edition (*ICD-10*; World Health Organization [WHO], 2016), *DSM-III-R* (APA, 1987), *DSM-IV* (APA, 1994), or *DSM-5* (APA, 2013). All diagnoses of current ADHD and lifetime or current CUD were

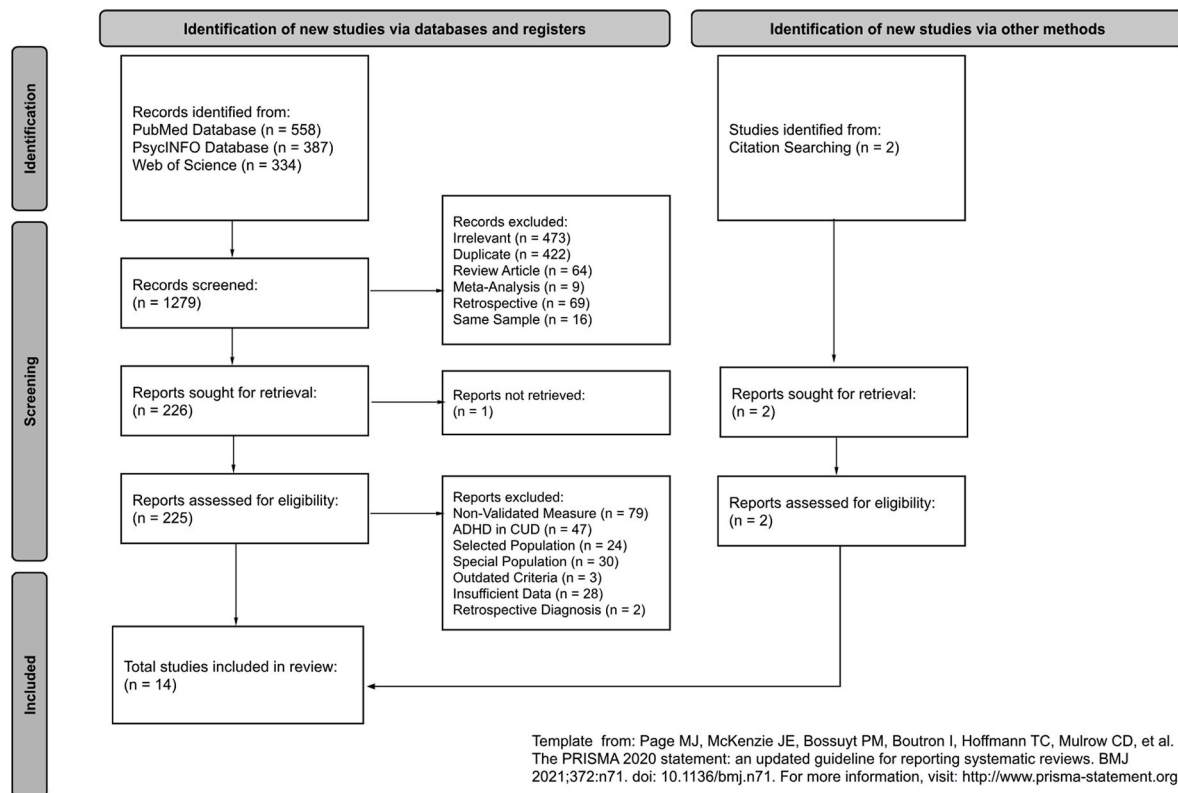


Fig. 1. Meta-analysis inclusion flowchart.

similarly required to be supported by empirically validated screening tools or semi-structured diagnostic measures. For example, employment of the Adult ADHD Self-Report Scale (ASRS; Kessler et al., 2005) or the Cannabis Use Disorders Identification Test-Revised (CUDIT-R; Adamson et al., 2010). Lifetime CUD prevalence was defined as meeting criteria at any point in one's life (may or may not meet CUD criteria currently), whereas current prevalence was defined as point (e.g., past 2 weeks) or period prevalence (e.g., 12-month period prevalence; National Institute of Mental Health, 2023). Specifically, CUD prevalence windows were determined by the particular assessment measures utilized by each study (e.g., past month CUD assessed via the Structured Clinical Interview for DSM versus 12-month CUD estimates from the Composite International Diagnostic Interview Substance Abuse Module). Samples of those with remitted ADHD diagnoses were not eligible for inclusion. All articles included in the meta-analysis must have reported lifetime or current CUD prevalence (i.e., DSM-5 CUD), or cannabis abuse and/or dependence for prior versions of the DSM).

1.3. Exclusion criteria

Articles reporting on samples with a primary diagnosis of CUD were excluded from the analysis. A diagnosis of ADHD based upon DSM-III (APA, 1980) and ICD-9 (WHO, 1978) or earlier criteria and a diagnosis of CUD, cannabis dependence, or abuse with non-validated measures were deemed ineligible for inclusion. Previous meta-analyses, review articles, retrospective studies, selected samples (those specifically recruiting individuals with an existing diagnosis of CUD and ADHD), and special populations (e.g., military personnel; True et al., 1999) were also excluded.

1.4. Data extraction and analytic approach

The following data were extracted: (a) author name, (b) year of publication, (c) sample size, (d) sample type (inpatient, outpatient,

community or control), (e) special population type (if applicable), (f) country, (g) region, (h) mean age, (i) age group (child or adult), (j) gender, (k) percent female, (l) diagnostic criteria, (m) ADHD diagnostic measure, (n) CUD diagnostic measure, (o) prevalence window for CUD (lifetime and/or current), and (p) the overall CUD prevalence in ADHD and control samples. Control samples were operationally defined as healthy individuals without a diagnosis of ADHD (Biederman et al., 2008; Breyer et al., 2014; Elkins et al., 2020; Estévez et al., 2016; Faraone et al., 2007; Lovett et al., 2021; Murphy et al., 2002). Quality ratings are reported in Table 1, with more details about development and scoring criteria for quality assessment found in the supplement (eMethods 1; eTable 2).

1.5. Effect size calculation and analysis

Data analyses were conducted via R version 4.1.1 (R Core Team, 2021) using the *brms* package (Bürkner, 2021). Aggregate estimates of the lifetime and current prevalence of CUD in ADHD were calculated using Bayesian multilevel logistic regression models with study and sample as random effects (as appropriate). In addition to estimating the aggregate prevalence of CUD in a typical sample (excluding control groups), risk ratios were further calculated in two ways. First, the prevalence models were re-fit including control samples, with sample type included as a predictor and random slope; here, the risk ratio depicting heightened risk of CUD in ADHD samples was reflected as the ratio between the posterior prevalence estimate for the ADHD and control samples. This permits use of the full data set (including studies without control groups) and further permits some prior knowledge pertaining to the prevalence of CUD in the general population to be incorporated into the ratio. Second, log-transformed risk ratios were calculated for each study reporting a control group – using the *escal* function from the *metafor* package (Viechtbauer, 2010) – and analyzed using a comparable Gaussian model. In either case, all results were back-transformed (into percentages or risk ratios) to simplify

Table 1
Studies reporting prevalence of cannabis use disorder in populations with attention-deficit hyperactivity disorder.

| Author(s) | Country | ADHD Criteria | ADHD Measure | CUD Criteria | CUD Measure | Prev. Window | Sample Type | Mean age (SD) | % Female | CUD in ADHD % (n) | CUD in Control % (n) | Quality Rating |
|---|---------------|-------------------|--------------------|-------------------|------------------|--------------|-------------|---|------------------------------|---------------------------------------|----------------------|----------------|
| Anker et al. (2020) | Norway | DSM-5 | DIVA 2 | DSM-IV | MINI | LT | O | 36.8 (11.4) | 45.8 | 17.1 (585) | ... | High |
| Elkins et al. (2020) | United States | DSM-5 | ASRS DICA-R | DSM-5 | SAM/CIDI | CUR | CM | 24 | 44.7 | 35.57 (253) | 12.7 (459) | High |
| Estévez et al. (2016) ^a | Switzerland | DSM-IV | ASRS | DSM-IV | CUDIT-R | LT | CM | ... | 0 | 23.5 (221) | 8.1 (5418) | Moderate |
| Vingilis et al. (2014) | Canada | DSM-IV | ASRS | DSM-IV | ASSIST | LT | CM | ... | 54.3 | 12.5 (104) | 4.37 (3250) | Moderate |
| Hechtman et al. (2018) | United States | DSM-5 | CAARS | DSM-IV | DISC | CUR | CM | 24.7 | ... | 26.7 (226) | 12.3 (241) | Moderate |
| Breyer et al. (2014) | United States | DSM-IV | ADI | DSM-IV | ADI | CUR | CM | 18.3 (1.11) | 25.3 | 32.9 (79) | 7.2 (69) | High |
| Biederman et al. (2008) | United States | DSM-IV | K-SADS-E/ SCID | DSM-IV | SCID | LT | O | 21.6 (3.3) | 0 | 19.6 (122) | 8.6 (105) | Moderate |
| Faraone et al. (2007) ^b | United States | DSM-IV | SCID | DSM-IV | SCID | LT | CM/O | 36.1 (10.8) 36.5 (10.6) | 47 (Full) 52 (Late-onset) | Full: 47 (117) Late-onset: 43 (75) | 13.9 (115) | Moderate |
| Faraone et al. (2007) ^b | United States | DSM-IV | SCID | DSM-IV | SCID | CUR | CM/O | 36.1 (10.8) 36.5 (10.6) | 47 (Full) 52 (Late-onset) | Full: 10 (117) Late-onset: 5 (75) | 1.7 (115) | Moderate |
| Agnew-Blais et al. (2016) ^c | UK | DSM-5 | SI | DSM-IV | SI | CUR | CM | 18 | 33.3 (CP) 55.4 (LO) | 14.8 (54) 11.6 (112) | 3.2 (1681) | High |
| Castaño Pérez and Sierra Hincapié (2016) ^d | Colombia | DSM-IV/ ICD-10 | CIDI | DSM-IV/ ICD-10 | CIDI | LT | CM | ... | ... | 80 (45) | ... | Moderate |
| Murphy et al. (2002) ^e | United States | DSM-IV | SI | DSM-IV | SI | CUR | O | ADHD-C 21.3 (2.7) ADHD-I 20.1 (2.1) | 28.3 13.9 | ADHD-C: 20 (60) ADHD-I: 19.4 (36) | 1.5 (64) | Moderate |
| Lovett et al. (2021) ^f | United States | DSM-IV | BAARS-IV | DSM-IV | CUDIT-R | CUR | CM | ... | ... | 10.8 (93) | 7.3 (817) | Low |
| Ohlmeier et al. (2011) | Germany | DSM-IV | CI_CAARS/ Brown | DSM-IV | ICD-10 (IDCL)/CI | LT | O | 35.11 (9.33) | 54.1 | 29.5 (61) | ... | Moderate |
| Bidwell et al. (2014) ^g | United States | DSM-IV | ASRS | DSM-IV | MDS | CUR | CM | ... | ... | 32.7 (171) | ... | Moderate |

Abbreviations: ADHD-C = ADHD Combined Type; ADHD-I = ADHD Inattentive Type; ADI = Adolescent Drinking Index; ASRS = Adult ADHD Self-Report Scale; ASSIST = Alcohol, Smoking and Substance Involvement Screening Test; BAARS-IV = Barkley Adult ADHD Rating Scale-IV; CAARS = Conners' Adult ADHD Rating Scales; CI = Clinical Interview; CIDI = Composite International Diagnostic Interview; CM = Community; CP = Childhood-Persistent ADHD; CUDIT-R = The Cannabis Use Disorder Identification Test - Revised; CUR = Current Prevalence Window; DICA-R = Diagnostic Interview for Children and Adolescents - Revised; DISC = Diagnostic Interview Schedule for Children; DIVA = Diagnostic Interview for ADHD in Adults; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders 4th Edition; DSM-5 = Diagnostic and Statistical Manual of Mental Disorders 5th Edition; ICD-10 = International Classification of Diseases, 10th Edition; LO = Late-Onset ADHD; LT = Lifetime Prevalence Window; MDS = Marijuana Dependence Scale; MINI = Mini-International Neuropsychiatric Interview; O = Outpatient; SAM = Substance Abuse Module; SCID = Structured Clinical Interview for DSM.

^a Mean age (SD) across the entire sample (ADHD and non-ADHD controls).

^b Obtained prevalence data from authors. *N*'s are different from those presented in the paper since they are from the authors. In the full ADHD group, 117/127 participants had Structured Clinical Interviews for DSM-IV (SCID) data available for cannabis use disorder; In the late-onset ADHD group, 75/79 participants had SCID data available for cannabis use disorder.

^c For both the childhood-persistent ADHD group (CP) and the late-onset ADHD group (LO), the mean age was not provided but listed as 18 as participants were examined at age 18.

^d Data was received from correspondence with the first author. ADHD is past-year. DSM-IV and ICD-10 - CIDI used.

^e Sample type was listed as "clinically referred", which we coded as outpatient; Prevalence estimates were provided separately for ADHD combined type (ADHD-C), and ADHD inattentive type (ADHD-I).

^f Data was received from correspondence with authors. ADHD Criteria was considered met by having five or more symptoms according to the BAARS.

^g Data was received from correspondence with authors. ADHD Criteria was based on the number of people who endorsed six or more current symptoms of ADHD according to the BAARS-IV, and past-year marijuana dependence was based on individuals who scored a 3 or higher on the Marijuana Dependence Scale.

interpretation of our figures. Priors for each model are detailed in the supplementary material (eMethods 2).

In addition to our primary analyses estimating the prevalence and risk of CUD in populations with ADHD, we further coded a series of moderators for analysis (mean age, proportion of the sample that was female, diagnostic criterion, diagnostic method, and quality ratings or categories).

2. Results

2.1. Description of studies

Of the 1279 studies initially identified, a total of 14 were included in our analysis (see Fig. 1). Seven studies provided estimates of lifetime CUD prevalence, with four providing lifetime prevalence estimates for control samples. Further, eight studies provided estimates of current CUD prevalence, with seven reporting current prevalence estimates for the control samples. In our analysis, 64% of the studies included were from North America with an ADHD sample that was 34.9% female and a mean age of 25.9 years. The control sample was 36.3% female and on average 22.6 years old. Overall, 86% of the samples were exclusively adults and 14% of the samples included both adolescents and adults. Self-report measures for both ADHD and CUD were utilized for 29% of the sample, while 71% of the samples relied on diagnostic interviews for diagnosis of ADHD and CUD. Due to the small number of studies included – compounded by missing data – we were unable to analyze most moderator variables, and none of those considered excluded 0 (i.e., no difference or effect) as a probable value. Due to these concerns – and in the interest of brevity – we have excluded those models from the present manuscript.

Lifetime Prevalence of CUD in Populations with ADHD. Prior to analysis, Castaño Pérez and Sierra Hincapié (2016) was identified as an influential outlier (with an estimate of 80% whereas the next highest estimate was 47%). Due to its prevalence estimate being substantially higher than other estimates (roughly 4 standard deviations above the mean in our sample), and idiosyncrasies inherent in its sample (e.g., inclusion of participants as young as 13), we opted to exclude this study from our models.¹ Following this exclusion, as depicted in Fig. 2, the aggregate lifetime prevalence of CUD in populations with ADHD was found to be 26.9%, 95% CI [16.8%, 39.2%], with a prediction interval ranging from 12.4% to 48.8%. This implies that a prevalence estimate in a new sample might range as low as ~12% or as high as ~49%.²

Lifetime Risk of CUD Compared to General Populations. Having established the prevalence of CUD in populations with ADHD, we next explored whether those populations were at greater risk of a lifetime diagnosis of CUD than the general population. Those with ADHD were found to be 2.85, 95% CI [1.71, 3.99], and 2.94, 95% CI [1.43, 5.20],

¹ Inclusion of this study increases our estimated aggregate effect to 34.1%, 95% CI [19.6%, 51.4%] with a prediction interval ranging from 11.9% to 79.1%. Further sensitivity analyses conducted excluding two additional studies (not identified as outliers) that were unusual in their sample being comprised of 100% male identifying participants and/or including individuals younger than 18 years of age (Biederman et al., 2008; Estévez et al., 2016) slightly increased the aggregate prevalence estimate to 30.5%, 95% CI [15.8%, 48.8%], with a prediction interval ranging from 13.4% to 51.5%.

² Although the reported prediction intervals may appear broad, they are typical of those observed in epidemiological meta-analyses dealing with comorbidity. For example, a meta-analytic review of the prevalence of OCD in those with diagnosed eating disorders reported prediction intervals ranging from ~1% to ~40% (Drakes et al., 2021). Although such a range may limit the utility of the aggregate estimate (which reflects expected performance in a “typical” study), it is still an important reflection of variability within this field. Further, the present analyses would likewise permit us to conclude that in any given sample (similar to those included) no less than 12% of those individuals would be expected to have comorbid CUD.

times more likely to be diagnosed with CUD than the general population, as estimated via our analysis of log-risk ratios or a logistic model inclusive of control samples, respectively. Prediction intervals ranging from 1.7 to 4.0 (based on the log-risk ratio model, as also depicted in Fig. 3), indicating that all studies similar to those included would be expected to demonstrate at least some heightened risk of CUD in populations with ADHD.

Current Prevalence of CUD in Populations with ADHD. As depicted in Fig. 4, the aggregate current prevalence of CUD in populations with ADHD was found to be 19.2%, 95% CI [12.5%, 27.0%] with a prediction interval ranging from 5.5% to 39.1%. This implies comparable heterogeneity to that observed in the lifetime prevalence estimates.³

Current Risk of CUD Compared to General Populations. Our analyses again found those with ADHD to be 2.91, 95% CI [2.12, 4.04], and 3.33, 95% CI [1.99, 5.44], times more likely to be diagnosed with CUD than the general population, as estimated via our analysis of log-risk ratios or a logistic model inclusive of control samples, respectively. Unlike our analysis of prevalence, prediction intervals were narrow, ranging from 1.3 to 5.1 (based on the log-risk ratio model, as depicted in Fig. 5). This indicates that all studies similar to those included would be expected to demonstrate at least some heightened risk of CUD in populations with ADHD.

3. Discussion

Our meta-analysis compiles existing research and provides a more comprehensive understanding of the scope of CUD in ADHD for researchers, clinicians, and policymakers. Through the assessment of findings from existing studies, our meta-analysis further helps to identify knowledge gaps, inconsistencies, heterogeneity, and areas where data is lacking in the existing literature. In doing so, we reveal where further investigation is needed to gain a more nuanced understanding of the relationship between ADHD and CUD. Specifically, we conducted a meta-analysis of the prevalence of CUD in individuals currently meeting criteria for an ADHD diagnosis, given the considerable variability in the literature with estimates ranging from 5% (Faraone et al., 2007) to as high as 80% (Castaño Pérez and Sierra Hincapié, 2016). The meta-analytic aggregate lifetime prevalence estimate of CUD in populations with ADHD was greater (26.9%, 95% PI [12.4%, 48.8%]) than the estimate of current CUD prevalence in this population (19.2%, 95% PI [5.5%, 39.1%]). Moreover, lifetime (2.85, 95% PI [1.7 to 4.0]) and current (2.91, 95% PI [1.3, 5.1]) risk-ratio estimates found those with ADHD to be at nearly three times greater risk for experiencing CUD than the general population. Our findings are consistent with our predictions and support the hypothesis that individuals with ADHD are at greater risk than those without ADHD for experiencing CUD.

Our meta-analysis underscores the extensive heterogeneity present within existing CUD in ADHD literature. This degree of heterogeneity is typical of epidemiological meta-analyses dealing with comorbidity (see Footnote 2), revealing where further investigation is needed to gain a more nuanced understanding of the relationship between ADHD and CUD. Due to the failure of many studies to include information specific to ADHD populations, such as age, diagnostic history, and gender, our study was unable to perform moderator analyses as intended. As a result of such lacking data identified through our analysis, we further highlight the need and the importance for future studies to report on such information. Providing a clearer report of CUD prevalence, especially as cannabis use continues to rise across the globe, will help to guide future

³ Although not identified as an outlier, one study was included in this analysis that reported participants as young as 17 years of age (Murphy et al., 2002). A sensitivity analysis excluding this study produced an estimate of 19.0%, 95% CI [11.6%, 27.8%] with a prediction interval ranging from 4.8% to 39.9%, suggesting inclusion of this study failed to substantially impact either our aggregate estimate or heterogeneity.

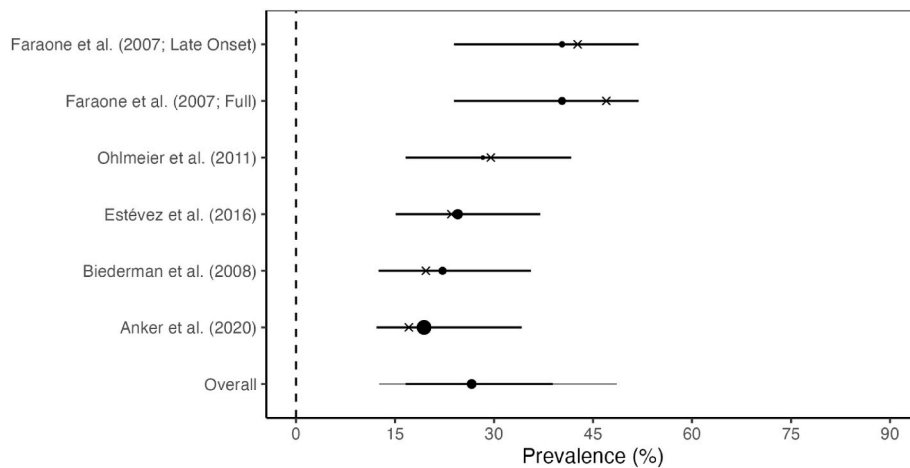


Fig. 2. Forest Plot Representing CUD Prevalence (%) for the Lifetime Measurement Window

The forest plot in Fig. 2 illustrates the prevalence of lifetime CUD diagnoses (%) among populations with ADHD.

Note. Points and error bars reflect model estimates with corresponding 95% confidence intervals. The prevalence reported in each publication is marked with an “X” and the aggregate estimate is illustrated by the final entry labelled “Overall.” The 95% prediction interval is denoted by the thin grey line radiating from the aggregate point estimate.

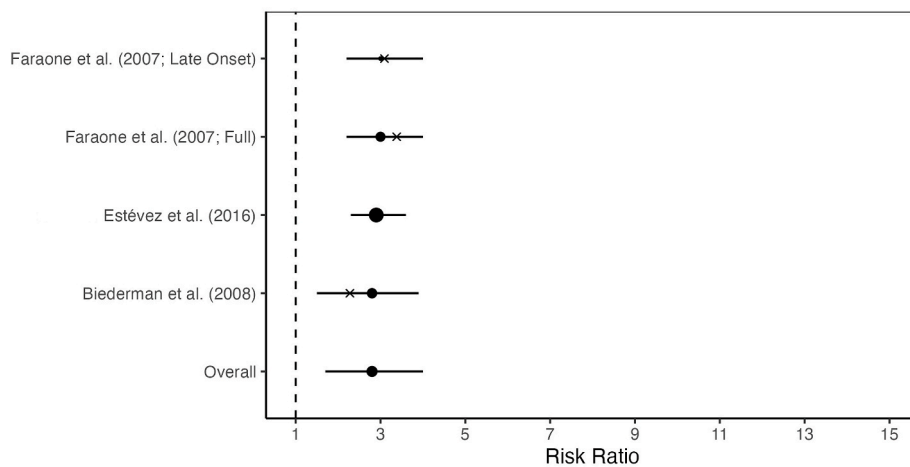


Fig. 3. Forest Plot Representing the Risk Ratio Between the Prevalence of CUD in Populations with and without ADHD for the Lifetime Measurement Window

The forest plot in Fig. 3 illustrates the risk ratio between lifetime CUD diagnoses among those with and without a diagnosis of ADHD.

Note. Points and error bars reflect model estimates with corresponding 95% confidence intervals back-transformed from our analysis of log-risk ratios. The risk ratio reported in each publication is marked with an “X” and the aggregate estimate is illustrated by the final entry labelled “Overall.” The 95% prediction interval is denoted by the thin grey line radiating from the aggregate point estimate.

research efforts and clinical practice.

Unlike prior meta-analyses that have examined the later development of SUDs (including CUD) among individuals with a childhood ADHD diagnosis (Charach et al., 2011; Groenman et al., 2017; Lee et al., 2011), our meta-analysis was the first to include only studies that assessed for current ADHD diagnostic symptoms while examining lifetime and current prevalence of CUD in ADHD populations. In our literature review, a sizable proportion of longitudinal follow-up studies assessing CUD in both adolescent and adult ADHD populations failed to measure current ADHD status and instead relied solely on archival ADHD diagnosis data (e.g., Molina and Pelham, 2003; Molina et al., 2013). However, ADHD diagnoses can lack temporal stability (Breyer et al., 2014). This is supported by Sibley et al. (2022), who found that nearly 64% of individuals diagnosed with ADHD exhibited fluctuating periods of symptom persistence, while only 10.8% of individuals diagnosed with ADHD demonstrated stable symptom persistence. Similarly, in a meta-analysis examining the persistence of ADHD into adulthood, Faraone et al. (2006) found that when using DSM-IV criteria, only ~15% of children diagnosed with ADHD will continue to meet full criteria for

the disorder at age 25, while ~40–60% will meet DSM-IV’s definition of ADHD in partial remission. While it is important to understand later outcomes associated with childhood ADHD including rates of CUD, due to the fluctuation of ADHD diagnosis persistence, it is also important to understand prevalence rates of CUD in individuals with a ‘current’ ADHD diagnosis.

There are a number of factors that help explain high rates of comorbidity between ADHD and CUD. Perugi et al. (2019) propose three common mechanisms to help understand this relationship. First, impairments in executive functioning including impulsivity can increase an individual’s susceptibility to engage in substance use, and subsequently, their risk of developing a CUD. Second, individuals with ADHD often display difficulties in modulating or delaying their reward response. As a result, they have greater difficulty with self-control, and thus, their ability to consider the consequences of their actions including substance use is impaired. Lastly, psychosocial risk factors such as earlier exposure to addictive substances, poor educational outcomes, and adverse peer group influences are more commonly associated with ADHD, putting individuals at greater risk of developing SUDs, such as

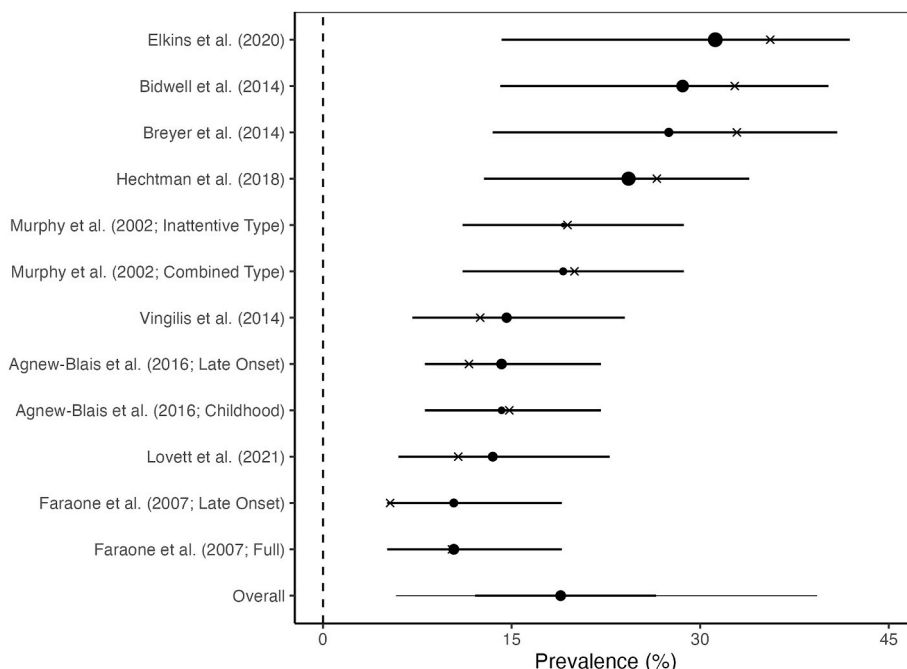


Fig. 4. Forest Plot Representing CUD Prevalence (%) for the Current Measurement Window
 The forest plot in Fig. 4 illustrates the prevalence of current CUD diagnoses (%) among populations with ADHD.
 Note. Points and error bars reflect model estimates with corresponding 95% confidence intervals. The prevalence reported in each publication is marked with an “X” and the aggregate estimate is illustrated by the final entry labelled “Overall.” The 95% prediction interval is denoted by the thin grey line radiating from the aggregate point estimate.

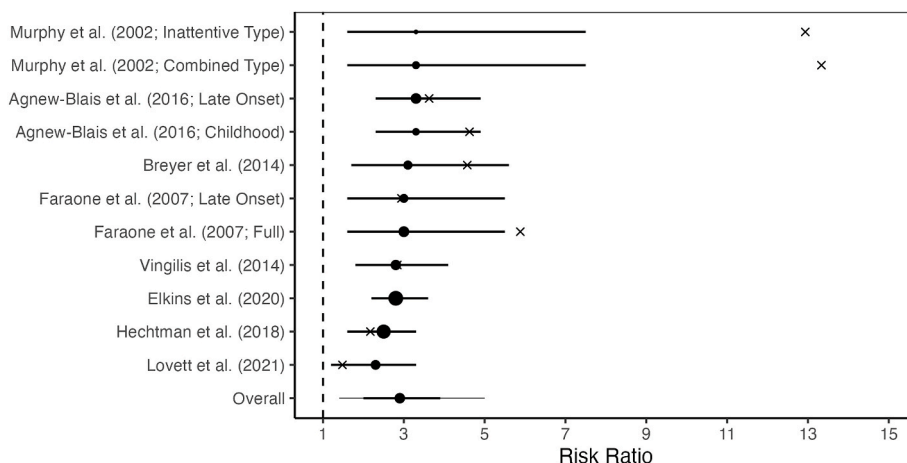


Fig. 5. Forest Plot Representing the Risk Ratio Between the Prevalence of CUD in Populations with and without ADHD for the Current Measurement Window
 The forest plot in Fig. 5 illustrates the risk ratio between current CUD diagnoses among those with and without a diagnosis of ADHD.
 Note. Points and error bars reflect model estimates with corresponding 95% confidence intervals back-transformed from our analysis of log-risk ratios. The risk ratio reported in each publication is marked with an “X” and the aggregate estimate is illustrated by the final entry labelled “Overall.” The 95% prediction interval is denoted by the thin grey line radiating from the aggregate point estimate.

CUD.

Likewise, there is evidence that females with ADHD have a higher prevalence of comorbid SUDs, such as CUD (Ottosen et al., 2016). While we were unable to test gender differences in the present study, problematic substance use has been found to be more prevalent among women with comorbid psychiatric conditions due to coping and self-medication (Fonseca et al., 2021), along with women developing a quicker progression to substance use problems relative to men (Hernández-Avila et al., 2004).

The finding that individuals with ADHD were at nearly three times greater risk of having a lifetime or current diagnosis of CUD speaks to the

importance of prevention in this population. A greater understanding of social factors that contribute to the development of CUD in ADHD may help better inform prevention strategies for youth. Due to increased risks of more frequent performance failures, low self-esteem, and an increased association with deviant peers, adolescents with ADHD are more likely to develop SUDs (Hussong et al., 2011; Molina and Pelham, 2014; Zulauf et al., 2014). In populations with ADHD, social rejection may lead to an affiliation with substance-using peers, thereby increasing one’s risk of cannabis use and the development of CUD. According to Chauchard et al. (2018) the most common strategy for maintaining cannabis abstinence reported by participants with ADHD and comorbid

cannabis dependence was to stop associating with individuals that engage in cannabis use. Additionally, [Esparza-Reig et al. \(2021\)](#) suggests that prosociality is a protective factor against behaviours of addiction, therefore, promotion of prosocial involvement (e.g., volunteering or sports team participation) in adolescents with ADHD may mitigate future development of CUD. Moreover, educating teachers, students, parents and guardians about the risks associated with cannabis during adolescence may be beneficial in preventing early cannabis initiation and subsequent CUD development, particularly among at-risk groups.

Our findings support the need for mental health professionals to carefully consider the potential implications of co-occurring cannabis use among patients presenting with symptoms associated with ADHD. For example, deficits in executive functioning such as attention, impulsivity, decision making, and risk-taking behaviours among frequent cannabis users can mimic ADHD symptoms ([APA, 2013](#); [Crean et al., 2011](#)). Thus, symptoms may be attributed to cannabis use, resulting in ADHD being overlooked and misdiagnosed as CUD ([Crean et al., 2011](#)). In contrast, if an individual does not report or is not assessed for cannabis use, a false-positive ADHD diagnosis may be given. In either circumstance, the exacerbation of ADHD or CUD symptoms could occur, as individuals cannot take advantage of available treatments. Consequently, appropriate clinical intervention (both pharmacologic and psychosocial) may be difficult to achieve without a proper understanding of CUD and ADHD symptom overlap. As such, our findings are in line with previous literature that supports the inclusion of CUD screening in ADHD populations ([Molinero and Hinckley, 2023](#)).

The current study has some important limitations. First, our sample size was small, with only 14 studies deemed eligible for inclusion which limited our ability to draw strong inferences regarding gender. Given that the ADHD sample in our study was 34.9% female and the control sample was 36.3% female, our results may not be representative of the true prevalence of CUD in ADHD for females. This study allowed for the inclusion of studies with all male participants ([Biederman et al., 2008](#); [Estévez et al., 2016](#)) as we had planned to examine the gender composition of the sample as a moderator. However, due to the small proportion of eligible studies recovered through our search, we were unable to do so, as such these studies may have added heterogeneity to our estimates. Small sample sizes also impacted our ability to examine moderators such as ADHD subtype and age of initiation which play unique roles in the progression of cannabis use and CUD. For example, [Bidwell et al. \(2014\)](#) reported inattentive subtype to be linked to more severe cannabis use, cravings, and problematic use-related outcomes in young adults, whereas hyperactive-impulsive subtype symptoms were linked to an earlier age of cannabis initiation. Furthermore, [Elkins et al. \(2018\)](#) found that females displaying greater hyperactivity-impulsivity symptoms progressed further toward daily cannabis use than their male counterparts. Additionally, regular cannabis consumption prior to the age of 16 can increase cannabis frequency (twice as often), magnitude of use (nearly three times as much cannabis), and impact executive functioning compared to those who initiated cannabis later in life ([Gruber et al., 2012](#)). It should also be noted that we were unable to examine the impact of moderators such as mean age and calendar year to determine their influence on current CUD prevalence rates. For instance, future studies should examine whether CUD prevalence rates have increased over time, with respect to increased legalization and acceptability of cannabis consumption. Further, for lifetime prevalence, studies where samples had an older mean age (e.g., 36.8; [Anker et al., 2020](#)) compared to a younger mean age (e.g., age 18; [Agnew-Blais et al., 2016](#)), would have resulted in a longer assessment window for CUD development, and thus may have introduced further heterogeneity into the lifetime prevalence estimates. Future studies should examine and report information pertaining to potential moderator variables between ADHD and CUD.

Another limitation in our study relates to our estimate of CUD prevalence. The majority of our included studies used *DSM-IV* as opposed to *DSM-5* criteria for SUDs. Therefore, our prevalence estimates

may better reflect the *DSM-IV*. Furthermore, some of our included studies assessed diagnoses of ADHD and CUD using purely self-report measures ([Bidwell et al., 2014](#); [Estévez et al., 2016](#); [Lovett et al., 2021](#); [Vingilis et al., 2014](#)). Self-report ADHD measures, such as the Conners' Adult ADHD Rating Scale ([Conners et al., 1999](#)), are effective tools for identifying clinically significant attention problems, however, they fail to distinguish between ADHD and other psychiatric disorders ([Van Voorhees et al., 2011](#)). Although hetero-evaluation (e.g., parent/teacher reports) is more common in childhood than adult ADHD assessment, given concerns with the accuracy of self-reporting level of impairment ([Suhr et al., 2017](#)) and the poor discriminant validity of adult self-rating scales and cognitive tests such as the continuous performance test for ADHD symptoms ([Söderström et al., 2014](#)), clinicians are encouraged to integrate these measures with multiple data sources and not use them exclusively for diagnostic or accommodation decisions ([Söderström et al., 2014](#); [Suhr et al., 2017](#)). Similarly, self-report SUD measures (e.g., the Marijuana Dependence Scale; [Stephens et al., 2000](#)) have been shown to provide inaccurate estimates of prevalence rates ([Khalili et al., 2021](#)). To identify differential diagnoses among those seeking ADHD evaluation and accurate representations of SUD prevalence, self-report measures should be used in combination with clinical diagnostic interviews ([Khalili et al., 2021](#)). Notably, however, all the samples included utilized validated measures, with 71% having used semi-structured or structured clinical diagnostic interviews for ADHD and CUD diagnoses.

Lastly, an important limitation pertains to geographic location. Our results pertain largely to North America, accounting for nine of the 14 studies. Four studies came from Europe and one came from South America. As such, our results are limited as they fail to provide representation from Africa, Asia, or Australia. Further research on CUD estimates in ADHD populations are needed in these regions to determine a more accurate global prevalence of lifetime and current CUD. Cross-cultural comparisons could improve knowledge of risk factors and allow for the development of appropriate harm prevention strategies for CUD and problematic substance use in ADHD populations.

CRediT authorship contribution statement

Anna M. Froude: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Emily J. Fawcett:** Writing – review & editing, Visualization, Validation, Supervision, Project administration, Investigation, Formal analysis, Data curation. **Ashlee Coles:** Writing – review & editing, Visualization, Validation, Investigation, Formal analysis, Data curation. **Dalainey H. Drakes:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation. **Nick Harris:** Writing – review & editing, Validation, Supervision, Project administration, Methodology, Investigation, Formal analysis, Conceptualization. **Jonathan M. Fawcett:** Writing – review & editing, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Formal analysis, Conceptualization.

Declaration of competing interest

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jpsychires.2024.02.050>.

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