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The effects of cannabis use disorder on cognitive functions: A meta-analysis

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ABSTRACT

Background and aims: Impairments in cognition are frequently associated with acute cannabis consumption; on the other hand, controversies persist regarding the residual cognitive impairments of cannabis, with some estimates highlighting significant or mild cognitive impairment. One of the main limitations of the available research syntheses is that little attention has been paid to individuals with cannabis use disorder. Thus, our main objectives are to determine the amplitude of the cognitive deficits associated with cannabis use disorder, and to identify the cognitive domains the most and least impaired.

Methods: Studies with a patient group with a cannabis use disorder diagnosis and data from at least one validated neurocognitive test were selected. After manual extraction, data were pooled in a multivariate meta-analysis and effect size estimates were calculated for 13 cognitive domains. Meta-regression analyses on potential moderators were performed.

Findings: There were small-to-moderate impairments in 10 out of the 13 cognitive domains. Deficits in verbal learning/memory, speed of processing and working memory were more prominent (d = 0.4/0.5) whereas verbal fluency and attention were the least affected. No association was observed between the potential moderators and global cognition.

Conclusion: This meta-analysis shows that cannabis use disorder is associated with moderate deficits in verbal learning/memory, speed of processing and working memory. Despite the limitation of the studies in the field, our results should serve as a reminder that the residual cognitive impairments associated with cannabis should not be under-estimated prematurely.

1. Introduction

With the legalization of cannabis for recreational and medical purposes in a growing number of jurisdictions, mainly in North America, the study of the potential benefits and harms of the substance has become a public health priority (Hasin, 2018). While there is a dearth of controlled clinical trials on the possible therapeutic effects of cannabis *per se* (Fisher et al., 2021), robust evidence shows that cannabis use is associated with a risk of dependence, even more so when the substance is initiated in early adolescence (Petrilli et al., 2022), and with an increased risk of developing psychotic symptoms (Marconi, Di Forti, Lewis, Murray, & Vassos, 2016). Randomized placebo-controlled studies have shown that acute administration of delta-9-tetrahydrocannabidol (Δ^9-THC) – the main psychoactive ingredient of cannabis – produces significant cognitive deficits in experimental settings, particularly deficits in verbal and working memory (Zhornitsky et al., 2021). By comparison, the magnitude of residual cognitive deficits associated with chronic cannabis use remains controversial, with some estimates suggesting that deficits can be substantial, while others describe them as minimal (Meier et al., 2012; Scott et al., 2018). Although most studies have shown impairing effects of cannabis on memory, the effects on other cognitive domains (for example, executive functions) have been more heterogeneous (Broyd, van Hell, Beale, Yucel, & Solowij, 2016; Schreiner & Dunn, 2012). Some studies have shown a negative association between frequency and/or duration of cannabis use and cognitive performance, while others have not shown dose-responses (Dellazizzo, Potvin, Giguere, & Dumais, 2022; Meier et al., 2012). Finally, the impact of duration of cannabis abstinence on cognition remains unclear. While most studies suggest that cognitive performance becomes normalized after 2–4 weeks of abstinence, some studies have shown residual effects lasting up to 12 months (Morin et al., 2019; Schreiner & Dunn, 2012).

One of the important clinical issues that may significantly influence

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the results regarding the residual cognitive deficits of cannabis use is that a large proportion of studies have focused on recreational use (Bourque & Potvin, 2021; Broyd et al., 2016), with less attention being paid more specifically to cannabis use disorder (CUD) (Meier et al., 2012; Solowij et al., 2002). For psychoactive substances other than cannabis, such as alcohol for instance, this distinction has been shown to influence results significantly, with larger cognitive deficits being observed in individuals meeting criteria for alcohol use disorder, as compared to recreational users (Akagi et al., 2022; Brennan et al., 2020; Crowe, Cammisuli, & Stranks, 2019; Stavro, Pelletier, & Potvin, 2013; Zhang et al., 2020).

As for cannabis, a meta-analysis of 69 studies in adolescents and young adults showed that cannabis use was associated, overall, with cognitive deficits of small amplitude ($d = 0.2$) (Scott et al., 2018). It is noteworthy, however, that in a sub-analysis comprising 12 studies conducted in treatment-seeking participants, greater cognitive deficits ($d = 0.43$) were observed. Unfortunately, no results on specific cognitive domains were presented apart from a global cognition outcome. While the main analysis produced results suggesting that the residual cognitive deficits of cannabis use are relatively minor, the sub-analysis involving treatment-seeking individuals revealed cognitive deficits of similar amplitude as those observed in alcohol, cocaine and methamphetamine use disorders (Crowe et al., 2019; Potvin, Grot, Hébert, Barr, & Lecomte, 2018; Potvin, Rizkallah, & Pelletier, 2014; Stavro et al., 2013). Similarly, a meta-analysis was carried out on the cognitive effects of cannabis in studies involving only heavy users (Figueiredo, Tolomeo, Steele, & Baldacchino, 2019). Based on the aggregation of a small set of studies ($n = 13$), their results showed moderate deficits in short-term and long-term memory. Unfortunately, the populations of interest comprised mostly chronic users, with only 5 studies included in the meta-analysis investigating people with CUD. Finally, a meta-analysis of 30 studies examining the residual cognitive effects of cannabis (abstinence > 12 h) in long-term, regular, and recreational users was performed (Lovell, Akhurst, Padgett, Garry, & Matthews, 2020). Results revealed small-magnitude deficits in executive functions and verbal learning/memory. Out of 30 included studies, 9 studies investigated individuals with CUD, but no specific analysis was performed on these studies. Finally, the results of the previous syntheses from Scott et al. (2018), Figueiredo et al. (2019) and Lovell et al. (2020) raise the need for focused analyses on CUD to determine if the cognitive effects of cannabis are larger in those with a more problematic use. The publication of new studies on the cognitive effects associated with CUD in recent years makes possible the pursuit of this objective (Koenis et al., 2021; Manza, Shokri-Kojori, & Volkow, 2020; Selamoglu, Langley, Crean, Savulich, Cormack, Sahakian, & Mason, 2021).

The aim of the current meta-analysis is to determine the amplitude of the cognitive deficits associated with CUD, and to identify the cognitive domains the most and least impaired. To facilitate comparisons between cannabis and other psycho-active substances, we used a meta-analytic approach successfully used by our research team in several meta-analyses on the acute and residual cognitive effects of several psychoactive substances.

2. Methods

2.1. Literature search

The systematic search was done on April 22th 2025 and records were queried from three different databases: Web of Sciences, Pubmed and EMBASE. Together, they host all the records from: Web of Science Core Collection, MEDLINE, KCI-Korean Journal Database, Current Contents Connect, SciELO Citation Index and Preprint Citation Index. The search query was as followed: [(cannabis use disorder OR cannabis user OR cannabis abuse OR cannabis dependence OR cannabis addiction) AND (cognit* OR neuropsychologi* OR memory OR executive functions OR attention OR problem solving) NOT (recreational)].

2.2. Selection criteria

To be included, records had to meet the following criteria: (i) included patients with a CUD diagnosis (current; abuse and/or dependence), (ii) included a healthy group for comparison, (iii) reported the data from at least one validated neurocognitive test, (iv) did not focus on the acute effects of cannabis. Studies were excluded if: (i) there was no cannabis use disorder, (ii) there was no healthy group, (iii) there was missing cognitive data, (iv) there was an Axis 1 psychiatric comorbidity in all participants, (v) studies including any participant with another substance use disorder, apart from nicotine use disorder (in the case of alcohol, studies were excluded if 100 % of participants had an AUD), (vi) there was an overlap with data from another study, and (vii) the number of subjects was lower than five. The selection of reports was reviewed by two researchers (FP and SP) based on the inclusion/exclusion criteria. Moreover, the Preferred Reported Items for Systematic Reviews and Meta-Analysis (PRISMA) guidelines were followed (see Supplementary Table 1) (Page et al., 2021).

2.3. Cognitive domains

Scores (means & SD) from the neuropsychological tests as well as their direction were manually extracted. The neuropsychological tests measured within each of these studies were grouped according to 13 cognitive domains: attention, emotion recognition, executive functions, impulsivity, intelligence quotient (IQ), speed of processing, verbal fluency, verbal learning, verbal memory, visual learning, visual memory, visuo-spatial abilities and working memory. To determine how neuropsychological tests would be assigned to a specific cognitive domain, the authors (FP & SP) based their final decisions on test classification according to Lezak, Howieson, Bigler, and Tranel (2012) and our previous meta-analyses on cognition in substance use disorders (Lezak et al., 2012; Potvin et al., 2018; Potvin et al., 2014; Potvin, Joyal, Pelletier, & Stip, 2008; Stavro et al., 2013). Please refer to Supplementary Table 2.

2.4. Quantitative data synthesis

All multivariate analyses were performed in *R* (Team, 2023) using the *metafor* package (Viechtbauer, 2010). For each analysis, a combined effect-size (e.g. Cohen's *d*) was estimated using a multivariate model with a random intercept nested in studies, accounting for the intra-study correlation of multiple outcomes from the same study. Positive effect sizes indicated worse cognitive performance in individuals with CUD compared to healthy controls. Following the convention from Cohen (1988), effect size estimates of 0.2, 0.5 and 0.8 were considered as small, medium and large, respectively.

Heterogeneity was assessed using the *Q* statistics (Paulson & Bazemore, 2010) and the magnitude quantified with the I^2 index (Lipsey & Wilson, 2001), following guidelines for multivariate models (Higgins & Thompson, 2002). Considering that the data set was characterized by significant levels of heterogeneity (see section 3.2), the aggregation of study outcomes across studies was performed using random-effects models (DerSimonian & Laird, 1986). To evaluate the potential of publication bias, a funnel plot and an Egger's regression test were performed (Egger, Davey Smith, Schneider, & Minder, 1997).

Then, we examined the effect, across studies, of chronic cannabis consumption in individuals with a CUD, relative to healthy controls, both across all cognitive outcomes (i.e. global cognition) and within each of the 13 above-mentioned cognitive domains. To assess the robustness of our findings, we conducted two sensitivity analyses for both the combined model (including all outcomes) and the domain-specific model (grouped by cognitive domain). First, we examined the distribution of standardized residuals to identify potential outliers or extreme values. Second, we performed a leave-one-out sensitivity analysis adapted to the multivariate meta-analytic framework. In this

approach, each of the 24 studies was sequentially excluded from the model to evaluate the influence of individual studies on the overall and domain-specific effect size estimates.

Finally, to explore the effects of continuous variables (e.g. mean age, percentage of women, percentage of CUD participants, percentage of AUD participants, frequency of use (days/week), quantity (g/day) and duration of cannabis use (years) on the cognitive effects, we performed meta-regression analyses. Moreover, we performed sub-analyses on duration of abstinence before cognitive assessment (short $\leq$ 10 days; versus long $>$ 25 days). The multivariate effect size estimates were used for both the meta-regression and sub-analyses.

3. Results

3.1. Study selection

The initial search yielded 7917 articles: 2104 from Web of Science, 2800 from PubMed and 3013 from EMBASE (see Fig. 1). Most of the duplicates were then removed using EndNote (version 20.5) and the rest was removed manually. 5412 articles were exposed to the screening process, based on the abstract, the records were rejected if found nonrelevant. The remaining 158 articles were assessed for eligibility based on the full text. Of these, 133 were excluded for different reasons: (i) 18 articles had no healthy group, (ii) 63 articles had no CUD group, (iii) 20 articles were missing cognitive data, (iv) 2 had an Axis 1 psychiatric comorbidity in all participants, (v) 23 had another substance use disorder in all participants, (vi) 5 overlapped with data from another study, and (vii) 2 had a number of subjects less than five. Therefore, 23 studies were included in the *meta*-analysis, with all studies including a minimum of 60 % of participants with a CUD (note: it was not 100 % in only 4 studies). Only 6 of these studies have included a subgroup of participants with comorbid AUD (see Supplementary Table 3 for studies' characteristics).

3.2. Combined outcomes across cognitive deficits

The primary analysis which included all cognitive measures (n = 108) showed a positive effect of cannabis in individuals with a CUD compared to healthy controls on global cognition. Supplementary Table 4 shows a global cognitive impairment with a small-to-moderate effect size across all cognitive domains (Cohen's d = 0.35). For the combined outcomes, there was significant heterogeneity (I^2 = 60.2 %) (see Supplementary Table 4). Visual inspection of the funnel plot suggested asymmetry in our meta-analytic results. This was supported by Egger's test, which was statistically significant (t = 2.80; $p < 0.05$), indicating that the observed asymmetry is unlikely to be due to chance. This pattern may reflect potential publication bias, where smaller studies disproportionately report larger effect sizes, possibly due to selective publishing.

Then, visual inspection of the histogram of standardized residuals confirms that they all fell within the standard range of −3 to +3, indicating the absence of apparent outliers or extreme values. Then, the leave-one out analysis demonstrated that the pooled effect size remained highly consistent across iterations, with minimal variation (range: d = 0.32 to d = 0.37) compared to the full model estimate (d = 0.35). Overall, these findings suggest that our results are not unduly influenced by any single study and are robust across the dataset.

3.3. Outcomes in specific cognitive domains

The analysis on cognitive domains revealed significant differences ($p < 0.001$) between performance in the clinical and healthy group in ten cognitive domains with a small-to-moderate magnitude. Moreover, the Q-test for subgroup differences indicates a trend toward differences across the cognitive domains (p = 0.07), suggesting that some domains may be more affected than others. Although this result is not statistically significant, it still provides evidence that domain type could contribute

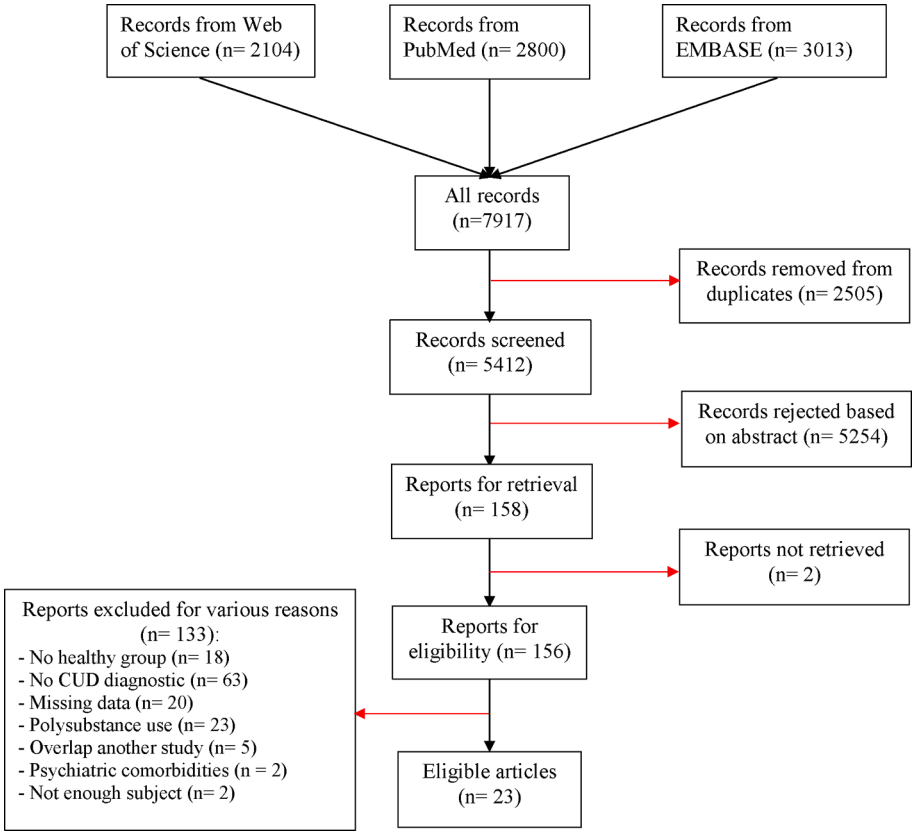

Fig. 1. Flow chart of study selection for a *meta*-analysis assessing cognitive function in patients with a cannabis use disorder.

to explaining variability in the observed outcomes, justifying further analysis of the domains separately. Table 1 and Fig. 2 displays effect size estimates by neurocognitive domains which ranged from small (d = 0.25) to moderate magnitude (d = 0.50). The smallest effects were observed in verbal fluency (d = 0.25), attention (d = 0.26) and impulsivity (d = 0.28) while the largest deficits were observed in verbal memory (d = 0.40), working memory (d = 0.40), speed of processing (d = 0.40), verbal learning (d = 0.48), and IQ (d = 0.50).

Again, visual inspection of the histogram of standardized residuals confirms that they all fell well within the standard range of −3 to +3, indeed they lied between −1 and +1, indicating the absence of apparent outliers or extreme values. Then, the leave-one out analysis demonstrated that effect sizes by specific domains remained consistent across iterations, with minimal variation compared to the model estimates. Overall, these findings suggest that our results are not unduly influenced by any single study and are robust across the dataset.

3.4. Sub-analyses of potential confounding variables

Meta-regression and sub-analyses revealed no significant association between potential confounding variables (i.e. mean age of CUD participants, % of females, % of CUD, % of AUD, frequency of cannabis use, duration of use and duration of abstinence) and global cognition were observed (Supplementary Table 5).

4. Discussion

In view of the controversy regarding the residual cognitive effects of cannabis, the current *meta*-analysis synthesized 23 cognitive studies involving individuals with CUD. Moderate impairments were observed in the case of speed of processing, verbal memory (learning) and working memory; deficits in executive functions were small-to-moderate; and deficits in impulsivity, attention and verbal fluency were small. A publication bias was observed. Meta-regression and sub-analyses revealed no effect on results of mean age of CUD participants, sex ratio, percentage of AUD and CUD participants, frequency of cannabis use, duration of use and duration of abstinence.

The main finding of the meta-analysis is that CUD is associated with residual cognitive deficits in the moderate range in the case of speed of processing, verbal memory (learning) and working memory. These results are highly coherent with cognitive impairments that have been described during acute Δ^9-THC administration. Indeed, a quantitative synthesis of 52 experimental randomized placebo-controlled studies showed that the acute administration of Δ^9-THC produces moderate impairments in verbal memory and working memory (Zhornitsky et al., 2021). The coherence between the results on the acute and residual cognitive effects of cannabis are important, as it demonstrates the pharmacological plausibility of the residual cognitive effects of cannabis. Likewise, the results of the current meta-analysis are coherent with the results of the functional neuroimaging literature, which have shown that both the acute administration of Δ^9-THC and the residual effects of cannabis use are associated brain activity alterations in prefrontal and temporal regions (Blest-Hopley, Giampietro, & Bhattacharyya, 2018; Gunasekera et al., 2022) which are known to be involved in executive functions and episodic memory, respectively (Clark, 2018; Friedman & Robbins, 2022). Another element to consider for the interpretation of our findings is that the amplitude of verbal memory and working memory impairments observed here is in a similar range as the deficits observed in alcohol, cocaine and methamphetamine use disorders ((Potvin et al., 2018; Potvin et al., 2014; Stavro et al., 2013); please refer to Supplementary Table 6). The similitude of findings between substances confirms the importance of paying attention to individuals with a CUD when studying the residual cognitive effects of cannabis. While some of the previous syntheses on recreational cannabis use have failed to show that cannabis can produce significant cognitive deficits (Scott et al., 2018), the current meta-analysis shows that such deficits can be detected in individuals with CUD.

One important difference between CUD and alcohol, cocaine and methamphetamine use disorders is that CUD is associated with less diffuse cognitive deficits. Indeed, our meta-analysis shows that CUD is associated with moderate deficits in speed of processing, verbal memory and working memory only, whereas AUD, cocaine and methamphetamine users disorders have been shown to be associated with moderate deficits in a wider range of cognitive domains ((Potvin et al., 2018; Potvin et al., 2014; Stavro et al., 2013); see Supplementary Table 6). From a pharmacological point of view, the relative specificity of findings observed in the studies on both the acute and residual cognitive effects of cannabis may seem surprising. In preclinical research, the cognitive deficits produced by cannabis have been attributed to the fact that Δ^9-THC binds to cannabinoid-CB_1 receptors as a partial agonist (Paronis,

Table 1
Results from the model including all 108 outcomes in a multivariate model by type.

N	Domain	Estimated combined effect-size				95 % C.I.		95 % P.I.	
		Estimate	se	t	p-value	Lower bound	Upper bound	Lower bound	Upper bound
11	**Attention**	**0.259**	**0.078**	**3.327**	**<0.001**	**0.106**	**0.411**	**−0.209**	**0.726**
2	Emotion recognition	0.221	0.127	1.741	0.08	−0.028	0.469	−0.286	0.727
14	**Executive functions**	**0.328**	**0.077**	**4.233**	**<0.001**	**0.176**	**0.480**	**−0.139**	**0.795**
11	**Impulsivity**	**0.281**	**0.078**	**3.607**	**<0.001**	**0.128**	**0.433**	**−0.187**	**0.748**
9	**IQ**	**0.497**	**0.088**	**5.671**	**<0.001**	**0.325**	**0.668**	**0.023**	**0.971**
7	**Speed**	**0.402**	**0.090**	**4.467**	**<0.001**	**0.226**	**0.579**	**−0.073**	**0.878**
7	**Verbal fluency**	**0.248**	**0.101**	**2.466**	**0.014**	**0.051**	**0.445**	**−0.236**	**0.732**
11	**Verbal learning**	**0.479**	**0.081**	**5.939**	**<0.001**	**0.321**	**0.638**	**0.01**	**0.948**
9	**Verbal memory**	**0.396**	**0.085**	**4.654**	**<0.001**	**0.229**	**0.563**	**−0.076**	**0.868**
4	**Visual learning**	**0.324**	**0.099**	**3.282**	**0.001**	**0.130**	**0.517**	**−0.158**	**0.806**
3	**Visual memory**	**0.294**	**0.131**	**2.238**	**0.025**	**0.037**	**0.551**	**−0.217**	**0.805**
5	Visuospatial	0.201	0.108	1.860	0.06	−0.011	0.412	−0.289	0.69
15	**Working memory**	**0.401**	**0.072**	**5.548**	**<0.001**	**0.259**	**0.542**	**−0.063**	**0.865**

	Heterogeneity				
Effects	Q	df	p-value	Variance (study)	sd (study)
Residuals	**199.71**	**95**	**<0.001**	0.0508	0.2253
Domain	19.74	12	0.07		
	Egger's test				
	Value	**t**		**df**	**p-value**
	2.61	**3.25**		**94**	**0.001**

Legend: SE = standard error; C.I. = confidence interval; P.I. = prediction interval; Df = degree of freedom; SD = standard deviation.

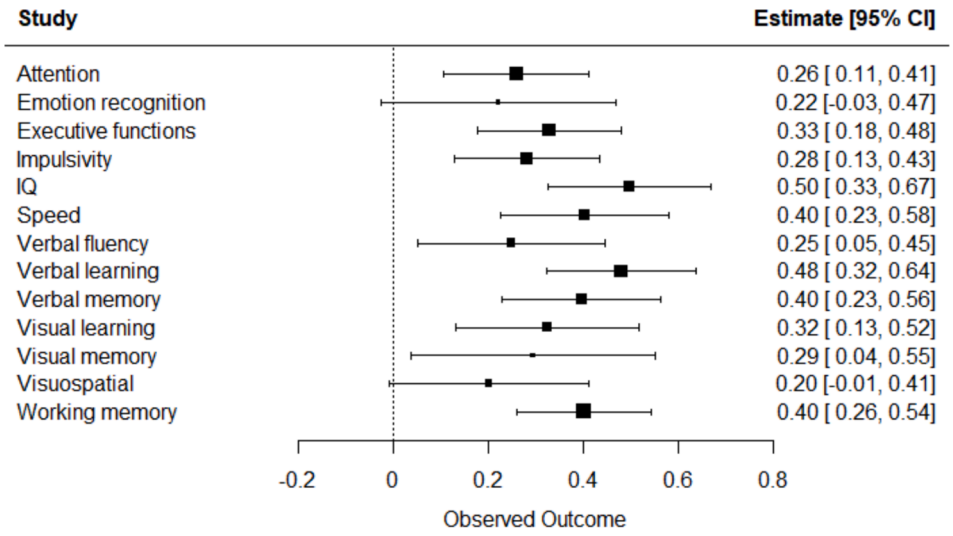

Fig. 2. Forest plot of the model by cognitive domains. Note: Study labels were removed because of the large number of studies.

Nikas, Shukla, & Makriyannis, 2012). The problem is that CB_1 receptors are among the most abundant throughout the brain (Kendall & Yudowski, 2016). Thus, if the effects of cannabis on cognition were solely explained by the binding of Δ^9-THC to CB_1 receptors, then, cannabis should be producing diffuse effects on several cognitive domains, which is not the case, as shown here. The discrepancy in results indirectly suggests that other cannabinoid receptors than CB_1 receptors are mediating the cognitive effects of cannabis.

The main strength of the current meta-analysis is that it is the first to examine the effects of CUD on several cognitive domains, using an appropriate number of studies (i.e. 23 studies, a notable increase as compared to the previous meta-analyses). In addition, the current meta-analysis used a system of classification of cognitive tests into cognitive domains that has been successfully used previously in several meta-analyses on the cognitive effects of several psychoactive substances by our research team (Potvin et al., 2018; Potvin et al., 2014; Stavro et al., 2013; Zhornitsky et al., 2021; Zhornitsky et al., 2022). This methodological homogeneity ensures consistency and simplifies comparisons between substances. Despite these strengths, our meta-analysis suffers from a few limitations that need to be acknowledged. The first is that the most studies included in the current meta-analysis employed a cross-sectional design, making it challenging to establish causality between CUD and the observed cognitive deficits. In theory, these deficits may have been present, at least in part, before the onset of cannabis use. Previous longitudinal studies on recreational use have carefully investigated the "chicken-and-egg problem". Thus far, verbal memory (learning) is the cognitive domain that has been found to be the most consistently impaired in longitudinal studies (for a review, see Bourque and Potvin (2021) (Bourque & Potvin, 2021)), a result clearly consistent with the main findings of the current meta-analysis. Regarding the causality issue, longitudinal studies have both shown that (i) cannabis and cognition share common genetic and environmental antecedents, and that (ii) cannabis produces a persisting cognitive decline, when adjusting for cognitive functioning prior to cannabis use (Castellanos-Ryan et al., 2017; Meier et al., 2012; Meier et al., 2018; Morin et al., 2019). Even though the current meta-analysis has included a reasonable number of studies ($n = 23$), some cognitive domains were only assessed in a small subset of studies ($n < 5$). This is the case of emotion recognition, visual learning and visual memory. For these cognitive domains, no firm conclusion can be reached. Another concern is that a publication bias was detected, meaning that our results may have been slightly overestimated. Another limitation concerns the fact that we found no effect of cannabis abstinence duration on results. This may be explained by the fact that only 5 studies assessed CUD participants abstinent for more than 25 days; in the other studies, abstinent duration was shorter than 10 days. It is therefore premature to conclude that abstinence has no effect on the cognitive impairments associated with CUD.

The current meta-analysis showed that CUD is associated with residual deficits in speed of processing, verbal memory (learning) and working memory of similar magnitude as the deficits observed in other substance use disorders. These deficits are generally larger than those that have been previously estimated in non-problematic cannabis users. The residual deficits observed here are coherent with the cognitive deficits observed during acute Δ^9-THC administration, meaning that they are pharmacologically plausible. Our results have important implications for public health, considering that the perceived risk associated with regular cannabis use has been declining in youths since the legalization of the substance (Duperrouzel, Granja, Pacheco-Colon, & Gonzalez, 2020). In the future, longitudinal studies on the residual cognitive deficits associated with CUD are warranted. In such studies, recruitment of large samples will be required, and cognitive assessments will need to be performed at baseline and after long periods of abstinence. Careful attention will need to be paid to both the duration and frequency of cannabis use. With the increase in cannabis potency over the years (Hasin, 2018), studies on the potential associations between cognitive impairments and Δ^9-THC concentrations in cannabis are also warranted. Finally, the functional impacts of the cognitive impairments associated with CUD will need to be determined.

CRediT authorship contribution statement

Florence Pilon: Methodology, Validation, Writing – original draft, Data curation. **Alexandre Dumais:** Supervision, Conceptualization. **Charles-Édouard Giguère:** Software, Formal analysis, Methodology. **Stéphane Potvin:** Supervision, Conceptualization, Methodology, Writing – original draft, Formal analysis.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.addbeh.2025.108434.

Data availability

Data will be made available on request.

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