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CANNABIS USE IN ADOLESCENTS WITH ADHD: IMPACT ON SYMPTOMS AND EXECUTIVE FUNCTION

USO DE CANNABIS EM ADOLESCENTES COM ADHD: IMPACTO NOS SINTOMAS E FUNÇÃO EXECUTIVA

USO DE CANNABIS EN ADOLESCENTES CON ADHD: IMPACTO EN LOS SÍNTOMAS Y FUNCIÓN EJECUTIVA

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ABSTRACT

Adolescents with attention-deficit/hyperactivity disorder (ADHD) have an elevated risk of early substance use and cannabis use disorder, while cannabis is frequently perceived as calming, sleep-promoting, or therapeutic. This narrative review examines whether cannabis improves ADHD symptoms and how use relates to attention, inhibition, working memory, planning, motivation, and everyday executive function. The evidence does not support cannabis as a treatment for ADHD. Acute tetrahydrocannabinol exposure impairs attention, learning, memory, reaction time, and inhibitory control; frequent adolescent use is associated with small-to-moderate cognitive disadvantages, although effect sizes, persistence, and causality vary across studies. Evidence specific to young people with ADHD is sparse. A systematic review found no consistent additive ADHD-by-cannabis deficit on neuropsychological tests, but the included studies were few, heterogeneous, and often underpowered; absence of a detected interaction should not be interpreted as safety. Cross-sectional comparisons are strongly confounded because executive dysfunction, impulsivity, conduct problems, family risk, sleep disturbance, and school disengagement can precede cannabis initiation. Longitudinal and co-twin studies indicate that both selection effects and

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exposure-related change contribute, with earlier onset, frequent use, high-potency products, and cannabis use disorder representing greater concern. Several abstinence studies demonstrate at least partial recovery of memory or executive performance over weeks. Clinically, assessment should separate intoxication, withdrawal, persistent ADHD, sleep problems, mood symptoms, and learning difficulties; quantify product, tetrahydrocannabinol content, route, frequency, and functional consequences; and screen confidentially for cannabis use disorder and safety risks. Evidence-based ADHD treatment should continue when indicated, with diversion precautions, motivational interviewing, family-supported behavioral care, cognitive-behavioral treatment for cannabis use disorder, and contingency management. Cannabis should not be recommended to manage ADHD symptoms in adolescents.

Keywords: Attention-Deficit/Hyperactivity Disorder. Cannabis. Adolescents. Executive Function. Cognition. Cannabis Use Disorder. Harm Reduction.

RESUMO

Adolescentes com transtorno do déficit de atenção/hiperatividade (TDAH) apresentam risco elevado de uso precoce de substâncias e transtorno do uso de cannabis, enquanto a cannabis é frequentemente percebida como calmante, promotora do sono ou terapêutica. Esta revisão narrativa examina se a cannabis melhora os sintomas de TDAH e como o uso se relaciona com atenção, inibição, memória de trabalho, planejamento, motivação e função executiva cotidiana. As evidências não apoiam a cannabis como um tratamento para o TDAH. A exposição aguda ao tetrahidrocanabinol prejudica atenção, aprendizagem, memória, tempo de reação e controle inibitório; o uso frequente na adolescência está associado a desvantagens cognitivas pequenas a moderadas, embora os tamanhos de efeito, persistência e causalidade variem entre os estudos. As evidências específicas para jovens com TDAH são escassas. Uma revisão sistemática não encontrou déficit aditivo consistente TDAH-por-cannabis em testes neuropsicológicos, mas os estudos incluídos eram poucos, heterogêneos e frequentemente com poder estatístico insuficiente; a ausência de uma interação detectada não deve ser interpretada como segurança. As comparações transversais são fortemente confundidas porque disfunção executiva, impulsividade, problemas de conduta, risco familiar, distúrbio do sono e desengajamento escolar podem preceder a iniciação do uso de cannabis. Estudos longitudinais e de co-gêmeos indicam que tanto efeitos de seleção quanto mudanças relacionadas à exposição contribuem, com início mais precoce, uso frequente, produtos de alta potência e transtorno do uso de cannabis representando maior preocupação. Vários estudos de abstinência demonstram pelo menos recuperação parcial da memória ou desempenho executivo ao longo de semanas. Clinicamente, a avaliação deve separar intoxicação, abstinência, TDAH persistente, problemas de sono, sintomas de humor e dificuldades de aprendizagem; quantificar produto, conteúdo de tetrahidrocanabinol, via, frequência e consequências funcionais; e rastrear confidencialmente o transtorno do uso de cannabis e riscos de segurança. O tratamento baseado em evidências para TDAH deve continuar quando indicado, com precauções de desvio, entrevista motivacional, cuidados comportamentais apoiados pela família, tratamento cognitivo-comportamental para transtorno do uso de cannabis e gerenciamento de contingência. A cannabis não deve ser recomendada para controlar os sintomas de TDAH em adolescentes.

Palavras-chave: Transtorno do Déficit de Atenção/Hiperatividade. Cannabis. Adolescentes. Função Executiva. Cognição. Transtorno do Uso de Cannabis. Redução de Danos.

RESUMEN
Los adolescentes con trastorno por déficit de atención/hiperactividad (TDAH) tienen un riesgo elevado de uso temprano de sustancias y trastorno por uso de cannabis, mientras que el cannabis frecuentemente se percibe como calmante, promotor del sueño o terapéutico. Esta revisión narrativa examina si el cannabis mejora los síntomas de TDAH y cómo el uso se relaciona con la atención, la inhibición, la memoria de trabajo, la planificación, la motivación y la función ejecutiva cotidiana. La evidencia no respalda el cannabis como tratamiento para el TDAH. La exposición aguda al tetrahidrocannabinol afecta la atención, el aprendizaje, la memoria, el tiempo de reacción y el control inhibitorio; el uso frecuente en la adolescencia se asocia con desventajas cognitivas de pequeñas a moderadas, aunque los tamaños de efecto, la persistencia y la causalidad varían entre estudios. La evidencia específica para jóvenes con TDAH es escasa. Una revisión sistemática no encontró un déficit aditivo consistente TDAH-por-cannabis en pruebas neuropsicológicas, pero los estudios incluidos fueron pocos, heterogéneos y frecuentemente con poder estadístico insuficiente; la ausencia de una interacción detectada no debe interpretarse como seguridad. Las comparaciones transversales están fuertemente confundidas porque la disfunción ejecutiva, la impulsividad, los problemas de conducta, el riesgo familiar, la perturbación del sueño y el desenganche escolar pueden preceder al inicio del uso de cannabis. Los estudios longitudinales y de co-gemelos indican que tanto los efectos de selección como los cambios relacionados con la exposición contribuyen, siendo el inicio más temprano, el uso frecuente, los productos de alta potencia y el trastorno por uso de cannabis motivo de mayor preocupación. Varios estudios de abstinencia demuestran al menos una recuperación parcial de la memoria o el desempeño ejecutivo a lo largo de semanas. Clínicamente, la evaluación debe separar la intoxicación, la abstinencia, el TDAH persistente, los problemas de sueño, los síntomas del estado de ánimo y las dificultades de aprendizaje; cuantificar el producto, el contenido de tetrahidrocannabinol, la vía, la frecuencia y las consecuencias funcionales; y examinar confidencialmente el trastorno por uso de cannabis y los riesgos de seguridad. El tratamiento basado en evidencia para el TDAH debe continuar cuando esté indicado, con precauciones de desviación, entrevista motivacional, cuidado conductual apoyado por la familia, tratamiento cognitivo-conductual para el trastorno por uso de cannabis y manejo de contingencias. El cannabis no debe recomendarse para controlar los síntomas de TDAH en adolescentes.

Palabras clave: Trastorno por Déficit de Atención/Hiperactividad. Cannabis. Adolescentes. Función Ejecutiva. Cognición. Trastorno por Uso de Cannabis. Reducción de Daños.

1 INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder defined by developmentally inappropriate inattention and/or hyperactivity-impulsivity that begins in childhood, occurs across settings, and causes functional impairment. Executive difficulties commonly involve response inhibition, working memory, planning, time management, emotional regulation, and persistence. These vulnerabilities intersect with adolescent novelty seeking, peer influence, expanding autonomy, and access to psychoactive substances. Cannabis use may therefore be both an outcome of ADHD-related risk and a potential contributor to further impairment.[1]

Adolescents often report that cannabis quiets thoughts, reduces restlessness, improves sleep, relieves boredom, or offsets adverse effects of prescribed stimulants. Subjective relief can be genuine without representing treatment of the underlying disorder. Intoxication may reduce arousal or distress while simultaneously worsening encoding, reaction time, monitoring, and goal-directed behavior. Withdrawal after frequent use can cause irritability, insomnia, anxiety, low appetite, restlessness, and concentration problems that resemble or amplify ADHD symptoms.

The clinical literature is difficult to interpret. ADHD precedes cannabis exposure in many cohorts and increases the probability of early initiation or problematic use; thus, poorer executive performance among users may reflect baseline liability. Cannabis exposure is also heterogeneous: frequency, duration, age at onset, tetrahydrocannabinol (THC) concentration, cannabidiol content, route, time since last use, and co-use of nicotine, alcohol, or other drugs vary widely. Studies that compare a frequent-use group with nonusers cannot automatically distinguish cause, consequence, and shared risk.

These uncertainties should produce calibrated counseling rather than false equivalence. There is no convincing evidence that cannabis is an effective adolescent ADHD treatment, whereas acute impairment and cannabis use disorder are established clinical risks. At the same time, claims that every exposed adolescent will sustain permanent executive damage exceed current evidence. The appropriate task is to integrate what is known, identify high-risk exposure patterns, and translate uncertainty into a careful diagnostic and therapeutic plan.

2 OBJECTIVE

To synthesize evidence on cannabis use in adolescents with ADHD, emphasizing perceived symptom effects, objective executive function, confounding and causality, clinical assessment, ADHD pharmacotherapy, treatment of co-occurring cannabis use, and practical risk-reduction counseling.

3 METHODS

A structured narrative review was performed using ADHD and cannabis position statements, systematic reviews and meta-analyses, prospective cohorts, co-twin studies, neuroimaging studies, abstinence trials, and treatment reviews available through September 2026. Searches combined ADHD, adolescent, cannabis, marijuana, THC, executive function, attention, memory, inhibition, abstinence, stimulant, and cannabis use disorder. Evidence directly involving adolescents or emerging adults with childhood ADHD was prioritized; broader adolescent cannabis research was used when disorder-specific data were unavailable. Results are presented by clinical question because exposure definitions and cognitive measures were too heterogeneous for a new pooled analysis. Associations are not described as causal unless the design supports temporal or within-person inference.

4 RESULTS AND DISCUSSION

4.1 ADHD AS A RISK FACTOR FOR CANNABIS USE AND DISORDER

ADHD is associated with earlier initiation and a higher probability of substance-related problems, particularly when symptoms persist and when conduct disorder, oppositional behavior, mood symptoms, school failure, family substance use, or peer risk are present. Meta-analytic longitudinal work has linked childhood ADHD with later nicotine and substance use disorders, including cannabis-related outcomes.[4,5] The pathway is not explained by one mechanism. Impulsivity can favor immediate reward, impaired prospective memory can undermine limits, emotional dysregulation can promote negative-reinforcement use, and academic or interpersonal difficulties can increase affiliation with substance-using peers.

A 2024 clinical epidemiologic meta-analysis estimated substantial current and lifetime prevalence of cannabis use disorder among people with ADHD, while also showing wide prediction intervals across settings.[3] These figures should not be transferred mechanically to every adolescent because included samples differed in age and referral intensity. They do,

however, justify routine, nonjudgmental screening in ADHD care rather than waiting for intoxication, school exclusion, or family conflict to reveal the problem.

The relationship is bidirectional at the symptom level. Children with weaker everyday executive control may be more likely to progress to frequent cannabis use, and frequent use can then interfere with learning, routines, sleep, and adherence to treatment. Prospective ratings of executive function have predicted later frequent use in some adolescent cohorts.[6] This means an observed association between cannabis and executive dysfunction may contain both vulnerability and exposure effects. Effective prevention begins with treatment of ADHD-related impairment before substance use becomes entrenched.

4.2 PERCEIVED BENEFIT, SELF-MEDICATION, AND ADHD SYMPTOMS

Self-medication is a useful hypothesis about motivation, not proof of therapeutic efficacy. Adolescents may experience immediate reductions in tension, perceived hyperactivity, or sleep latency. These effects can reinforce use, particularly after stressful days or stimulant offset. Yet a sedating or rewarding experience does not demonstrate improvement in sustained attention, organization, academic productivity, or safe decision-making. The distinction between feeling calmer and functioning better should be explored collaboratively.

Published evidence for cannabinoids as ADHD treatment is extremely limited and is concentrated in adults, not adolescents. Small experimental studies cannot establish developmental safety, and commercially available products differ from standardized research preparations. The Canadian ADHD Resource Alliance states that there is no evidence that cannabis effectively treats ADHD and advises clinicians to assess risk, provide education, and treat ADHD with established interventions.[2] No major pediatric ADHD guideline recommends THC- or cannabis-based products for core symptoms.

Cannabis can also create a symptom-maintenance loop. Evening use may initially facilitate sleep onset, but tolerance, late-night routines, withdrawal-related insomnia, and morning residual effects can worsen circadian stability. Reduced motivation or memory for tasks may be experienced as worsening ADHD, prompting more use for frustration. Likewise, intoxication-related anxiety or paranoia may be misread as a separate anxiety disorder, while withdrawal-related irritability may be attributed to stimulant medication or family conflict. Timeline-based assessment is essential.

Table 1

Common perceived benefits of cannabis and their clinical interpretation in adolescents with ADHD

Adolescent report	Possible short-term experience	Potential hidden cost	Clinical response
'It calms me down'	Reduced arousal, reward, or sedation	Slower reaction, weaker monitoring, avoidance of emotion-regulation skills	Compare calmness with task completion, safety, and next-day function
'It helps me focus'	Narrowed attention to an immediately rewarding activity	Poor divided attention, encoding, working memory, and sustained academic output	Use objective examples and propose a cannabis-free comparison period
'I need it to sleep'	Shorter perceived sleep latency	Tolerance, withdrawal insomnia, irregular timing, morning residual effects	Assess circadian pattern and treat sleep behavior directly
'It replaces my medication'	Avoidance of adverse effects or stigma	Untreated ADHD, variable THC dosing, cannabis use disorder	Reassess ADHD regimen and side effects; explain lack of therapeutic evidence
'It helps anxiety'	Temporary emotional dampening	Rebound anxiety, panic, paranoia, avoidance-based coping	Screen anxiety and psychosis risk; offer evidence-based treatment

The patient's experience should be validated without presenting cannabis as an evidence-based treatment.[2,7]
Source: Prepared by the authors.

4.3 ACUTE AND RESIDUAL NEUROCOGNITIVE EFFECTS

THC acts primarily through cannabinoid CB1 receptors that are widely expressed in neural systems involved in memory, salience, reward, and executive control. During adolescence, these systems are still maturing, with continuing changes in prefrontal networks, myelination, synaptic refinement, and connectivity. Biological plausibility for age-related vulnerability is therefore strong, but human developmental effects cannot be inferred from receptor distribution alone. Dose, prior exposure, product composition, sleep, expectancy, and testing conditions all modify performance.

Acute intoxication reliably impairs domains relevant to ADHD management: attention, reaction time, new learning, episodic memory, psychomotor coordination, and aspects of inhibitory or decision control. A meta-review of meta-analyses found small-to-moderate acute or residual deficits across executive function, learning and memory, attention, processing speed, and related domains, with overall evidence quality ranging from low to moderate.[10,24] For a student who already struggles with working memory and monitoring, even a modest transient decrement may have disproportionate functional consequences during driving, examinations, sports, work, or medication decisions.

Residual effects after intoxication are less uniform. A meta-analysis focused on adolescents and young adults found a small overall association between frequent or heavy cannabis use and cognitive performance; effect sizes were diminished in studies requiring more than 72 hours of abstinence.[9] This pattern is compatible with partial recovery, but it does not prove that all observed deficits are short-lived. Studies vary in their ability to verify abstinence, and heavy exposure, early onset, cannabis use disorder, or psychiatric comorbidity may define subgroups with greater or more persistent risk.

Neuroimaging studies sometimes show altered recruitment of executive, reward, or default-mode networks even when task accuracy is similar. Such findings may reflect compensation, residual drug effects, pre-existing differences, or exposure-related adaptation. A meta-analysis of functional imaging in cannabis-using youth reported differences across executive control, emotion processing, and reward systems, but heterogeneity by sex, severity, comorbidity, and abstinence limited a simple interpretation.[18] Imaging is informative at the group level and has no established role in diagnosing cannabis-related cognitive impairment in an individual adolescent.

4.4 EXECUTIVE FUNCTION IN ADOLESCENTS WITH BOTH ADHD AND CANNABIS EXPOSURE

Direct evidence for an interaction between ADHD and cannabis is sparse. A systematic review identified only 11 studies examining neurodevelopmental outcomes in adolescents or emerging adults with ADHD who used cannabis or synthetic cannabinoids. No included study demonstrated a consistent additive ADHD-by-cannabis interaction on neuropsychological executive tasks, although structural and functional imaging differences were reported and early-onset use appeared adverse in some analyses.[7] Small samples, retrospective exposure, variable abstinence, and selection of relatively high-functioning participants constrain the conclusion.

The best-known neuropsychological study followed young adults drawn from the Multimodal Treatment Study of ADHD. Childhood ADHD was associated with poorer executive performance, whereas adult cannabis-use history was not independently associated with broad executive deficits after covariate adjustment. Exploratory findings suggested that regular use beginning before age 16 might be linked to weaker performance.[8] This result argues against assuming universal synergistic impairment, but it also illustrates why broad group averages can miss timing- or dose-specific risk.

Executive function is not a single construct. Laboratory measures of inhibition, set shifting, planning, working memory, and decision-making correlate only modestly with one another and with everyday organization. ADHD may strongly affect parent- and teacher-rated daily executive behavior even when a brief task is normal. Cannabis may produce episodic impairment around use, interfere with school attendance or practice-dependent learning, and alter motivation without generating a large deficit on a decontextualized test. A clinically meaningful assessment therefore combines standardized tests, rating scales, history, and functional data.

The absence of a statistically significant interaction is not evidence that cannabis is protective or harmless in ADHD. Interaction tests require larger samples than tests of main effects, and exposure misclassification reduces power. Moreover, the outcome of concern is not limited to test score. Motor-vehicle crashes, psychosis vulnerability, panic, treatment nonadherence, legal or school consequences, and progression to cannabis use disorder may occur even if measured executive performance appears unchanged.

Table 2

Executive-function domains and the current interpretation of cannabis-related findings

Domain	Possible cannabis-related finding	Major interpretive problem	Clinical marker
Attention and processing speed	Reduced sustained attention or slower responding during intoxication and early abstinence	Sleep loss, ADHD severity, nicotine and recent use	Missed instructions, driving errors, slowed schoolwork
Working memory and learning	Weaker encoding and recall; improvement reported during abstinence	Practice effects and incomplete abstinence verification	Forgetting new material, repeated prompts, lost task steps
Inhibitory control	More errors or altered neural recruitment in some studies	Impulsivity may predate initiation	Use despite consequences, unsafe decisions, difficulty stopping
Planning and flexibility	Small or inconsistent group differences	Laboratory tasks may not capture daily organization	Late assignments, poor sequencing, failure to adapt plans
Reward decision-making	Preference for immediate reward and altered reward processing	Both a vulnerability for and possible consequence of use	Escalating use, abandoned long-term goals, risky peer contexts

ADHD-specific interaction evidence is limited. General adolescent evidence supports acute impairment and small average residual effects, with substantial heterogeneity.[7–10]
Source: Prepared by the authors.

4.5 LONGITUDINAL EVIDENCE, CAUSALITY, AND RECOVERY

Prospective studies strengthen temporal inference but remain vulnerable to time-varying confounding. In a cohort followed from early adolescence to young adulthood, poorer short-term and working memory preceded cannabis onset, while greater cannabis frequency subsequently predicted decline in verbal IQ and trial-and-error learning and lower high-school graduation rates.[11] This pattern supports a bidirectional model in which neurocognitive vulnerability influences use and exposure contributes to selected outcomes.

Co-twin control designs address shared family and genetic factors. In one large longitudinal twin study, adolescents who used cannabis had poorer executive performance at age 18, but many associations were attenuated within twin pairs, suggesting that familial background explains an important portion of the population-level difference.[12] Such attenuation does not prove that exposure has no effect: twins can differ in other experiences, dose measurement is imperfect, and within-pair analyses may have limited power. It does warn against attributing every deficit to cannabis.

Earlier onset and frequent use remain concerning. Adults reporting chronic use before age 15 performed more poorly on several executive measures than later-onset users and controls in a cross-sectional study, although pre-use cognition could not be established.[13] Longitudinal work has associated early adolescent use with later attentional and inhibitory-control difficulties after accounting for several covariates.[14] These studies support delaying initiation as a concrete prevention goal even while causal magnitude remains uncertain.

Abstinence studies offer clinically useful information. Adolescents and young adults randomized or encouraged to sustain abstinence have shown improvement in verbal learning or memory, often within the first weeks.[15] A 2025 randomized contingency-management study found signals of improvement in executive performance after four weeks of verified abstinence and suggested convergence toward nonusing peers on selected measures.[16] Earlier work also demonstrated change across three weeks of abstinence, although some subtle deficits have been detected after approximately one month.[17],[19] The overall message is hopeful: cognitive complaints should prompt a monitored abstinence trial rather than a deterministic prediction of permanent injury.

4.6 CLINICAL ASSESSMENT

Cannabis screening should occur privately with the adolescent, with confidentiality and its safety limits explained before questions begin. Ask about age at first use; days used in the

past month; use before school, work, sports, or driving; route; approximate THC and cannabidiol content; concentrates or synthetic cannabinoids; quantity; source; co-use with nicotine, alcohol, sedatives, or stimulants; motives; tolerance; withdrawal; unsuccessful efforts to cut down; and consequences. Frequency alone is insufficient because a high-potency concentrate used weekly may carry different risks from a lower-potency product.[25]

Assessment should distinguish four states: acute intoxication, withdrawal, early abstinence, and stable baseline. Inattention, slowed processing, or poor recall during intoxication should not be used to adjust the ADHD diagnosis. Irritability and insomnia during the first days of abstinence may transiently worsen concentration. Conversely, ADHD that clearly began before cannabis exposure and persists during verified abstinence should not be dismissed as substance-induced. Collateral information and childhood records are often decisive.

Functional questions are more actionable than a debate about whether cannabis has damaged the brain. Has the adolescent missed classes, abandoned activities, driven after use, lost money, changed peers, experienced panic or paranoia, failed to take medication, or become unable to sleep without cannabis? School attendance, grades, late assignments, employment, family conflict, accidents, and risky sexual behavior provide a longitudinal outcome map. Screening should also cover depression, suicidality, psychotic symptoms, trauma, anxiety, conduct problems, eating difficulties, and sleep disorders.

Urine testing can support a collaboratively agreed abstinence trial or treatment program but should not replace history or be used punitively. THC metabolites can remain detectable long after impairment has resolved, especially with frequent use, and a positive result does not establish time of use. Unexpected results require discussion of assay limits, prescribed products, and the therapeutic purpose of testing. Cognitive testing is considered when impairment persists after a meaningful abstinence period or when learning disorder, traumatic brain injury, or another neurodevelopmental condition is suspected.

Table 3

Structured clinical approach to co-occurring ADHD and cannabis use

Step	Core assessment	Action
Establish timeline	ADHD onset, cannabis onset, recent use, withdrawal, abstinence periods	Avoid diagnosing or titrating solely during intoxication or acute withdrawal
Quantify exposure	Days/month, product and THC, route, concentrates, co-use, use before school or driving	Identify early onset, frequent use, high potency, and unsafe contexts
Assess disorder and risk	Craving, tolerance, withdrawal, loss of control, consequences, psychosis, suicidality	Diagnose cannabis use disorder when criteria are met; escalate urgent risk
Measure function	Attendance, grades, work, sleep, relationships, driving, medication adherence	Select concrete outcomes and obtain collateral information with consent
Treat both conditions	ADHD impairment and substance-use goals	Use evidence-based ADHD care plus motivational, cognitive-behavioral, family, and contingency approaches
Reassess	Use, symptoms, cognition, side effects, diversion, agreed outcomes	Adjust intensity; consider neuropsychological assessment after meaningful abstinence if deficits persist

Confidentiality, developmental capacity, family involvement, and local legal requirements should be addressed explicitly.
Source: Prepared by the authors.

4.7 MANAGEMENT OF CO-OCCURRING ADHD AND CANNABIS USE

Management begins with a shared formulation: what the adolescent seeks from cannabis, what costs have emerged, and which goals matter now. Motivational interviewing is particularly suitable when readiness to stop is low. The clinician elicits perceived benefits and harms, reflects discrepancies between use and goals, and offers a menu of changes rather than conducting a confrontation. Family involvement improves monitoring and reinforcement when developmentally appropriate, while preserving private clinical space for honest disclosure.

For cannabis use disorder, evidence-supported psychosocial approaches include cognitive-behavioral therapy, motivational enhancement, family-based treatment, and contingency management. A systematic review of adolescent interventions found benefits across cognitive-behavioral and combined models but substantial heterogeneity, with no single universally superior protocol.[20] Treatment should address triggers, refusal skills, alternative rewards, sleep, peer networks, and relapse planning. Co-occurring nicotine dependence often sustains cannabis routines and requires direct treatment.

ADHD should continue to be treated with established interventions.[23] Untreated symptoms can undermine substance-use treatment, but prescribing requires an individualized risk assessment. Long-acting stimulants may reduce opportunities for misuse compared with immediate-release preparations; supervised dosing, limited quantities, secure storage, and monitoring for diversion are practical safeguards. Atomoxetine, guanfacine extended release, or clonidine extended release may be considered when stimulant risks outweigh benefits, recognizing that nonstimulants are not treatments for cannabis use disorder.

Evidence does not show that appropriately prescribed stimulant treatment inevitably causes later substance use disorder. Longitudinal and meta-analytic findings are mixed regarding protection, but they do not support withholding effective ADHD treatment solely because of a theoretical future substance risk.[21],[22] In adolescents with active substance use, medication response, adherence, intoxication, diversion, cardiovascular risk, and psychiatric symptoms should be reviewed more frequently. A stimulant should not be taken in a way intended to counteract cannabis intoxication or sleep loss.

If immediate abstinence is not acceptable, harm reduction can reduce acute risk while engagement continues. The clinician should advise against driving or riding with an impaired driver; use before school, work, sports, water activities, or operating equipment; high-THC concentrates; synthetic cannabinoids; mixing with alcohol or sedatives; sharing vaping devices; and using alone when severe anxiety or psychosis has occurred. Delaying initiation, reducing frequency, avoiding morning use, and building cannabis-free days are meaningful intermediate goals, not endorsements of use.

4.8 HIGH-RISK PRESENTATIONS

Urgent assessment is required for psychosis, severe panic, suicidality, marked agitation, chest pain, syncope, persistent vomiting, altered consciousness, or unsafe behavior. Synthetic cannabinoids are not pharmacologically equivalent to cannabis and may produce severe agitation, seizures, cardiovascular toxicity, kidney injury, or psychosis. Edible products create delayed onset and redosing risk; concentrates can deliver very high THC exposure. Product labels may be inaccurate, and illicit products may contain contaminants.

A personal or first-degree family history of psychotic disorder, prior cannabis-induced psychosis, bipolar disorder with mania, severe anxiety reactions, or recurrent hyperemesis substantially changes counseling. So do pregnancy, major cardiovascular disease, and use

of sedatives. The threshold for recommending complete avoidance should be low in these groups. Clinicians should ask specifically about paranoia and perceptual change because adolescents may not volunteer these symptoms.

Driving deserves explicit attention. ADHD itself is associated with driving risk, and cannabis impairs reaction time, tracking, divided attention, and judgment. Combining the two vulnerabilities, especially with sleep deprivation or alcohol, is clinically dangerous even when the adolescent reports tolerance. Counseling should be concrete and documented, with family safety planning consistent with local law and confidentiality requirements.

4.9 EVIDENCE GAPS AND INTERPRETATION

The central evidence gap is not whether intoxication can impair cognition; it can. The unresolved questions concern the magnitude and persistence of deficits from specific adolescent exposure patterns, whether ADHD modifies susceptibility, and how much recovery occurs after abstinence. Existing ADHD-specific studies are small, frequently retrospective, and often extend into young adulthood. Modern products with higher THC concentrations are not well represented in older cohorts.

Future research should measure cognition before initiation, use repeated biological and self-report exposure measures, quantify THC and cannabidiol, distinguish vaping, combustion, and edibles, and model nicotine and alcohol co-use. ADHD diagnosis should be well characterized, including presentation, persistence, medication exposure, conduct problems, sleep, and everyday executive function. Within-person change and genetically informed designs are more informative than simple user–nonuser comparisons.

Trials of abstinence are especially valuable because they answer a question patients care about: whether functioning improves when use stops. Studies should pair objective cognitive tests with school performance, driving-related skills, sleep, mood, and daily organization. Until such evidence is stronger, clinicians should communicate a dual conclusion: causal estimates remain uncertain, but cannabis is not an evidence-based ADHD treatment and frequent adolescent use creates preventable cognitive and functional risk.

5 CONCLUSION

Adolescents with ADHD are a high-priority population for cannabis prevention, screening, and early intervention. Cannabis may be experienced as calming or sleep-promoting, but subjective relief does not equal improvement in core ADHD symptoms or

executive function. Acute THC impairs several domains already vulnerable in ADHD, and frequent early use is associated with poorer cognitive and educational outcomes. Direct evidence for an additional ADHD-by-cannabis neuropsychological effect is insufficient, not reassuring.

Clinical care should separate baseline ADHD from intoxication and withdrawal, characterize exposure and consequences, screen for cannabis use disorder and psychiatric risk, and offer a monitored abstinence period when cognitive concerns are present. Evidence-based ADHD treatment, motivational and family interventions, cognitive-behavioral care, and contingency management should be integrated. The most accurate recommendation is clear: cannabis should not be prescribed or recommended to treat ADHD in adolescents, and delaying initiation or reducing and stopping use offers the best opportunity to protect learning, safety, and development.

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