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Review Article

The Effects of the Most Commonly Used Recreational Substances on Key Cognitive Functions

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Abstract

Despite the known risks of recreational substance use, it remains widespread worldwide, with millions of individuals using substances like cannabis, cocaine, and MDMA each year. These substances primarily affect neurotransmitter systems, including the endocannabinoid, serotonergic, and dopaminergic systems, which are integral to cognitive functions. This review examines both the acute and long-term effects of cannabis, cocaine, and MDMA on key cognitive domains, specifically retrospective memory, prospective memory, and executive functions. After briefly reviewing the neurochemical mechanisms underlying their actions in the brain, the review provides an overview of the cognitive impairments linked to these substances. However, the interpretation of these findings is complicated by research challenges such as polydrug use, participant recruitment issues, poor control of confounds, and difficulties in establishing causality.

Keywords: Cannabis; Cocaine; Cognition; Episodic memory; Executive functions; MDMA; Prospective memory; Recreational substance use; Retrospective memory

Introduction

Recreational substances refer to chemical substances used casually and without dependence, primarily for enjoyment rather than for medical purposes. While substances like alcohol, tobacco, and caffeine can also fall under this category, this review focuses specifically on cannabis, MDMA, and cocaine. The recreational use of these substances are illegal in many countries, including the United Kingdom. Despite the risks, illegal recreational substance use remains high worldwide. According to the United Nations Office on Drugs and Crime's World Drug Report 2022, 284 million people aged 15-64 used substances at least once in the previous year, up from 226 million in 2010 (26% increase). This is around 1 in every 18 persons, or 5.6% of the population of the world in that age group. The Report further noted that substance use is much more common among young people [1].

The most commonly used illegal recreational substance is cannabis, followed by cocaine, MDMA [2]. Therefore, this review primarily explores the cognitive effects of cannabis, cocaine, and MDMA on key cognitive domains such as memory and executive functions. These substances are thought to affect cognition by disrupting neurotransmitter systems essential for these cognitive processes [3]. The review begins with an overview of the neurochemical pathways through which these substances influence brain function, followed by a summary of the literature on their cognitive effects. While a comprehensive analysis is beyond the scope of this review, the following overview aims to provide insight into the mechanisms of action of these substances and their potential impact on cognitive functioning.

Effects of Commonly Used Substances on the Brain

Substances affect the brain primarily by interacting with various neurotransmitter systems. While their exact mechanisms are not fully understood, advanced research techniques have provided valuable insights. This section explores how the most commonly used recreational substances (namely cannabis, MDMA, and cocaine) alter neurotransmitter activity and their potential connections to cognitive functions.

Cannabis

Cannabis, typically smoked, releases compounds like Δ9-tetrahydrocannabinol (THC; the primary psychoactive component) and cannabidiol (CBD; a non-psychoactive, medically used compound) into the bloodstream [4,5]. While its exact mechanisms remain unclear, cannabis may impact cognition by interacting with the Endocannabinoid System (ECS), which includes endogenous cannabinoids, receptors (CBs), and enzymes [6]. The ECS regulates neurotransmitter systems, including dopamine [7,8]. THC primarily activates CB receptors [9,10], which are densely located in brain regions like the hippocampus, amygdala, basal ganglia, and prefrontal cortex [11,12]. Disruption of the ECS may affect neurobehavioral processes such as learning, memory, motivation, motor control, reward processing, and executive functions [13-16]. Neuroimaging studies support this, showing reduced Prefrontal Cortex (PFC) size and activity in heavy cannabis users, linked to the disruption of endocannabinoid-mediated synaptic plasticity [17-20]. Animal studies further corroborate these findings [21-23].

MDMA

MDMA (3,4-Methylenedioxymethamphetamine) is a synthetic stimulant derived from amphetamine, commonly found in crystallized

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or tablet form. The powder form, typically referred to as MDMA, usually contains pure MDMA, while the tablet form, known as ecstasy or Molly, is often mixed with various other substances. MDMA primarily affects neurotransmitter systems by promoting serotonin (5-HT) release from presynaptic neurons, reversing the serotonin transporter's which normally recycles serotonin), and subsequently increasing 5-HT availability at postsynaptic receptors [24,25].

Although its precise mechanisms remain unclear, MDMA primarily influences cognition through the serotonergic system, which plays a key role in memory, attention, and perception [26,27]. For example, serotonin depletion, induced through acute tryptophan depletion, has been shown to impair memory [28-31], whereas increasing serotonin levels via selective serotonin reuptake inhibitors is linked to memory improvements [32]. Long-term MDMA use is associated with reduced serotonin signalling, as neuroimaging studies reveal decreased Serotonin Transporter (SERT) binding in the frontal, parietal, and temporal lobes [33-36]. A meta-analysis confirmed widespread reductions in SERT binding in the brain [37,38]. Those findings might explain the observed cognitive impairments in people with a history of MDMA use. Animal studies further support MDMA-induced serotonergic disruption and memory impairments [39,40]. Additionally, MDMA interacts with the glutamatergic and dopaminergic systems [41-44], which may contribute to broader cognitive deficits, particularly in learning and memory.

Cocaine

Cocaine is a stimulant substance derived from the Erythroxylum coca plant, available in forms like powdered cocaine (snorted) and crack cocaine (smoked or injected). Cocaine exerts its effects by blocking or slowing down the monoamine transporters, in particular those for Dopamine (DA). This action primarily increases DA levels, as well as serotonin and Norepinephrine (NE) in the brain [45]. As a result, cognitive deficits related to cocaine use are believed to arise from disruptions in the DA system, which plays a crucial role in memory, executive functions, and attention. For instance, DA neurons increase firing in response to salient stimuli in areas like the hippocampus and Ventral Tegmental Area (VTA), which are involved in encoding new, episodic-like memories [46]. Furthermore, Otmakhova and Lisman found that dopamine receptors (D1 and D5) enhance glutamatergic transmission, facilitating memory encoding [47]. Animal studies support this, with Li et al., showing that DA levels rise when animals encounter new environments, but this improved memory is lost when hippocampal DA receptors are blocked [48]. Additionally, DA is known to regulate executive functions, particularly working memory [49,50].

Cocaine has been shown to reduce DA neuron activity, leading to decreases in DA release and DA receptors' functions [51]. This suggests that cocaine-induced cognitive impairments may primarily result from disruptions to the dopamine system. It is important to note that several studies have argued that cocaine-related cognitive deficits are driven by long-lasting neuroplastic changes in Prefrontal Cortex (PFC) circuitry, rather than direct cell damage or neurotoxicity [52,53]. Additionally, cocaine use has been associated with significant white matter changes in the brain [54-56].

Challenges in Conduction Studies on the Effects of Substance Use on Cognition

It is important to highlight the challenges involved in conducting studies on the effects of substance use before reviewing the relevant literature, as these challenges must be considered when interpreting the findings.

Polysubstance use

Polysubstance use, or the combined use of multiple substances, is common among people with a history of substance use [57]. Individuals often combine substances to achieve specific effects, such as the combination of cocaine and ketamine produces intense euphoric highs along with hallucinations [58]. They may also mix substances to counteract negative effects, like using cannabis to help sleep after consuming stimulants like MDMA or cocaine [59]. The widespread availability of various substances further encourages polysubstance use [60]. Polysubstance use complicates studies on substance-related cognitive effects, as different substances interact through distinct neural mechanisms, making it difficult to isolate the impact of one substance [61]. Researchers have attempted to address this by examining ecstasy users with minimal other substance use [62] or comparing MDMA polysubstance users to non-users [63], but these strategies still struggle to fully capture the complex interactions of multiple substances.

Difficulties to recruiting participants

Another challenge is recruiting substance-using populations who are difficult to reach and may actively conceal their identities due to concerns about legal consequences [64]. The illegal status of many substances and the threat of imprisonment can discourage individuals from participating in research [65]. For instance, in the UK, illicit substances are classified into four categories, each carrying different penalties for possession and distribution. Possessing Class B substances (e.g., cannabis), and Class A substances (e.g., cocaine, MDMA) can result in up to 5, or 7 years in prison, respectively, along with fines. Consequently, many studies suffer from small sample sizes (often averaging around 30 participants; [66]), limiting the ability to generalise findings.

The causality between substance use and cognitive impairments

Most studies examining the long-term effects of substance use on cognition rely on cross-sectional designs, which collect data from participants at a single point in time. These studies lack prospective or retrospective follow-up, making it challenging to draw causal conclusions between substance use and cognitive impairments. It is possible that cognitive deficits may be present prior to first substance use which can act as a risk factor for substance use initiation [67].

Poor control for confounding variables

Many studies neglect to account for potential confounding variables such as age, education, depression, sleep quality, and IQ, all of which have been shown to impact cognitive functions [68-76]. In line with this, a comprehensive review highlights that the majority of studies in this area have not properly controlled for these critical confounds [53].

Issues in defining and measuring levels of illicit substance use

Inconsistent definitions and measurements of illicit substance use, such as frequency, duration, dosage, age at first use, and total lifetime consumption pose a significant challenge to drawing definitive conclusions about their effects. The lack of a standardised framework to classify substance use as heavy, moderate, or light further complicates the interpretation of findings across studies. For example, the same level of ecstasy use (e.g., 400 tablets) may be classified as "heavy" in one study [77] and "moderate" in another [78]. This lack of consistency creates confusion and hinders the ability to make clear, comparable conclusions across research on the effects of illicit substance use.

Lack of substance-naïve control groups

Many studies on the cognitive effects of illicit substance use lack substance-naïve control groups. Instead, the non-user groups often include individuals who have used other substances, such as cannabis. While statistical methods can control for the influence of these substances, it is impossible to eliminate their effects entirely. This limitation introduces potential confounding factors, making it difficult to attribute observed cognitive deficits solely to the substance under investigation. As a result, findings may be less conclusive, and the true extent of substance-specific effects on cognitive functions remains unclear.

Generalisability of study findings

Most studies on substance use have been conducted in industrialised nations, such as the United Kingdom and the United States [79], with predominantly white participants. However, substance use prevalence has been reported as higher among individuals from non-white ethnic backgrounds, such as those of mixed race [80]. This may reflect underreporting among non-white individuals, potentially influenced by racial bias within the criminal justice system, where substance-related arrests disproportionately affect minority groups. For instance, Black, Asian, and minority ethnic individuals are 240% more likely to be imprisoned for substance-related offences compared to white offenders [81]. It has been suggested that substance use may have different, potentially more severe, impacts on minority groups [82], though this remains under-researched. Consequently, the findings from these studies may not be fully generalisable beyond white/western populations.

Effects of Commonly Used Substances on Cognitive Functions

Establishing a direct link between recreational substance use and cognitive impairment is complex due to various factors. However, this section aims to clarify substance-induced cognitive changes by reviewing key literature on human participants, providing insights into how these substances may disrupt cognitive processes. While animal studies offer valuable insights, the focus here is on human studies to better understand the real-world impact of substance use on cognition. Evidence on the possible effect of recreational substance use on cognitive functions comes from two lines of research: retrospective studies and placebo-controlled studies. In placebo-controlled studies, participants receive either a dose of the substance or a placebo to measure its acute effects on cognition, providing insights into how substances affect neurocognitive functioning during intoxication. However, it is important to note that these studies often include people with regular or occasional substance use, with substance-naïve controls rarely being included. Retrospective studies, on the other hand, compare current or abstinent substance users with drug-naïve individuals or non-specific substance users (e.g., people with a history of cocaine use vs. those with non-cocaine polysubstance use), offering valuable insights into long-term cognitive deficits that may persist even after acute intoxication has subsided.

Cognitive functioning encompasses a range of mental processes involved in acquiring knowledge, processing information, and reasoning. It includes abilities such as learning, memory, attention, and problem-solving- skills essential for navigating daily life, making decisions, and interacting with the world. The relevant literature will be discussed under three subheadings: retrospective memory, prospective memory, and executive functions.

Retrospective memory

Memory is one of the most crucial cognitive functions in a person's life for tasks like communication, learning, and developing personality. It involves encoding, storing, and recalling information [83]. Information is encoded through perception and association with prior knowledge, then stored for later retrieval. Memory is often classified under two broad concepts: retrospective and prospective memory. Retrospective memory involves memory of events, people or experimental stimuli that were experienced in the past, such as remembering the detail of a friend's birthday party or recalling a list of words presented in an experiment. Whereas, prospective memory involves remembering to carry out a planned action or recall a planned intention at some future point in time. When studying explicit forms of retrospective memory, particularly objective episodic memory, participants are typically asked to learn specific materials (e.g., a list of words). After a predefined delay, they are then prompted to intentionally retrieve the learned information through free recall, cued recall, or recognition tasks. Retrospective memory can also be assessed via autobiographical memory tests, which focus on evaluating subjective episodic memory.

The following sections will first review the literature on the acute and long-term effects of commonly used illicit substances on objective episodic memory and then subjective episodic memory.

Objective episodic memory via learning tests

Acute effects

Cannabis

Cannabis has been extensively studied for its acute effects on learning and memory where participants were given a single dose of THC and tested. Numerous studies have consistently demonstrated that THC can impair learning and memory processes [84-90]. Additionally, several review papers further supported the evidence for THC's negative impact on learning and memory [8,17,91-94]. As aforementioned, cannabis contains two main active compounds: THC, responsible for its psychoactive effects, and CBD, which appears to counteract some of THC's cognitive impairments. For instance, Englund et al., found that pre-treatment with CBD reduced THC-induced cognitive deficits. In their study, participants who received oral CBD before intravenous THC performed better on verbal learning tasks, compared to those given a placebo [85]. Similarly, Morgan et al., reported that individuals using low-CBD cannabis showed poorer performance on verbal learning tasks compared to those using high-CBD strains [95]. These findings suggest that high-THC/low-CBD cannabis is associated with greater cognitive dysfunction.

A recent report showed that THC potency has increased significantly over recent decades, rising from 4% in 1995 to 9.75% in 2009, and 13.88% in 2019, while CBD content has remained comparatively low, rising only from approximately 0.28% in 2001 to 0.39% in 2009, and 0.56% in 2019 in the USA [96-98]. A United Nations report further shows that cannabis potency has quadrupled in parts of the world over the past 24 years [1]. This trend raises concerns about the potential for more severe cognitive impairments in current cannabis users, especially with high-THC, low-CBD strains.

MDMA

Although research on the acute effects of MDMA on learning and memory is limited, existing studies consistently suggest a negative impact. For example, Kuypers et al., reported significant impairments in verbal memory performance on the Word Learning Task following MDMA administration [99]. These findings have been consistently replicated, with MDMA-intoxicated individuals performing worse on verbal learning tasks compared to placebo controls [100-104]. Most of these studies were conducted in controlled, daytime laboratory settings, which may limit their ecological validity. However, a few studies have examined the effects of MDMA on verbal learning during real-world nighttime settings, yielding similar results [105,106]. A recent review further confirmed MDMA's negative impact on memory, with 16 studies reporting acute memory impairments across 23 different tasks [107].

Cocaine

Research on the acute effects of cocaine on learning and memory is limited [108]. Existing studies have found no significant impact on learning following intranasal cocaine administration in recreational users [109-111].

Conclusion

In summary, the acute effects of recreational substances on objective episodic memory differ across substances. Cannabis, particularly due to THC, consistently impairs learning and memory, with higher THC and lower CBD levels linked to greater deficits. MDMA also negatively impacts learning and memory both in controlled laboratory settings and real-world environments. In contrast, acute cocaine use appears to have no effects on learning and memory, though research on this is limited.

Long-term effects

Cannabis

Existing literature on the long-term cognitive effects of cannabis use predominantly relies on cross-sectional studies, where cannabis users, after a period of abstinence, are compared to non-users on cognitive performance. These studies consistently report that regular cannabis users who have been abstinent for periods ranging from 12 hours to 21 days demonstrate poorer immediate and delayed recall [112-119]. Multiple reviews suggest that long-term or heavy cannabis use impairs key memory processes, including encoding, storage, manipulation, and retrieval [8,17,91,92,120-122]. These impairments are closely linked to the frequency, duration, quantity, and age of onset of cannabis use [91,118,122,123].

However, not all studies align with these findings. A small number of investigations have reported no significant association between cannabis use and learning deficits [124-126]. Given the limitations of cross-sectional designs, which may reflect individual factors rather than direct effects of cannabis use, the need for longitudinal studies becomes apparent. Longitudinal designs can track cognitive changes over time, offering stronger evidence for causal relationships between cannabis use and cognitive outcomes. For example, a study of chronic daily adolescent-onset cannabis users showed that verbal memory deficits observed at baseline persisted after two years of continued heavy use [127,128]. Another longitudinal study also found that greater cannabis use was associated with poorer episodic memory, especially for immediate recall [129]. In line with these findings, a review of longitudinal studies showed that episodic memory performance was the measure most likely affected by persistent cannabis use [130].

While the evidence points to cannabis-related cognitive impairments, there is growing consensus that these effects may be reversible following extended abstinence. For instance, Medina et al., found that after 30 days of abstinence, cannabis users exhibited only subtle deficits in episodic memory compared to controls [131]. Similarly, Hanson et al., observed significant improvements in verbal learning among adolescent cannabis users after two weeks of abstinence [114]. A meta-analysis by Schreiner and Dunn further supports this notion, reporting no significant cognitive deficits in individuals abstinent from cannabis for at least 25 days across various domains, including learning, and memory [132].

More recent research by Burke et al., examined cognitive performance in heavy cannabis users after periods of abstinence ranging from 3 days to over 90 days. The results indicated that cognitive impairments were detectable during short-term abstinence but largely remitted after 90 days [133]. Similarly, Fried et al., found that learning deficits in young adults who were former heavy cannabis users were no longer apparent three months after cessation of use [134]. These results suggest that the brain may adapt to or compensate for the effects of chronic cannabis use over time. However, the extent of recovery may depend on factors such as lifetime use and dosage, with high lifetime use potentially limiting recovery [135].

MDMA/Ecstasy

Similar to cannabis studies, MDMA studies also primary used cross sectional study design to investigate long-terms effects of MDMA on learning and memory. Multiple studies found the similar results where MDMA users impaired in learning tasks [63,121,124,125,136-140]. Furthermore, it has been well documented that there is a dose-dependent association between ecstasy use and poorer verbal learning and memory abilities where people with moderate to heavy ecstasy/polysubstance use performed significantly worse on learning tasks than those with very mild ecstasy/polysubstance use or drug naïve controls [136,141–144]. Furthermore, a limited number of longitudinal studies have been conducted to assess the relationship between ongoing MDMA use and cognitive performance in novice MDMA users, addressing the methodological limitations of cross-sectional designs. In one such study, the group of incident Ecstasy users showed significantly greater declines in immediate and delayed recall and recognition compared to persistent Ecstasy-naive participants [62]. Such a decline on learning and memory abilities in new MDMA users was evident in other follow-up studies [145]. However, another follow-up study found no significant changes in cognitive performance among MDMA users, controlling for potential confounders such as age, sleep patterns, subjective well-being, recent medical treatments, sports participation, nutrition, and general intelligence [146].

A comprehensive review by Kalechstein et al., assessed the effects of MDMA on learning and memory using two approaches: one with stringent inclusion/exclusion criteria to match participants on key moderator variables and another with more lenient criteria. Analysis of 11 studies using the stricter criteria and 23 studies with more flexible criteria both revealed that MDMA use is associated with impairments in learning and memory [147]. These findings were further supported by a meta-analysis of 21 studies, which found significant negative effects of MDMA use on learning and memory [148].

Several studies have also examined whether cognitive impairments from MDMA use persist after cessation. Reneman et al., found that both recent and former MDMA users (abstinent for over 12 months) performed worse than controls on learning tasks [36]. Similarly, other studies reported poor learning performance in former users [149,150], suggesting that the effects of MDMA may be long-lasting. A follow-up study by Thomasius et al., indicated that memory impairments could persist for over 2.5 years after cessation [151]. These findings align with a review by Klugman and Gruzelier, which concluded that ecstasy-related cognitive impairments, particularly in memory, may endure even after prolonged abstinence [152].

Cocaine

Extensive research has shown that repeated cocaine use is associated with learning and memory impairments [153-164,165]. These deficits appear to be particularly pronounced in individuals with cocaine dependence [154-158,161,162,164,165]. A meta-analysis of 46 studies, including 1,452 chronic cocaine users and 1,411 controls, found moderate cognitive impairments across eight domains, including learning and memory, during intermediate abstinence [166]. Similarly, another review reported moderate deficits in immediate and delayed recall among cocaine users [108].

Some studies suggest that cognitive impairments may be reversible after sustained abstinence. A longitudinal study by Vonmoos et al., found that, following moderate cocaine exposure, cognitive deficits significantly improved within one year of abstinence [167]. Additionally, a meta-analysis reported that long-term abstinence (five months or more) was associated with only small effect sizes for learning and memory deficits, suggesting the potential for cognitive recovery over time [166].

Conclusion

Overall, the evidence suggests that chronic use of cannabis, MDMA, and cocaine is associated with impairments in objective episodic memory. Cross-sectional studies consistently report deficits among long-term users of these substances, with heavy or prolonged use leading to more pronounced impairments. However, findings from longitudinal research indicate that while some cognitive deficits may persist, recovery is possible with sustained abstinence, particularly in cannabis and cocaine users.

Subjective episodic memory via autobiographical memory tests

Autobiographical Memory (AM) is a type of retrospective memory that encompasses recollections of events from an individual's life, combining subjective episodic memory (personal experiences involving specific people, places, and events at particular times) with semantic memory (general knowledge and facts about the world). AM is thought to be a vital part of one's life since it aids in self-awareness, interpersonal connections, decision-making, and stress management [168]. Despite the importance of AM, research into the effects of recreational substance use on this memory system is very limited, with most studies focusing on cannabis use and occasional investigations into substances like MDMA.

Acute and Long-term effects

Cannabis

Research on cannabis use and AM has largely focused on its long-term effects. Pillersdorf and Scoboria found that chronic cannabis users (defined as at least 3-4 uses per month for a year) exhibited reduced specificity in AM compared to non-users. This suggests that cannabis may impair the ability to retrieve detailed past experiences [169]. Similarly, Mercuri et al., reported that regular cannabis users (at least three times per week) performed worse on autobiographical interviews than both recreational users and non-users, highlighting the role of frequency in AM deficits [170]. Further evidence of a dose-dependent effect was provided by Sofis et al., who found that more frequent cannabis use was associated with reduced specificity and emotional richness in recent positive autobiographical memories [171]. This aligns with longitudinal findings by Gandolphe and Nandrino, which demonstrated that increasing cannabis use over time correlated with declining specificity in both positive and negative autobiographical recollections [172].

MDMA

The effects of MDMA on AM have primarily been explored in the context of acute use. In a double-blind, placebo-controlled study, Doss et al., investigated MDMA's impact on emotional memory. Participants viewed emotionally neutral, negative, and positive images paired with descriptive labels before completing memory tests 48 hours later. MDMA administration during encoding or retrieval impaired the recollection of emotional events, while recognition memory remained unaffected [173]. These findings suggest that MDMA specifically disrupts the recollection component of AM, rather than general memory function.

Cocaine

Although less extensively studied, research suggests that substance dependencies, including cocaine dependence, may also impair autobiographical memory. Studies have reported that individuals with cocaine dependence exhibit reduced specificity in AM compared to non-using controls [174,175].

Conclusion

Research on AM is very limited, but the existing literature suggest that cannabis, MDMA, and cocaine use can negatively impact AM. Chronic cannabis use, particularly with higher frequency, is associated with reduced specificity and emotional richness in AM.

Prospective memory

Prospective Memory (PM) refers to the ability to remember to execute future intentions [176,177]. This crucial cognitive function underlies everyday activities, ranging from simple tasks like remembering to buy groceries to more critical ones like remembering taking daily medications. Indeed, individuals who forgot to take their blood pressure medication were found to have a higher likelihood of experiencing a heart attack or dying compared to those who remembered [178].

Acute effects

Cannabis

The existing literature is very limited and mixed. One study with a double-blind, placebo-controlled study found that cannabis impaired performance across various memory tasks, including PM [179]. On the contrary, other two studies found no acute effects of cannabis use on PM [180,181].

MDMA

A study examining the acute effects of MDMA on PM tested twelve recreational MDMA users who received a single dose of MDMA and a placebo in separate sessions, followed by completion of an objective PM task during functional imaging. The behavioural data revealed that a single dose of MDMA led to an increase in PM failures, with the number of failures positively correlating with MDMA plasma concentrations. This study offers direct evidence of the impact of MDMA on PM performance and the associated brain activity [182].

Cocaine

To date, only one study has explored the immediate effects of cocaine on PM. This placebo-controlled, three-way crossover study involved 15 participants with regular polysubstance use and aimed to determine how oral cocaine and vaporised cannabis influence performance on an event-based PM task. Participants received either cocaine, cannabis, or a placebo in separate sessions and then completed the PM task. The results demonstrated that cocaine administration significantly enhanced PM performance compared to both the placebo and cannabis conditions [181].

Conclusion

Research on acute substance effects on PM is very limited. Findings suggest mixed effects of cannabis, increased PM failures with MDMA, and improved performance with cocaine.

Long term-effects

Cannabis

Several studies have reported that cannabis users exhibit impaired performance on various PM tasks [66,180,183-188]. These deficits have been linked to duration of cannabis use, dosage, frequency, cumulative exposure, and early onset of use [66,184,188,189]. A meta-analysis further supports these findings, demonstrating a significant association between cannabis use and impaired PM [94]. Notably, PM deficits appear more pronounced in laboratory-based assessments than in self-reported measures. For instance, Bartholomew et al., found that while cannabis users did not report significant PM difficulties, they performed worse on a video-based PM task, recalling fewer location-action combinations [190]. This suggests that cannabis-related PM impairments may go unnoticed by users. However, some studies have not found significant association between PM impairments and cannabis use [191-194].

MDMA

Multiple studies found PM impairment in MDMA/ecstasy users [66,193-204]. These deficits have been linked to the level of ecstasy use, with heavier users exhibiting greater impairments [66,194,202]. Some evidence suggests that PM deficits in MDMA users may be more pronounced under specific conditions, particularly when there is a longer delay between forming an intention and executing it. For instance, Weinborn et al., found that while ecstasy users performed comparably to controls on short-delay PM tasks, their performance was significantly impaired on long-delay tasks [205]. Additionally, some studies suggest that PM impairments in MDMA users may be attributable to concurrent cannabis use rather than MDMA alone [66,183,194].

Cocaine

Currently, no research has specifically examined the long-term effects of cocaine on PM. However, some studies investigating MDMA's impact on PM have included cocaine use as part of broader analyses. For instance, Hadjiefthyvoulou et al., studied a group of people with a history of ecstasy/polysubstance use and those with non-ecstasy substance use, which included individuals who used cocaine. Their findings indicated a positive correlation between lifetime cocaine use and PM impairments, suggesting that higher levels of cocaine consumption may contribute to PM deficits [196]. Further evidence comes from a study by Levent and Davelaar, which examined a group of people who primarily used cocaine. The results showed that while people with a history of substance use exhibited impairments on lab-based PM tasks compared to drug-naïve individuals, they did not report significant deficits on self-reported PM measures [206]. A similar discrepancy was highlighted in a comprehensive systematic review, where substance users demonstrated deficits in objective PM tasks but did not perceive impairments in their everyday PM performance [177]. This divergence between self-report and lab-based findings may be explained by impaired metacognition in substance users [207], which refers to an individual's awareness of their own cognitive abilities [208]. Reduced metacognitive awareness may lead people with substance use to underestimate their PM difficulties in real-world settings, despite measurable impairments in controlled experimental conditions.

Conclusion

Overall, the long-term effects of substance use on PM remain complex and varied across substances. Cannabis and MDMA use are both associated with PM impairments, particularly in heavy and long-term users, with deficits more evident in laboratory-based tasks than self-reports. Cocaine's impact on PM is less studied, though preliminary evidence suggests potential impairments linked to higher lifetime use.

Executive function

Executive Functions (EFs), also known as executive control or cognitive control, are a group of mental processes that help individuals plan, organise, integrate, and manage thoughts and behaviours. Often described as the "CEO" of the brain, EFs are essential for performing everyday tasks such as such as organising, planning, prioritising, paying attention and remembering details, and governing emotional responses [209]. It is generally agreed that three core EFs: working memory, cognitive flexibility, and inhibition [210]. Therefore, this section will review the literature on the acute and long-term effects of cannabis, MDMA, and cocaine use on EFs under three subsections: cognitive inhibition, working memory, and cognitive flexibility.

Cognitive inhibition

Cognitive inhibition, also known as inhibitory control, involves a set of processes that regulate thoughts, behaviours, attention, and emotions by suppressing dominant impulses or external distractions in favour of more contextually appropriate action [211]. This ability is crucial for self-regulation and decision-making, particularly in the context of addiction. Deficits in inhibitory control may contribute to the difficulty individuals face in resisting substance-related urges, thereby increasing the risk of relapse [212]. Additionally, poor inhibitory control has been linked to key behavioural characteristics commonly observed in substance use disorders, such as heightened impulsivity [213,214], increased sensation seeking [215], and impaired decision-making [216].

Acute effects

Cannabis

Cannabis use has been widely studied in the context of its acute effects on cognitive inhibition, revealing consistent impairments across different tasks designed to measure this domain [179,182,217-220]. Similarly, Hunault et al., reported that THC decreased response times and increased errors on a cognitive inhibition task in a dose-dependent manner, with the number of errors increasing significantly as the dose increased [221]. Such findings have been corroborated by multiple review studies [14,91,222,223]. Interestingly, the acute effects of THC on cognitive inhibition appears to differ between occasional and heavy cannabis users, potentially reflecting a tolerance effect. Ramaekers et al., observed that THC administration impaired psychomotor control in occasional users but not in heavy users [182]. This suggests that heavy cannabis users might develop a degree of tolerance to some of the impairing behavioural effects of cannabis, as proposed by Crane et al., and Theunissen et al., [224,225].

MDMA

Research on the acute effects of MDMA on cognitive inhibition has yielded mixed findings, with some studies suggesting no significant impairments while others highlight potential improvements in specific inhibitory processes. For instance, multiple studies have reported that MDMA does not negatively affect cognitive inhibition [226,227]. A review also found that most studies that used measures of inhibition have failed to provide evidence for a relationship between ecstasy use and lower levels of inhibition [228]. Interestingly, one study demonstrated that acute doses of MDMA improved impulse control. Ramaekers and Kuypers found that participants exhibited enhanced cognitive inhibition task performance following MDMA administration [229].

Cocaine

Research examining the effects of acute cocaine doses on cognitive inhibition yielded mixed results. Some studies suggest that cocaine can enhance inhibitory control. For example, Garavan et al., and Fillmore et al., observed improved task performance in cocaine users following cocaine administration, indicating a potential enhancement of inhibitory control [230,231]. However, this enhancement may be dose-dependent. Fillmore et al., found that cocaine reduced the time required to inhibit a response, but only at doses of 100 mg and 200 mg. Higher doses (300 mg) did not produce the same speeding effect [232]. In contrast, other studies have demonstrated that cocaine can impair specific aspects of inhibitory control. For example, Fillmore et al., found that cocaine impaired the ability to inhibit behavioural responses, but did not affect response speed or accuracy, indicating a selective effect on behavioural suppression [233]. Similarly, van Wel et al., observed decreased response times alongside increased errors on impulsivity tasks, highlighting impairments in accuracy despite faster responses [220].

Conclusion

Overall, the acute effects of these substances on cognitive inhibition vary significantly. Cannabis consistently impairs inhibitory control, while MDMA's effects remain inconclusive. Cocaine shows a complex, dose-dependent pattern, with both improvements and impairments reported.

Long-term effects

Cannabis

The evidence regarding deficits in cognitive inhibition among abstinent cannabis users is mixed, with more favour no effect. Some studies suggest chronic cannabis use is associated with impairments in cognitive inhibition, particularly when use begins at an earlier age. For instance, Battisti et al., reported that chronic cannabis users exhibited poorer cognitive inhibition, with earlier age of onset predicting worse performance [112]. This finding implies that deficits may be more pronounced in individuals who initiate cannabis use during adolescence. Supporting this, multiple studies have shown that early-onset users (before age 15) tend to perform worse than both controls and late-onset users [234-236]. Such results align with the notion that cannabis exposure during adolescence (a critical period of neurodevelopment) may disrupt brain development and lead to lasting neuropsychological changes. A dose-dependent relationship has also been observed. For example, Piechatzek et al., found that more frequent cannabis use correlated with higher impulsivity [237].

However, the majority of studies examining long-term effects of cannabis use on cognitive inhibition report no significant deficits. Several studies have failed to find evidence of impairment [113,224,238-243]. Meta-analyses focusing on abstinent users also concluded that past cannabis use does not significantly impact executive functions, including cognitive inhibition [120,132,212]. Interestingly, some studies have revealed nuanced findings. Tapert et al., and Roberts and Garavan found that cannabis users exhibited intact inhibitory control but required greater brain processing effort to achieve comparable task performance to non-users [243,244]. This increased neural activation suggests that cannabis users may compensate for subtle deficits or inefficiencies in their cognitive processes. Tapert et al., proposed that this heightened effort might either predate the onset of regular cannabis use or be a consequence of it [244].

MDMA

The long-term effects of MDMA on cognitive inhibition present a mixed picture. While most cross-sectional studies comparing MDMA/ecstasy users to non-users found no significant group differences in cognitive inhibition [77,142,143,150,245,246,247], some studies reported poorer performance among ecstasy users [237,248-250]. Piechatzek et al., further demonstrated a dose-dependent relationship, with increased ecstasy consumption associated with poorer performance on cognitive inhibition tests [237]. A limited number of longitudinal studies have addressed the methodological limitations of cross-sectional designs by assessing novice MDMA users over time. After controlling for confounding factors such as age, general intelligence, polysubstance use, and lifestyle variables, these studies found no significant changes in cognitive inhibition over the follow-up period [146,251].

A meta-analysis by Roberts et al., further supports the lack of consistent evidence for impaired cognitive inhibition in ecstasy users. This analysis, which compared 632 sub-stance-using controls to 600 ecstasy polysubstance users across 20 studies, found no significant group differences in inhibitory control performance [37].

Cocaine

Multiple studies have found that cocaine users, particularly those with cocaine dependence, exhibit significantly poorer inhibitory control compared to non-users [165,208,252-260]. Notably, the severity of inhibitory deficits has been positively correlated with lifetime cocaine exposure [253,261] and the dose used [252]. Interestingly, cocaine users performed significantly worse than controls on more demanding cognitive inhibition tasks, while easier tasks show little to no difference [262]. This pattern has been corroborated by studies highlighting performance deficits specifically in complex inhibitory control tasks [208,256].

Conversely, some studies found no behavioural cognitive inhibition impairments [263,264]. However, neuroimaging evidence suggests that cocaine users exhibit distinct patterns of brain activity compared to non-users during inhibition tasks, indicating the engagement of alternative neural pathways. This supports the compensatory mechanism theory, which posits that cocaine users recruit additional neural resources to compensate for deficits in cognitive inhibition [265-267].

A comprehensive meta-analysis of 46 studies identified cognitive inhibition as one of the most impaired domains in cocaine users, with moderate deficits observed [166]. Another meta-analysis further reinforced the presence of response inhibition impairments, reporting moderate effect sizes and suggesting a robust and consistent effect [108]. These findings align with broader reviews on cocaine-related cognitive inhibition impairment [212,268], though it is important to note that most reviews focus specifically on individuals with cocaine dependence.

Relatively few studies have investigated the long-term effects of cocaine use on cognitive inhibition following prolonged abstinence. Bell et al., compared cognitive inhibition performance among current cocaine users, ex-users, and non-users, finding that despite a history of chronic cocaine use (average duration = 8.2 years), ex-users performed just as well as non-users [269]. Neuroimaging data further revealed no significant differences in brain activity between these groups, suggesting that inhibitory control may recover with sustained abstinence. Similarly, Connolly et al., reported that individuals with prolonged abstinence from cocaine (40-102 weeks) exhibited greater recruitment of cognitive control regions compared to those in short-term abstinence (1-5 weeks). These findings suggest that inhibitory control can improve over time with abstinence or, alternatively, that individuals with stronger inhibitory control are more likely to maintain long-term abstinence [269].

Conclusion

Overall, the long-term effects of substance use on cognitive inhibition vary across substances. Chronic cannabis use does not appear to cause lasting deficits in abstinent users, though compensatory neural mechanisms may be involved. MDMA's impact remains inconclusive, with no consistent evidence of long-term impairments. In contrast, cocaine use, particularly in cases of dependence, is strongly linked to inhibitory control deficits, which worsen with greater exposure but may be reversible with sustained abstinence.

Working memory

Working Memory (WM) combines the ability to keep information for a very short period of time while allowing the controlling and planning of that information [270]. Working memory serves as a mental workspace, aiding in learning, reasoning, decision-making, and problem-solving [271].

Acute effects

Cannabis

Early investigations of WM have indicated that acute cannabis use is associated with impairments in holding, manipulating and remembering information [272,273]. More recent studies also found that acute intoxication resulted in significant impairment in WM [86,89,179,274-282]. Hunault et al., identified a dose–response relationship, demonstrating that higher THC concentrations in cannabis cigarettes are linearly associated with poorer cognitive performance, including impairments in WM [221]. Additionally, a review by Crean et al., concluded that THC administration has a detrimental effect on executive functions, particularly WM [14].

MDMA

Research on the acute effects of MDMA on working memory is limited, and the findings are mixed. Some studies have reported that acute MDMA administration impairs performance on WM tasks [283,284], while others have found no significant effects [285]. More recently, Basedow et al., reviewed the literature and concluded that the majority of studies investigating the acute effects of MDMA on WM reported no evidence of impairment [107].

Cocaine

Most research on the acute effects of cocaine on WM has been conducted in animal models, with no studies to date found involving human participants.

Conclusion

Overall, the acute impact of these substances on WM differs. Cannabis consistently impairs WM in a dose-dependent manner, while evidence for MDMA remains inconclusive, with most studies reporting no significant deficits. Research on the acute effects of cocaine on WM is currently lacking.

Long-term effects

Cannabis

In studies examining WM, the evidence for cannabis-associated deficits is mixed. Multiple studies showed that cannabis use is significantly associated with WM deficits [115,186,240,286]. These deficits are associated with duration of use [287,288], age of onset of cannabis use [122] or with greater frequency and quantity of use [115,122,289]. However, some other studies reported intact WM among cannabis users [240,290-294], in particular after controlling for confounds [134]. Additionally, in a longitudinal study, WM performance remained intact among recently abstinent current cannabis users, former cannabis users, and controls over a period of eight years [135]. Interestingly, despite comparable task performance with control groups, cannabis users exhibited different brain activity patterns, suggesting the recruitment of additional brain regions not typically engaged during WM tasks [291,294-298]. These findings support the theory of compensatory mechanisms in cannabis users [243,244].

In line with those studies above, multiple reviews examining the long-term effects of cannabis on WM have yielded mixed findings. Some studies report inconsistent evidence for cannabis-induced impairments in WM [8,299], while others found no residual effects of regular cannabis use on overall WM [92]. A longitudinal study found that, while cannabis use acutely impaired WM, users showed significant improvement in WM performance after 3 weeks of abstinence [114], suggesting that the negative effects of cannabis can be reversed with prolonged abstinence. Other studies have also shown no working memory impairments after 25 or more days of abstinence [297,298]. Consistent with these findings, a meta-analysis of 12 studies found no significant impact of past cannabis use on executive functions, including WM, in participants who had been abstinent for at least 25 days [132]. Overall, these findings suggest that the long-term effects of cannabis on brain function may be reversible with sufficient abstinence [291].

MDMA

Findings on the long term effects of MDMA on WM are mixed [300], but more studies in favour of no effects. For example, some studies found that ecstasy users performed worse than non-user controls on various WM measures [78,247,301-305], with dose relation where increased ecstasy consumption was associated with poorer task performance [78,237,301,306] and which does not improve with abstinence [307,308]. Notably, the impact of MDMA appears to depend on the complexity of the WM task. Less demanding tasks have generally shown no significant differences between ecstasy users and non-users [142,191,309], whereas more cognitively demanding tests have revealed ecstasy-related deficits [302,303]. On the contrary, many studies found no effects [140,142,191,245,309-311]. In multiple follow-up studies, where participants were tested on different occasion to explore the effects on MDMA on WM found no deterioration in continuing MDMA-users was observed in the follow-up periods [62,146,312], potentially related to a low dose of ecstasy use [62,312].

It is worth noting that MDMA users appear to recruit additional cognitive resources to perform WM tasks compared to non-users, suggesting the involvement of compensatory neural mechanisms [310]. This aligns with findings from a comprehensive review of neuroimaging studies on ecstasy users, which also indicates potential compensatory activity in the brain [38].

Cocaine

Numerous studies have found that cocaine users often perform worse than non-users on WM tasks, particularly those with cocaine dependence [313-317]. The age of onset and severity of cocaine use also influence WM impairments, with increased use correlating with greater deficits [318,319]. A follow-up study by Vonmoos et al., provided further support for this relationship, showing that substantial increases in cocaine use over one year (mean +297%) were associated with marked declines in cognitive performance, particularly in WM. Conversely, participants who significantly reduced their cocaine use (-72%) exhibited modest cognitive improvements, while those who achieved sustained abstinence showed complete recovery, performing at levels comparable to non-user controls [167].

However, some studies report no differences in WM performance between cocaine users and controls [320,321]. Despite this, neuroimaging suggests cocaine users may rely on compensatory brain activity to maintain performance [256,322]. A comprehensive review of 46 studies on cognitive dysfunction in individuals with cocaine abuse or dependence found moderate impairments across eight cognitive domains, with WM being one of the most affected, especially during intermediate abstinence [166]. Interestingly, the review also highlighted small effect sizes for cognitive deficits in those with long-term abstinence, suggesting that extended cessation from cocaine use may lead to partial or even full recovery of WM function. These findings align with earlier research by Jovanovski et al., [323].

Conclusion

The long-term effects of cannabis, MDMA, and cocaine on WM remain ambiguous, with conflicting evidence across studies. For cannabis, some research identifies deficits linked to prolonged use, higher frequency, and earlier initiation, whereas other studies have found no such impairments. Notably, emerging evidence proposes that such deficits may diminish with sustained abstinence. Research on MDMA similarly reveals inconsistent results, with some studies reporting dose-dependent impairments, particularly in complex tasks, while others show no significant effects. Cocaine use, especially in heavy users, is generally associated with WM deficits, yet research indicates that reducing use or achieving abstinence can promote cognitive recovery. Neuroimaging findings across all three substances suggest that users may rely on compensatory neural mechanisms to maintain task performance, underscoring the brain's ability to adapt to substance-related cognitive challenges.

Cognitive flexibility

Cognitive flexibility is another component of EFs which is defined as the brain's ability to switch from thinking about one concept to another [324]. With cognitive flexibility, one is able to adapt his or her thinking and behaviour in response to the environment that constantly changes [325].

Acute effects

Cannabis

The evidence on the acute effects of cannabis intoxication on cognitive flexibility is limited and somewhat mixed. Some studies suggest that acute cannabis use disrupts cognitive flexibility, impairing an individual's ability to adapt to changing rules or switch between tasks [326-328]. However, contrasting findings have also been reported. For instance, Hart et al., found that while THC administration did not affect accuracy on measures of cognitive flexibility, although it significantly increased the number of premature responses and prolonged the time required to complete various tasks [277].

Furthermore, a systematic meta-review of meta-analyses by Dellazizzo et al., reported that cannabis use led to small deficits in cognitive flexibility [92].

MDMA

Limited number of research with double-blind, randomised, placebo-controlled study design showed that the single administration of MDMA did not affect cognitive flexibility [100,329].

Cocaine

To the authors' knowledge, no studies have been conducted on cognitive flexibility following acute cocaine administration in humans.

Conclusion

In conclusion, the evidence regarding the acute effects of cannabis, MDMA, and cocaine on cognitive flexibility is limited and mixed. Cannabis may impair cognitive flexibility in some studies, while others show no effects. Research on MDMA suggests no impact, and there is a lack of studies on the acute effects of cocaine on cognitive flexibility.

Long-term effects

Cannabis

Extensive research has explored the long-term effects of cannabis use on cognitive flexibility, with findings remaining mixed. Several studies have reported no significant differences in cognitive flexibility performance between cannabis users and non-users [330-333]. Conversely, other studies have found clear evidence of cognitive flexibility impairments among cannabis users [234,236,239,334-336]. These deficits have been linked to specific patterns of cannabis use, such as greater consumption in the past 30 days [334], early adolescent exposure [13,234-236], and heavy use [335]. Bolla et al., provided further support for dose-dependent effects, reporting that heavy cannabis users performed worse on cognitive flexibility tasks compared to moderate and occasional users, even after 28 days of abstinence. Their findings demonstrated a positive correlation between the frequency of cannabis consumption and poorer cognitive flexibility performance [337].

Meta-analysis findings also reflect these inconsistencies. An earlier meta-analysis by Grant et al., found no significant non-acute effects of cannabis use on cognitive flexibility [120]. Similarly, Schreiner and Dunn reported no significant impact of past cannabis use on cognitive flexibility [132]. However, a more recent meta-analysis by Figueiredo et al., identified a small but significant association between chronic cannabis use and cognitive impairments across multiple domains, including cognitive flexibility [338].

MDMA

There is limited number of studies long-term effects of MDMA on cognitive flexibility. The existing literature is mixed. For instance, Dafters reported that chronic MDMA users exhibited impairments in task-switching performance [245]. Additionally, Verdejo-García et al., found a positive correlation between self-reported lifetime MDMA exposure and the number of perseverative errors, lending support to the neurotoxicity hypothesis associated with MDMA use [339].

On the contrary, Several studies have reported no residual cognitive effects in ecstasy users, with performance on cognitive flexibility tasks comparable to that of non-users [78,150,237,302]. Moreover, a longitudinal study conducted over a two-year period found no evidence of cognitive deterioration in individuals who continued using MDMA, suggesting that ongoing use did not exacerbate deficits in cognitive flexibility during the follow-up period [146].

Cocaine

Several studies have examined the long-term effects of cocaine use on cognitive flexibility, with the majority focusing on individuals with cocaine dependence. Consistently, these studies have reported that cocaine-dependent individuals exhibit significant impairments in cognitive flexibility compared to non-users [153,165,238,254,263,314,318,340-347]. Although fewer in number, studies involving recreational cocaine users have also identified deficits in cognitive flexibility, suggesting that impairments may not be exclusive to individuals with clinical dependence [313,317]. Moreover, dose-related neurocognitive research indicates a clear association between the severity of cocaine use (in terms of both quantity and duration) and cognitive flexibility deficits as greater cocaine use has been consistently linked to poorer performance on cognitive flexibility tasks [252,345,348,349].

Conversely, some studies have found no significant differences in cognitive flexibility between cocaine users and non-user controls, particularly in samples of recreational users [158,315,321,350]. Early review papers concluded that chronic cocaine use is generally associated with mild impairments in cognitive flexibility [108,351]. However, more recent reviews have reported inconsistent findings, suggesting that observed deficits may be context-specific and influenced by particular experimental conditions rather than representing broad, generalizable effects [53].

Conclusion

In summary, the long-term effects of cannabis, MDMA, and cocaine on cognitive flexibility are inconsistent. While some studies show no significant differences between cannabis users and non-users, others report impairments linked to patterns of use and dose. Similarly, MDMA's long-term impact is mixed, with some studies reporting deficits and others showing no effects. Cocaine use, particularly in dependent individuals, is generally linked to cognitive flexibility impairments, but findings are more varied in recreational users.

Concluding Remarks

The body of research reviewed reveals that both the acute and long-term effects of cannabis, MDMA, and cocaine on retrospective memory vary. Acute use of cannabis and MDMA consistently impairs objective episodic memory, while acute cocaine use appears to have no effect. However, chronic use of all three substances is associated with deficits in objective episodic memory, with impairment severity increasing with prolonged or heavy use. Longitudinal studies suggest that cognitive recovery is possible, particularly for cannabis and cocaine users, following extended abstinence. Although research on subjective episodic memory remains limited, existing findings indicate that all three substances can negatively affect autobiographical memory. It is well established that impairments in retrospective memory, particularly objective episodic memory (tested via learning tests), are closely associated with poor academic performance [352], which has been found to increase the risk of substance abuse and subsequent addiction [353,354].

Research on the effects of cannabis, MDMA, and cocaine on PM is limited. While the acute administration of cannabis and MDMA impair PM, cocaine appears to enhance PM performance. Long-term use of those substances is linked to PM deficits, especially in heavy users, with stronger effects in laboratory tasks than self-reports. The observed discrepancy between self-reported and laboratory-based PM measures in people with substance use points to metacognitive deficits [177,206], wherein people with substance use exhibit diminished awareness of cognitive impairments potentially stemming from substance use. This accords with previous observations, which showed that people who use substance use are impaired in metacognition [207,208,241,355-360]. This lack of awareness could contribute to continued substance use, as individuals may underestimate the impact of these impairments. Consequently, this may lead to increased consumption, potentially escalating to addiction due to prolonged and excessive use.

The acute effects of cannabis, MDMA and cocaine on executive functions are mixed. Cannabis consistently impairs inhibitory control and working memory and may affect cognitive flexibility. MDMA's impact remains inconclusive across all domains, with no consistent evidence of impairment. Cocaine's effects are complex and dose-dependent, with potential improvements or impairments in cognitive inhibition, while research on its effects on working memory and cognitive flexibility remains limited. The long-term effects of these substances on executive functions vary across substances and executive functioning domains. Chronic cannabis use does not appear to cause lasting deficits in inhibitory control, though some studies suggest potential working memory and cognitive flexibility impairments, predominantly with heavier use. MDMA's long-term impact remains inconclusive, with inconsistent findings across studies. Cocaine, especially in cases of dependence, is strongly linked to inhibitory control and working memory deficits, though some recovery may occur with sustained abstinence.

The existing literature indicates that the observed deficits are associated with the duration of substance use, dosage, frequency, cumulative exposure, and early onset of use. Neuroimaging evidence across various substances suggests that compensatory mechanisms may help mitigate substance-induced cognitive impairments, mainly in executive function domains. People who use substance seem to recruit additional cognitive resources to perform executive function tasks, indicating the involvement of compensatory neural mechanisms [38,243,244,256,265-267,294,297,301,310].

The cognitive impairments observed in people with a history of substance use, including deficits in cognitive inhibition, AM, and WM, may increase the risk for further substance use and contribute to the progression from recreational use to addiction [361-364]. Some studies suggest that recovery from substance-induced cognitive impairments is possible with prolonged abstinence, particularly for cocaine and cannabis use [114,131,132,167,267,269,291,297,298], highlighting the brain's potential for compensatory mechanisms and cognitive restoration over time.

A substantial body of evidence indicates that adolescents, who typically engage in higher levels of illegal recreational substance use compared to other age groups [1], are at a higher risk of suffering potential harmful effects from substance use, particularly cannabis use [118,234-236,290,319,361]. One explanation for these harmful effects is that the adolescent brain undergoes significant development until approximately 25 years of age [365-367] and interference with these processes may manifest as the cognitive impairments ob-served in the existing literature. These substance-induced cognitive impairments may also contribute to the transition from recreational substance use to addiction [353,354,361,363-365,368,369].

It is important to acknowledge the challenges in conducting studies on the effects of substance use on cognition, including polysubstance use, difficulties in participant recruit-ment, and inadequate control of confounding variables, all of which complicate the interpretation of findings.

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