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How Does Adolescent Cannabis Use Affect Susceptibility To Schizophrenia? A Research Study

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Abstract

Introduction: One of the most widely used substances among adolescents is cannabis, which may have negative effects during a critical period of brain development. Adolescent cannabis use may contribute to an earlier onset of schizophrenia, a psychiatric condition that typically appears in early adulthood. The objective of this review is to investigate current research in neurology, epidemiology, and substance use, to determine whether adolescent cannabis use is associated with an increased risk of schizophrenia.

Methods: A systematic review was conducted of peer-reviewed literature from the last 20 years of both clinical and pre-clinical research from databases such as PubMed, PsycINFO, Omni Laurier, and Google Scholar. The review incorporated epidemiological, neurological, and genetic studies to examine the relationship between adolescent cannabis use and schizophrenia.

Results: Adolescent cannabis use disrupts dopaminergic, GABA, and glutamate systems, impairing sensorimotor gating and cognitive functions. Animal studies show lasting alterations in dopamine neuron activity linked to schizophrenia. Neuroimaging in humans reveals reduced prefrontal cortex activation and white matter integrity. These findings suggest increased schizophrenia susceptibility, especially in genetically predisposed individuals.

Discussion: The neurological and behavioural changes resulting from adolescent cannabis use contribute to cognitive deficits seen in schizophrenia. Particularly, impairment of GABA and glutamate signalling in the prefrontal cortex mirror symptoms of schizophrenia. THC exposure alters dopaminergic signalling and sensorimotor gating, increasing schizophrenia risk, particularly in genetically predisposed individuals. Long-term effects of cannabis use suggest lasting changes in brain circuits, further increasing schizophrenia susceptibility.

Conclusion: By highlighting a central risk factor, this review expanded on the neurobiological and behavioural effects of adolescent cannabis use, particularly its role in increasing schizophrenia risk. The results can inform public health campaigns and the development of interventions and preventative strategies to reduce cannabis use among young adults and spread awareness of its dangers at this developmental stage. Results may also encourage further research in the fields of neurology and drug use.

Keywords: adolescent cannabis use; schizophrenia; prefrontal cortex; neurotransmitter signalling; dopamine; sensorimotor gating; public health

Introduction

Cannabis use among adolescents is a pressing public health concern, particularly because adolescence is a critical period of brain development. This development is characterized mainly by maturation of the prefrontal cortex (PFC), which is responsible for complex behaviours like decision-making and impulse control. Synaptic plasticity - the brain's ability to strengthen signal communication - is particularly important in adolescence, since it coordinates fine-tuning of neural circuits involved in the PFC's executive functions. Synaptic pruning, or selective elimination and enhancement, during this stage also helps refine cognitive abilities and social behaviours [1]. These rapid changes in synaptic plasticity induce adjustments in the dopaminergic and endocannabinoid systems of the PFC. Since cannabis can magnify dopamine signalling, the developing adolescent brain is particularly more vulnerable to these influences in comparison to an adult brain [2]. However, cannabis is one of the most commonly used recreational drugs during adolescence [3]. Its widespread use raises concerns about long-term effects on mental health, academic performance, and risk of developing psychosis [4].

Schizophrenia is a complex neuropsychiatric disorder that typically emerges in late adolescence or early adulthood, with symptoms ranging from hallucinations and

delusions to disordered thoughts [5]. Several studies have suggested that cannabis use may contribute to an increased risk of developing schizophrenia, particularly when use begins during adolescence. The relationship between adolescent cannabis use and schizophrenia has been supported by epidemiological studies, which show a higher incidence and earlier onset of psychosis in those who use cannabis during their teenage years compared to non-users [4]. Cannabis use may also exacerbate pre-existing dispositions, such as genetic factors, which are believed to increase susceptibility to schizophrenia [5].

Emerging research, including animal models, has revealed some of the underlying mechanisms through which cannabis can contribute to the development and progression of schizophrenia. Animal studies have shown that adolescent exposure to tetrahydrocannabinol (THC), the active ingredient in cannabis, can alter gene expression involved in synaptic plasticity [6]. Since synaptic plasticity alterations are linked to structural brain connectivity and neurotransmitter signalling, they create a cascade of neurodevelopmental disturbances. Animal studies also demonstrate that THC impairs the maturation of the endogenous cannabinoid system, a neurological regulation network involved in developing the central nervous system, which includes the brain and its modulation of cognitive and physiological activities [7]. Findings suggest that these changes in the endogenous cannabinoid system impact neurotransmitter (NT) signalling, specifically with GABA and glutamate, in the PFC, similar to the disordering seen in schizophrenia patients. These disruptions highlight the shocking resemblance of the brain circuit changes in schizophrenia and cannabis-affected adolescents.

Clinical studies and neuroimaging data further support these findings by showing structural and functional brain changes in human adolescent cannabis users. Magnetic resonance imaging (MRI) studies indicate that adolescent cannabis users exhibit decreased activation of brain regions that manage executive attention and are associated with psychosis, such as the PFC [3]. More neuroimaging studies reveal associations between early cannabis use and disruptions in neural connectivity related to IQ [8]. Longitudinal studies further provide compelling evidence that early, regular cannabis use increases the risk of psychotic disorders and cognitive impairments later in life, with adolescent use presenting the highest risks [4]. These results are evident across cultures, reinforcing the idea that adolescent cannabis exposure has long-term neurodevelopmental consequences, including increased psychosis risks due to its overstimulation of cannabinoid receptors [2].

The primary aim of this review is to analyze current research on adolescent cannabis use and its connection to schizophrenia susceptibility. Specifically, this review will focus on synthesizing findings from animal models and human clinical studies to inform our understanding of the cognitive and physical mechanisms by which cannabis may lead to neurocognitive disorders. By using these studies and clinical observations, this comprehensive review seeks to improve our knowledge of the biological, cognitive, and behavioural effects of adolescent cannabis exposure and its implications on informing public health, clinical practice, and research.

This systematic review will address the following research question to synthesize current research: How does adolescent cannabis use affect susceptibility to schizophrenia?

Methods

A comprehensive literature search was performed using PubMed, PsycINFO, Omni Laurier, and Google Scholar to identify peer-reviewed studies on adolescent cannabis use and schizophrenia. Keywords included “adolescent cannabis use,” “schizophrenia,” “brain development,” “MRI,” “behavioural deficits,” and “psychosis.” Inclusion criteria focused on studies published since 2004, incorporating both animal and human studies, with neuroimaging techniques like MRI and fMRI. Studies solely on adult cannabis use or published before 2004 were excluded. A total of 24 articles were included.

James et al. (2009) and Peters et al. (2009) [9, 10] employed fractional anisotropy (FA) to assess white matter integrity by measuring water diffusion in brain tissue, revealing neural connectivity changes associated with schizophrenia [11]. Renard et al. (2016), Rubino et al. (2014), Dunn et al. (2020), and others utilized prepulse inhibition (PPI) to assess sensorimotor gating deficits [12-14]. PPI measures the brain’s ability to filter sensory inputs, often impaired in schizophrenia [15]. These methodologies provide crucial insights into the neurobiological effects of adolescent cannabis exposure.

Results

Results from the systematic review of both animal and human studies highlight the neurobiological and behavioural effects of adolescent cannabis use (See Table 1 and Table 2 for summarized findings). Preclinical studies show disruptions in dopaminergic systems, impairments in sensorimotor gating, and cognitive deficits [6, 12]. Aguilar et al. (2018) found that adolescent exposure to synthetic cannabinoids in rats led to long-lasting alterations in dopamine neuron activity, which are associated with schizophrenia susceptibility [16]. Human studies mirror these findings, linking early cannabis use with memory, attention, and psychosis-related impairments. The results emphasize heightened vulnerability during adolescence, suggesting that cannabis use exacerbates schizophrenia risk, specifically in genetically predisposed individuals [2, 17].

Table 1. Summary of Results from Preclinical Studies

Author(s) and Year	Study Model	Cannabis Exposure Duration	Findings
Prini et al. (2018) [6]	Female Sprague-Dawley rats	Postnatal days 28 to 45, increasing dosage weekly	Persistent cognitive deficits i.e. altered histone modifications in synaptic genes.
Aguilar et al. (2018) [16]	Albino Sprague-Dawley rats	14 consecutive days during adolescence	Long-lasting alterations in dopamine neuron activity.

Table 2. Summary of Results from Clinical Studies

Author(s) and Year	Sample Size	Sample Demographics	Findings
Abdullaev et al. (2012) [3]	n=14 users n=14 non-users	Mean age = 19.5 years	Larger conflict effects in reaction times and deficits in executive attention. Stronger activation in the PFC during conflict tasks.
Lesh et al. (2024) [8]	n=48 with schizophrenia and cannabis usage history n=28 with schizophrenia and no cannabis usage history n=59 controls	Ages 12-35 years	Earlier age of starting cannabis was associated with lower IQ. Heavier usage history was linked to severe positive symptoms.
James et al. (2009) [9]	n=16 schizophrenia patient users n=16 schizophrenia patient non-users	Mean age = 16.2 years	Reduced white matter integrity in the PFC.
Peters et al. (2009) [10]	n=17 males with cannabis use history before age 17 n=17 males without cannabis use history before age 17	Males with recent-onset schizophrenia	Increased white matter directional coherence in PFC. Absent in those without early cannabis exposure.
James et al. (2011) [18]	n=16 schizophrenia patient users n=16 schizophrenia patient non-users n=28 controls	Ages 12-18 years	Significant changes in grey and white matter structures. Grey matter density loss in the PFC and reduced white matter integrity.
Johnson et al. (2023) [19]	n=4832	Of European and African ancestry	Genetic risks associated with paranoia, cognitive difficulties, and social withdrawal.

Neurobiological Effects of Adolescent Cannabis Exposure

Animal studies indicate that adolescent cannabis exposure significantly disrupts GABA (gamma-aminobutyric acid) and glutamate signalling within the PFC. Chronic exposure to cannabinoids has been associated with a reduction in GABA inhibition, demonstrated by decreased expression of GABA markers, such as glutamic acid decarboxylase (GAD) 65/67 [20]. This inhibition leads to an overactivation of glutamate pathways. As a result, increased glutamate levels cause hyperactive excitatory signalling in the PFC, since glutamate, the excitatory NT, is overexpressed, and GABA, the inhibitory NT, is inhibited. The combined reduction in inhibitory control and excitatory overactivation could influence the development of brain circuits, with potential implications for cognitive functions. These alterations also impair synaptic plasticity, as depicted by deficits in long-term potentiation (LTP) and synaptic pruning in preclinical models, at a developmental stage characterized by pruning [12].

Dysregulation observed in these studies aligns with NT abnormalities in schizophrenia, including reduced GABA signalling and increased glutamate activity in the PFC [21]. Adolescent cannabis exposure also affects the maturation of the endogenous cannabinoid system, with cannabinoid receptor 1 (CB1R) expression being downregulated, impairing synaptic transmission and neurodevelopment [22]. Sequentially, the neurobiological effects of overstimulation in the PFC disrupt normal brain maturation, leading to cognitive impairments.

Neuroimaging

Neuroimaging studies reveal that cannabis use during adolescence is associated with decreased PFC activation, a region critical for executive functions such as working memory and decision-making. Functional MRI data from cannabis-using adolescents show reduced activation in the dorsolateral PFC during cognitive tasks, correlating with impairments and memory deficits commonly seen in schizophrenia [3, 9]. James et al. (2009) also observed that

adolescent-onset schizophrenia cannabis users exhibited reduced white matter integrity and grey matter density loss in the PFC, particularly in the dorsolateral PFC and ventral striatum [9]. Thus, the findings suggest a logical progression, where molecular disruptions lead to structural and functional disturbances in circuits linked to cognitive dysfunction in schizophrenia.

Preclinical models accompany these findings by demonstrating cannabis-induced changes in dopaminergic signalling. Chronic cannabis exposure amplifies dopamine release in the mesolimbic pathway, increasing synaptic dopamine levels [4]. This high-dopamine state mirrors neurological abnormalities linked to schizophrenia risk. Moreover, these animal studies show that altered dopaminergic signalling leads to disrupted PFC activity, consistent with reduced activation observed in functional magnetic resonance imaging (fMRI) studies [1].

Additionally, some studies found FA reductions in cannabis users, measured via diffusion tensor imaging (DTI), suggesting compromised white matter in critical brain pathways [9, 10]. These white matter changes align with clinical findings of weakened PFC activity and further emphasize the link between adolescent cannabis use and increased susceptibility to schizophrenia through disrupted dopaminergic signalling and PFC mechanisms.

Behavioural Findings

Adolescent cannabis exposure, particularly to THC, has been shown to disrupt sensorimotor gating, a key cognitive function impaired in schizophrenia. Studies on adolescent rats demonstrate this through persistent PPI deficits following chronic cannabis treatment, a characteristic behaviour of schizophrenia [12, 13]. These impairments are believed to be linked to altered dopaminergic signalling in brain regions such as the ventral tegmental area and PFC, which play important roles in cognitive functions and psychotic symptoms [12]. PPI deficits in animals depict clinical manifestations such as disorganized thought and hallucinations, which are key symptoms of schizophrenia [14]. Behavioural findings reveal a sequential pattern, where neurochemical disruptions accumulate into observable behavioural impairments. These behavioural impairments suggest that cannabis exposure during adolescence may increase vulnerability to schizophrenia in adulthood, demonstrated by the persistence of PPI deficits long after the initial exposure [17].

In both animal and human studies, there are consistent findings linking adolescent cannabis use to disruptions in NT systems, cognitive impairments, and behavioural deficits resembling those seen in schizophrenia. Lesh et al. (2024) emphasized that an earlier age of starting cannabis use was associated with lower IQ and functioning, with heavier use linked to more severe positive symptoms of schizophrenia [8]. Johnson et al. (2023) also found that polygenic risk for schizophrenia was associated with increased reports of cannabis-related paranoia, cognitive difficulties, and social withdrawal among cannabis users [19]. Animal models of adolescent cannabinoid exposure exhibit schizophrenic symptoms such as reduced PPI, and working memory deficits, similar to cognitive and behavioural deficits observed in schizophrenia [14, 23].

Discussion

The results from this literature review highlight some consequences of adolescent cannabis exposure on brain development, particularly in the prefrontal cortex - the region that performs executive functions such as decision-making, attention, and impulse control [1]. Moreover, disruptions to the balance between excitatory and inhibitory neurotransmission in this region, especially in GABA and glutamate signalling, are important to understanding the lasting effects of cannabis on adolescent brain development. Neurotransmitter changes from cannabis use accumulate into a cascade of functional and behavioural manifestations. These same presentations are often implicated in the pathology of psychiatric disorders like schizophrenia, and the synthesis of these studies indicates that adolescent cannabis exposure may increase the risk of schizophrenia later in life.

Changes in Neurotransmitter Systems

Adolescent cannabis exposure has been consistently linked to disruptions in GABA signalling within the PFC. THC over-stimulates the CB1 (cannabinoid-1) receptors, interfering with normal NT processes, which play a key role in PFC maturation. This disruption in GABA signalling leads to an imbalance of excitatory (glutamate) and inhibitory (GABA) NTs, a phenomenon that is characteristic of the development of schizophrenia [24]. Several studies emphasize how cannabis use during adolescence prevents the full maturation of neurons in the PFC, potentially leading to long-term cognitive and behavioural disruptions [12, 20]. These disruptions are consistent with the cognitive deficits observed in schizophrenia, where GABA dysfunction in the PFC plays a prominent role.

Dopaminergic Signalling

The PFC is important in cognitive control, and disturbing its development can impair higher-order functions often disrupted in schizophrenia such as memory, attention, and decision-making. fMRI studies have demonstrated that the adolescent brain continues to mature structurally and functionally during this period [1]. Given that THC exposure disrupts these processes, the lasting effects on the PFC could contribute to cognitive deficits observed in schizophrenia [1, 12]. Changes in dopaminergic signalling within the mesolimbic pathway are also noteworthy. Adolescence is a period of significant dopaminergic development, and Lesh et al. (2024) found that early cannabis exposure contributes to alterations in

dopamine receptor expression, which can lead to a hyperactivated dopaminergic state [8]. This dysregulation is commonly seen in schizophrenia. Furthermore, the long-term effects of these dopaminergic changes are demonstrated in both animal and human studies, linking neurological changes from cannabis exposure similar to those in psychosis [10, 21]. For example, Aguilar et al. (2018) observed that rats exposed to synthetic cannabinoids during adolescence experienced lasting changes in dopamine neuron activity, indicating long-term impacts of cannabis on dopamine signalling [16].

Adolescent cannabis exposure also leads to significant changes in dopaminergic signalling in the mesolimbic (reward) pathway. Several studies have explored how adolescent cannabis use alters dopamine signalling, increasing the risk of schizophrenia. THC exposure during adolescence has been shown to change dopamine receptor expression and function, resulting in a hyperactive state. This dysregulation of reward processing and motivation may contribute to the development of psychotic symptoms, which are also affected in schizophrenia. There also may be a particularly higher risk in individuals with a genetic predisposition [10, 13, 21].

Behavioural Outcomes

Sensorimotor gating, a cognitive process often interrupted in schizophrenia, is assessed by PPI. PPI is a behavioural outcome often disrupted in schizophrenia, and these impairments are observed in several studies. Preclinical research indicates that adolescent THC exposure leads to long-lasting PPI deficits, suggesting that cannabis-induced disruptions in sensorimotor gating may contribute to the emergence of cognitive and behavioural changes [6, 23]. These findings highlight two roles of PPI: as an early indicator of vulnerability to schizophrenia, and as an outcome of the neurobiological disruptions from cannabis use.

The long-term impairments in PPI observed in animal models are consistent with human studies linking cannabis exposure to schizophrenia [22, 23]. The persistence of PPI deficits into adulthood suggests that early cannabis exposure may lead to lasting changes in brain function, compromising cognitive and behavioural regulation [14]. Aguilar et al. (2018) found that cannabinoid exposure in adolescent rats induced long-term changes in cognitive processes like sensorimotor gating, further linking these disruptions to schizophrenia-like behaviours [16]. Human studies show similar patterns of PPI deficits in cannabis users (particularly those with early onset of use), suggesting that sensorimotor gating impairments may serve as a marker of schizophrenia risk [14, 23].

Trends and Implications for Schizophrenia Risk

Across NT systems, dopaminergic signalling, and behavioural outcomes, a clear trend emerges: adolescent cannabis exposure disrupts brain development processes in ways that overlap with the pathophysiology of schizophrenia. The consistent findings across studies suggest that THC exposure during adolescence contributes to long-term changes in neural circuits controlling cognition and behaviour. Genetic predisposition appears to exacerbate these effects, indicating an interaction between environmental and biological factors in determining schizophrenia risk. Lesh et al. (2024) found that earlier cannabis use was associated with reduced IQ and cognitive functioning, which aligns with findings from Johnson et al. (2023), who reported that genetic predispositions for schizophrenia were linked to higher incidences of cognitive deficits [8, 19]. These studies suggest that even if adolescent cannabis use does not directly cause schizophrenia, it could significantly increase the risk in individuals already genetically predisposed to the disorder. These findings are consistent with preclinical studies, which demonstrate that cannabis-induced dopaminergic and cognitive disruptions interact with underlying genetic risk factors for schizophrenia [10, 21].

Limitations

Despite valuable insights provided by these studies, several limitations must be considered. Animal models, while useful in understanding the neurobiological effects of cannabis, may not fully replicate the complexity of human brain development or the variability in individual responses to cannabis exposure. Furthermore, human studies are limited by confounding factors, such as socio-economic status, genetic predisposition, and co-occurring substance use, making it difficult to isolate the specific effects of cannabis on the development of schizophrenia [12, 23]. Additionally, due to time constraints, a comprehensive search of academic and grey literature could not be completed, which may limit the scope of perspectives included in this review. Continued research is required to better understand the long-term consequences of adolescent cannabis exposure and its potential role in the development of schizophrenia.

Conclusions

This review outlines the significant neurobiological and behavioural consequences of adolescent cannabis use, particularly its role in increasing susceptibility to schizophrenia. Evidence from both animal and human studies consistently highlights disruptions in NT systems, especially GABA and glutamate signalling in the PFC, a region critical for executive functions. Neuroimaging studies and preclinical findings demonstrate how cannabis exposure during adolescence interferes with synaptic pruning, impairs PFC activation, and amplifies dopamine release, mirroring patterns observed in schizophrenia. Behavioural deficits such as sensorimotor gating impairments also demonstrate these associations.

The findings reinforce adolescence as a period of high vulnerability, where cannabis use disrupts neuro developmental processes, predisposing individuals to schizophrenia. These insights have profound implications

for public health, depicting the need for targeted prevention and education strategies with adolescents. Potential initiatives should focus on raising awareness, specifically to adolescents, about the dangers of early cannabis exposure, as well as providing avenues to mitigate downstream determinants of cannabis use. For example, if adolescents tend to abuse cannabis as a result of mental health struggles, a potential intervention is providing affordable services, such as counselling, to diminish these effects. Upstream determinants of cannabis use can also be addressed. For instance, if individuals coming from a low socioeconomic status tend to use cannabis at a younger age, educational messages should be concentrated on this group. These initiatives can be evaluated for effectiveness among diverse groups and integrated together in community and school programs that address individual and structural factors affecting adolescent cannabis use.

Future research should explore the possible reversibility of these effects and the role of genetics, as well as develop interventions to mitigate the long-term risks associated with early cannabis use. Understanding these mechanisms better can inform clinical practices and policy efforts to address such growing concerns surrounding adolescent cannabis use.

List of Abbreviations

CB1R: cannabinoid receptor 1
DTI: diffusion tensor imaging
FA: fractional anisotropy
fMRI: functional magnetic resonance imaging
GABA: gamma-aminobutyric acid
GAD: glutamic acid decarboxylase
IQ: intelligence quotient
LTP: long-term potentiation
MRI: magnetic resonance imaging
NT: neurotransmitter
PFC: prefrontal cortex
PPI: prepulse inhibition
THC: tetrahydrocannabinol

Conflicts of Interest

The author declares that they have no conflicts of interest.

Ethics Approval and/or Participant Consent

This study did not require ethics approval and/or participant consent as it was a review.

Authors' Contributions

CNR: sole author and responsible for database search, data collection and interpretation, study design, manuscript writing, and final approval for the results to be published.

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