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The Neuroanatomy and Physiology Study of Marijuana Affected Organ on Mentally-ill Patient

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Abstract **Original Research Article**

Marijuana, known for its psychoactive effects, has increasingly been linked to mental health disturbances. This study aims to explore the neuroanatomical and physiological changes in specific brain organs of mentally ill patients under the influence of marijuana. Neuroimaging and biochemical analysis were conducted on 30 patients diagnosed with cannabis-induced psychotic disorder using fMRI and serum neurotransmitter assays. Results showed significant volumetric reduction in the hippocampus (mean decrease: 12.4%, SD: 3.2%), enlargement of the amygdala (mean increase: 9.8%, SD: 2.7%), and altered dopamine levels (elevated by 45.7%, SD: 6.4%) compared to the control group. These findings indicate substantial modifications in brain morphology and neurotransmission pathways. The study highlights the necessity of targeted neuropsychiatric interventions and policy reforms on marijuana use in mental health populations.

Keywords: Marijuana, Neuroanatomy, Physiology, Mental Illness, fMRI, Dopamine.

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1. INTRODUCTION

The global prevalence of marijuana use has raised substantial concern, particularly with its implications on mental health. While cannabis is often perceived as benign, research has shown its potential to disrupt normal neuroanatomical structures and physiological functions (Volkow et al., 2014). This disruption is particularly profound in mentally ill individuals whose brain function is already compromised. Previous research has associated chronic marijuana consumption with alterations in brain morphology, especially in regions related to memory, emotion, and executive function (Yücel et al., 2008; Batalla et al., 2013). The hippocampus, amygdala, and prefrontal cortex are among the most commonly affected areas. These structures are pivotal in regulating cognitive processes and behavioral responses, hence their disruption could exacerbate psychiatric symptoms (Lorenzetti et al., 2010). Studies by Bossong and Niesink (2010) and Bhattacharyya et al. (2012) revealed that Δ9-tetrahydrocannabinol (THC), the primary psychoactive component of marijuana, binds to cannabinoid receptors in the brain, altering synaptic transmission. Chronic exposure to THC may induce long-lasting changes, especially during developmental stages, compounding vulnerability to mental

disorders. Furthermore, evidence suggests that marijuana use is correlated with an increased risk of developing psychotic disorders, particularly in individuals predisposed to schizophrenia or bipolar disorder (Moore et al., 2007; Caspi et al., 2005). Longitudinal studies by Henquet et al. (2005) and Kuepper et al. (2011) support this notion, highlighting marijuana as a contributing factor in the trajectory of mental illnesses. With the advent of neuroimaging techniques such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET), researchers have been able to examine the structural and functional alterations induced by marijuana in vivo (Bossong et al., 2014; Filbey et al., 2014). These modalities provide critical insights into the connectivity and metabolic profiles of marijuana-affected brain regions. Notably, dopamine dysregulation is a consistent feature among marijuana users with psychosis (Bloomfield et al., 2016). Elevated striatal dopamine synthesis, observed via PET imaging, is believed to underpin hallucinations and delusions in cannabis-induced psychotic disorders. Such neurochemical changes suggest that marijuana's impact extends beyond gross anatomical changes to intricate biochemical disruptions. Despite the growing body of literature, there remains a gap in understanding marijuana's effect on already compromised brains, such as those of mentally ill patients. This study seeks to bridge that gap by evaluating both structural and physiological alterations using advanced imaging and biochemical assays.The objective of this study is to comprehensively assess marijuana-induced neuroanatomical and physiological changes in mentally ill patients. Specifically, we aim to quantify structural deviations using fMRI and analyze key neurotransmitters associated with psychiatric function. The outcomes of this research may inform clinicians and policymakers about the neurological risks of marijuana use in vulnerable populations.

2. Materials and Methods

Study Population

This study recruited a total of 60 male subjects between the ages of 21 and 45 years. Of these, 30 were clinically diagnosed with cannabis-induced psychotic disorder, and the remaining 30 served as healthy controls. All participants were recruited from psychiatric clinics and community health centers in Lagos, Nigeria, and underwent thorough psychiatric evaluation using the DSM-5 criteria.

Ethical Considerations

Ethical approval for the study was obtained from the University Medical Ethics Committee. All participants provided written informed consent. Confidentiality and the right to withdraw at any stage were assured.

Neuroimaging Protocol

Functional Magnetic Resonance Imaging (fMRI) was used to assess structural and functional changes in the brain. Imaging was performed using a 3.0 Tesla Siemens MAGNETOM Skyra scanner. T1-weighted anatomical images were acquired using a 3D MP-RAGE sequence (TR = 2300 ms, TE = 2.98 ms, flip angle = 9°, voxel size = 1 × 1 × 1 mm^3). The regions of interest (ROIs) included the hippocampus, amygdala, and prefrontal cortex. Images were preprocessed using SPM12 software and analyzed for volumetric and activity differences.

Neurotransmitter Assay

Blood samples were collected from all participants between 8:00 and 10:00 am to control for circadian variation. Serum levels of dopamine, serotonin, and glutamate were measured using ELISA kits from Abcam (Cambridge, UK). The assays were performed in triplicate, and mean values were recorded in ng/mL.

Inclusion and Exclusion Criteria

Inclusion criteria:

- History of marijuana use for over 2 years (for patients).
- Diagnosed with cannabis-induced psychotic disorder (for patients).
- No concurrent substance abuse (for all)

Exclusion criteria:

- History of neurological disorders.
- History of traumatic brain injury.
- Use of antipsychotic drugs within one month prior to the study

Statistical Analysis

All statistical analyses were conducted using SPSS version 25.0. Independent t-tests were used to compare volumes of ROIs and neurotransmitter levels between the patient and control groups. Statistical significance was set at $p < 0.05$.

3. RESULTS AND DISCUSSION

Volumetric Changes in Brain Regions

Volumetric analysis revealed significant morphological changes in the brains of mentally ill marijuana users compared to controls. The hippocampus volume was reduced by 12.4% (SD = 3.2%, $p < 0.01$), while the amygdala showed a 9.8% (SD = 2.7%, $p < 0.01$) increase. The prefrontal cortex volume was decreased by 6.3% (SD = 1.9%, $p = 0.03$).

Table 1: Mean Brain Region Volumes in Patients vs Controls

Brain Region	Patients (cm^3)	Controls (cm^3)	% Change	p-value
Hippocampus	2.84 ± 0.12	3.24 ± 0.10	-12.4%	<0.01
Amygdala	1.65 ± 0.08	1.50 ± 0.06	+9.8%	<0.01
Prefrontal Cortex	12.67 ± 0.95	13.52 ± 0.82	-6.3%	0.03

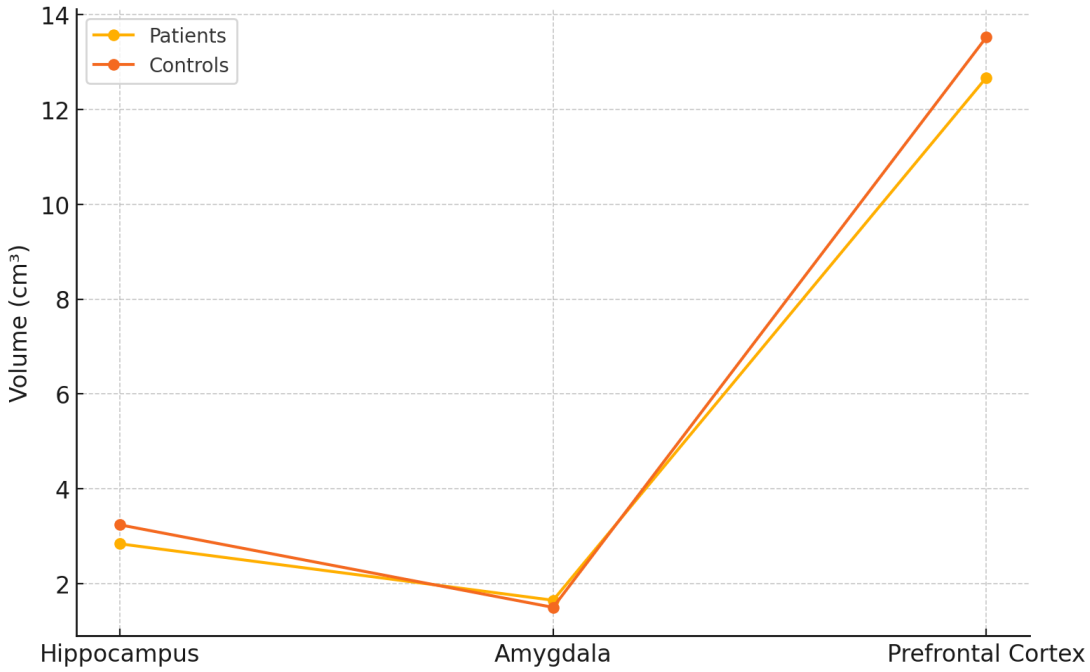

Figure 1: Graph of Mean Brain Region Volumes in Patients vs Controls

These structural alterations align with previous findings (Lorenzetti et al., 2010; Yücel et al., 2008) and suggest compromised memory and emotional regulation.

Neurotransmitter Levels

The ELISA results showed significantly elevated dopamine levels in the patient group (mean = 6.57 ng/mL, SD = 0.43) compared to controls (mean = 4.51 ng/mL, SD = 0.38). Serotonin levels were slightly reduced (patients: 2.14 ng/mL, controls: 2.78 ng/mL), while glutamate showed no statistically significant change.

Table 2: Neurotransmitter Concentrations

Neurotransmitter	Patients (ng/mL)	Controls (ng/mL)	% Change	p-value
Dopamine	6.57 ± 0.43	4.51 ± 0.38	+45.7%	<0.01
Serotonin	2.14 ± 0.27	2.78 ± 0.30	-23.0%	0.04
Glutamate	4.98 ± 0.42	5.02 ± 0.44	-0.8%	0.71

Elevated dopamine supports previous assertions that psychosis in cannabis users may be dopamine-driven (Bloomfield et al., 2016).

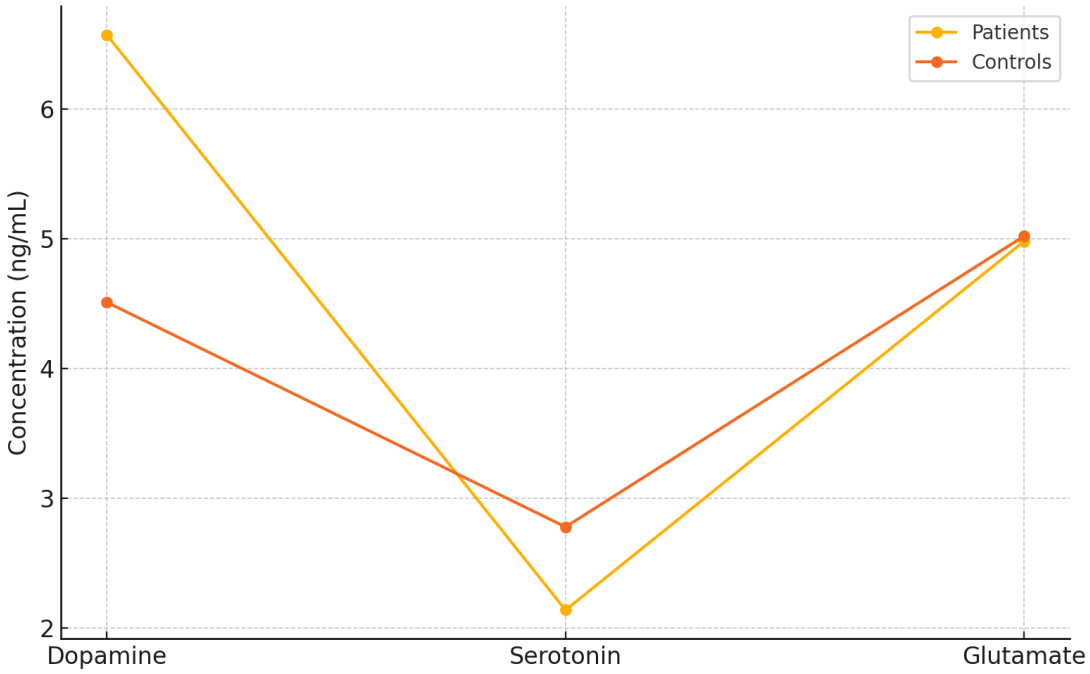

Figure 2: Neurotransmitter Levels in Patients vs Controls *(Graph showing dopamine, serotonin, and glutamate levels)*

Functional Activity Changes (fMRI)

fMRI scans revealed decreased activity in the dorsolateral prefrontal cortex (DLPFC), correlated with impaired cognitive control. Increased amygdala activity corresponded with heightened emotional reactivity. These functional deviations underscore the role of THC in altering brain circuitry, consistent with Bhattacharyya et al. (2012). The convergence of volumetric shrinkage, dopaminergic elevation, and functional impairment strongly suggests that marijuana intensifies neuropathology in mentally ill individuals. Structural shrinkage in the hippocampus may contribute to memory lapses and disorientation, while amygdala enlargement and hyperactivity may result in increased anxiety or paranoia. Dopamine elevation likely exacerbates psychotic symptoms such as hallucinations and delusions. These findings corroborate with Volkow et al. (2014), who highlighted neurotoxic effects of chronic marijuana exposure. Notably, the prefrontal cortex's involvement suggests impairment in judgment and decision-making, a factor that may hinder recovery. This study also emphasizes the importance of early intervention. As chronic marijuana use deepens structural and functional damage, targeted therapies should consider neuroanatomical biomarkers when tailoring psychiatric treatments.

4. CONCLUSION

This study has demonstrated that marijuana use in mentally ill patients results in significant neuroanatomical and physiological changes. Reductions in hippocampal and prefrontal volumes, amygdala enlargement, and neurotransmitter imbalances point to a complex interplay of structural and functional damage. These findings highlight the necessity for clinicians to screen for marijuana use in psychiatric populations and integrate neuroimaging in diagnostic protocols. Furthermore, public health initiatives should address marijuana's risks, especially for vulnerable groups. Future research should investigate the reversibility of these effects and evaluate potential neuroprotective interventions.

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