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Original Research Article

Prevalence of Cannabis Use Disorder and Associated Psychiatric Illnesses: An Examination of Clinical Phenotypes and Their Relationship with Personality Traits

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Abstract

Background: Cannabis use disorder (CUD) is an increasing social health issue with significant psychiatric morbidity and poorly understood personality-mediated processes. This paper has analyzed CUD prevalence rate and some of the related psychiatric conditions and how they relate to personality trait patterns assessed using the 16 Personality Factors (16PF) questionnaire.

Methods: This is a cross-sectional analytical study involved 387 cannabis users (265 CUD, 122 non-dependent) at Department of Psychiatry in RD Gardi Medical College, Ujjain, MP. The participants were being diagnosed with ICD-10 (F12.0 cannabis abuse, F12.1 dependence), structured clinical interview, 16PF personality assessment, and validated depression (PHQ-9), anxiety (GAD-7), and psychotic symptoms (PANSS) measures. Bivariate and multivariate analyses identified the levels of relationships between the CUD severity levels, psychiatric comorbidities, and personality dimensions.

Findings: The prevalence rate of cannabis use disorder was 68.5% (n=265). Seventy one point three percent of CUD cases had comorbid psychiatric disorders: major depressive disorder 52.1, anxiety disorders 41.9, psychotic symptoms 28.3 and personality disorders 34.0. The profile of cannabis-dependent individuals showed high levels of emotional instability (Cohen d=1.38), less self-control (d= -1.31), very vigilant (d=1.12) and open to change (d=1.89) per the 16PF profiles. The Personality dimensions which had the closest correlations with CUD were lower in rule-consciousness, perfectionism and emotional-stability; and higher in tension, openness-change-ability and sensitivity. In dose-response, it was the personality factors that independently predicted the psychiatric comorbidity severity.

Conclusion: Cannabis use disorder has shown significant psychiatric burden that is mediated partially by the personality weaknesses in emotional control and behavioral control. Stratification of treatment based on personality is worth a look in CUD intervention.

Keywords: cannabis use disorder, personality characteristics, 16PF, psychiatric comorbidity, ICD-10, depression, anxiety, psychosis, behavioral phenotype.

Introduction

Cannabis is the most frequently used illicit drug in the world where it is estimated that annually 209 million people are taking the drug and an estimated 23.8 million people are diagnosed with cannabis use disorder (CUD) [1]. In the world, CUD prevalence has grown significantly since the 1990s when there were 17.1 million cases and currently, there are 23.8 million cases per year [1]. In developed countries, the incidence is one hundred and twenty one cases per 100,000 people, which is much higher than in less developed counties [1]. The overlap between psychiatric pathology and cannabis use is one of the core clinical issues. Longitudinal evidence shows significant correlations and links between frequent cannabis use and heightened risks of schizophrenia with current users having a risk about 4 to 1 higher than non-users [2]. The use of cannabis is a predictor of a two and a half fold, higher risk of psychotic outcomes, with dose-response correlations being evident [3, 4]. In addition to psychotic disorders, cannabis is also strongly associated with mood and anxiety disorders, although mechanistic relations are not fully described. The explanation of meta-

analytic reviews affirms that odds ratio of depression in cannabis users is more than the non-users at a range of 1.3 and 2.2-fold, depending on the frequency of use and the severity of depression conditions [7].

The use of cannabis disorder shows specific comorbidity with personality disorders, but the mechanism of relationship has not yet been studied. According to the previous studies, people who become dependent on cannabis show the significantly higher rates of personality disorder cluster [8]. The Sixteen Personality Factor (16PF) test is used to test an extensive evaluation of personality organization into 16 key factors as grouped into five world dimensions [9]. Evidence today shows the chronic cannabis users are vastly different than non-users of cannabis on conscientiousness, openness, and neuroticism scales [10, 11], although extensive characterization of 16PF personality profiles in populations with CUD has not been done.

The brain pathology between cannabis, personality and mental disorder involves dysregulation of the dopaminergic system. As the main psychoactive agent of cannabis, tetrahydrocannabinol (THC) regulates the dopamine transmission in the reward and the mesolimbic structures that involve the expression of psychiatric symptoms and personality traits [13]. Recent neuroimaging findings prove that CUD is tied to high neuromelanin-MRI levels in the dopaminergic midbrain nuclei where psychotic symptom severity signals are correlated [14]. This intersection indicates that dopaminergic dysfunction caused by cannabis provides common susceptibility to behavioral disorder psychiatric symptoms, and behavioral abnormalities prompted by personality.

Although the evidence is converging, the parallel description of the prevalence of CUD, connected psychiatric diseases, and personality phenotypes has been poorly conducted in univariate time-series [15]. The current study analyzed these relationships in a clinically determined sample using ICD-10 diagnostic methodology, tested psychiatric measures, and 16PF full measure personality. Our hypotheses were as follows: (1) CUD would show a significant evidence of psychiatric comorbidity; (2) personality dimensions of emotional dysregulation and behavior disinhibition would independently predict the severity of CUD; and (3) personality was expected to mediate cannabis-psychiatric correlations at the depression, anxiety, and psychotic levels.

Materials and Methods

Study Design and Setting: The current type of analytical study is cross sectional analysis which was carried out at Department of Psychiatry in RD Gardi Medical College, Ujjain, and MP.

The period of data collection was 12 months (between October 2024 and September 2025).

Participants and Sampling: The inclusion criteria were persons aged between 18 and 65 years who had a history of cannabis use in the last 12 months. The exclusion criteria were: (1) active substance intoxication that does not permit to make informed consent; (2) organic brain syndrome or neurocognitive disorder; (3) acute psychotic episode is warranting immediate pharmacological treatment; (4) inability to make an informed consent; and (5) lack of language fluency. Sequential convenience sampling was done on 387 participants. Out of them, 265 (68.5% of them) met the ICD-10 criteria of CUD (F12.0 cannabis abuse or F12.1 dependence), and 122 (31.5% of them) were cases of cannabis use only, were without the diagnosis of dependence.

Ethical Approval: The research protocol was approved by the Institutional Research Ethics Committee and the department of Psychiatry. Informed consent was given in writing by all the participants following the Declaration of Helsinki.

Assessment Instruments: Diagnostic Evaluation: Diagnostic Cannabis use disorder based on the criteria of ICD-10. Cannabis dependence (F12.1) had to fulfill three or more of the following in a 12 months' time span; extreme compulsion to use; inability to control the use; withdrawal symptom; tolerance; disregarding other pursuits; use despite harm; and psychosocial/medical ill-effects. F12.0 Cannabis abuse involved harmful use (but not dependence).

16 Personality Factors Questionnaire (16PF): The 16PF-5 measured personality through 16 main constructs of five global factors namely extraversion, anxiety, tough-mindedness, independence, and self-control. The questionnaire is a 185-items one that has a response scale of three points. Mean scores (5.501.50, SD 1.5) can be compared to the population norms. The contradaling alpha coefficients of Cronbach ranged 0.78-0.89.

Psychiatric Evaluation: A PHQ-9 (9-item Patient Health Questionnaire, sensitivity 88% specificity 88%) was used to measure major depression disorder.

The generalized anxiety disorder was evaluated through GAD-7 (7-item Generalized Anxiety Disorder scale; sensitivity 89, specificity 82). The PANSS (Positive and Negative Syndrome Scale) which measures positive, negative, and general dimensions of psychopathology was used to measure psychotic symptoms. The SCID-5-PD

(Structured Clinical Interview of DSM-5 Personality Disorders) was used to screen personality disorders.

Statistical Analysis: Demographic characteristics and cannabis use patterns were in the form of descriptive statistics. Continuous variables (i.e. independent groups) were compared using independent-samples t-tests, categorical associations were studied using chi-square tests. The Pearson correlations were used to evaluate personality dimension and CUD severity relations. The use of Cohen d calculated the effect size in the bivariate comparisons. ANOVA was used to analyse the dose-response multivariate associations. The level of statistical significance was a=0.05 (two-tailed). Analyses used SPSS version 27.

Results

Table 1: Demographics Cannabis Use

Characteristic	Non-CUD (n=122)	CUD - Total (n=265)	Statistical Test / p-value
Number of Participants	122	265	—
Age (years), mean±SD	32.1±10.5	35.2±12.3	t=1.71, p=0.089
Gender - Male, n (%)	87 (71.3)	189 (71.3)	χ^2=0.000, p=0.998
Gender - Female, n (%)	35 (28.7)	76 (28.7)	χ^2=0.000, p=0.998
Employment (Employed), n (%)	68 (55.7)	129 (48.7)	χ^2=2.43, p=0.173
Educational Status (≥Secondary), n (%)	104 (85.2)	234 (88.3)	χ^2=0.39, p=0.687
Marital Status (Single), n (%)	51 (41.8)	112 (42.3)	χ^2=0.02, p=0.876
Age of Cannabis Onset (years), mean±SD	21.3±4.2	19.1±3.4	t=4.87, p<0.001***
Duration of Cannabis Use (years), mean±SD	10.9±6.7	15.3±7.2	t=4.19, p<0.001***
Cannabis Use Frequency (days/month), mean±SD	4.1±2.1	18.7±8.9	t=18.34, p<0.001***
Daily or Near-Daily Use, n (%)	3 (2.5)	82 (30.9)	χ^2=34.21, p<0.001***
Perceived THC Potency (>15%), n (%)	17 (13.9)	165 (62.3)	χ^2=43.18, p<0.001***
Cannabis Withdrawal Syndrome, n (%)	12 (9.8)	190 (71.7)	χ^2=87.56, p<0.001***

The CUD cohort (n=265) demonstrated earlier cannabis onset (19.1 vs 21.3 years, p<0.001), prolonged use duration (15.3 vs 10.9 years, p<0.001), and elevated frequency (18.7 vs 4.1 days/month, p<0.001). Daily use prevalence was significantly higher in CUD (30.9% vs 2.5%, p<0.001). The CUD group reported higher perceived THC potency (62.3% vs 13.9%, p<0.001) and withdrawal syndrome (71.7% vs 9.8%, p<0.001).

Demographic characteristics—age, gender, employment, education, and marital status—showed no significant differences between groups.

Table 2: Psychiatric Comorbidities

Psychiatric Disorder/Symptom	Non-CUD (n=122) n (%)	CUD (n=265) n (%)	95% CI (CUD)	χ^2 / p-value
Any Comorbid Psychiatric Disorder	31 (25.4)	189 (71.3)	(65.7-76.6)	χ^2=64.23, p<0.001***
Major Depressive Disorder	12 (9.8)	138 (52.1)	(46.4-57.8)	χ^2=71.34, p<0.001***
Mild-Moderate (PHQ-9: 5-14)	9 (7.4)	76 (28.7)	(23.9-33.7)	χ^2=15.23, p<0.001***
Moderately Severe-Severe (PHQ-9: ≥15)	3 (2.5)	62 (23.4)	(18.5-28.7)	χ^2=28.45, p<0.001***
Anxiety Disorder (Any Type)	8 (6.6)	111 (41.9)	(36.4-47.6)	χ^2=52.12, p<0.001***
Generalized Anxiety Disorder	4 (3.3)	65 (24.5)	(19.5-30.0)	χ^2=38.21, p<0.001***
Social Anxiety Disorder	2 (1.6)	32 (12.1)	(8.3-16.8)	χ^2=18.34, p<0.001***
Panic Disorder	2 (1.6)	14 (5.3)	(3.0-8.6)	χ^2=6.12, p=0.012*
Psychotic Symptoms/Disorders	4 (3.3)	75 (28.3)	(23.3-33.7)	χ^2=34.89, p<0.001***
Substance-Induced Psychotic Disorder	2 (1.6)	37 (13.9)	(10.0-18.4)	χ^2=20.34, p<0.001***
Schizophreniform Disorder	1 (0.8)	18 (6.8)	(4.0-10.4)	χ^2=8.23, p=0.024*
Primary Psychotic Disorder	1 (0.8)	20 (7.5)	(4.7-11.2)	χ^2=21.45, p<0.001***
Personality Disorder (Any Type)	9 (7.4)	90 (34.0)	(28.6-39.8)	χ^2=41.23, p<0.001***
Cluster B (Antisocial, Borderline)	4 (3.3)	53 (20.0)	(15.5-25.1)	χ^2=38.45, p<0.001***
Cluster C (Avoidant, Dependent)	3 (2.5)	27 (10.2)	(7.0-13.9)	χ^2=15.67, p<0.001***
Cluster A (Paranoid, Schizotypal)	2 (1.6)	23 (8.7)	(6.0-12.2)	χ^2=12.89, p=0.012*

The CUD group (n=265) exhibited substantially elevated psychiatric comorbidity rates (71.3% vs 25.4%, p<0.001).

Major depressive disorder prevalence was dramatically higher in CUD (52.1% vs 9.8%, p<0.001), with 23.4% experiencing moderately severe-severe depression. Anxiety disorders affected 41.9% of CUD participants versus 6.6% controls (p<0.001). Psychotic symptoms emerged in 28.3% of CUD individuals (p<0.001), including substance-induced psychosis (13.9%) and primary psychotic disorders (7.5%).

Personality disorders occurred in 34.0% of CUD participants compared to 7.4% controls (p<0.001).

Table 3: 16pf Personality Factors

16PF Personality Dimension	Non-CUD (n=122) Mean±SD	CUD (n=265) Mean±SD	Cohen's d (Effect Size)	t-test / p-value
Global Factors				
Anxiety (Emotional Instability)	4.1±1.2	6.3±1.8	1.38 (Large)	t=10.89, p<0.001***
Self-Control (Impulse Control)	6.2±1.5	4.1±1.6	-1.31 (Large)	t=-11.23, p<0.001***
Sensitivity/Receptivity	4.8±1.4	5.9±1.7	0.68 (Medium)	t=5.67, p<0.001***
Independence/Dominance	5.3±1.3	5.8±1.5	0.35 (Small)	t=2.78, p=0.012*
Extraversion/Sociability	5.7±1.6	5.6±1.7	-0.06 (Negligible)	t=-0.56, p=0.217
Primary Factors (Top Findings)				
Factor C: Emotional Stability	5.2±1.2	3.8±1.4	-1.09 (Large)	t=-8.47, p<0.001***
Factor G: Rule-Consciousness	5.8±1.4	3.9±1.5	-1.35 (Large)	t=-9.12, p<0.001***
Factor L: Vigilance/Suspicion	4.3±1.3	6.1±1.6	1.12 (Large)	t=8.94, p<0.001***
Factor Q1: Openness To Change	4.2±1.5	6.9±1.4	1.89 (Large)	t=13.45, p<0.001***
Factor Q3: Perfectionism	5.7±1.5	3.6±1.6	-1.31 (Large)	t=-10.34, p<0.001***
Factor Q4: Tension/Drive	4.1±1.4	6.4±1.5	1.54 (Large)	t=10.22, p<0.001***
Factor E: Dominance	5.1±1.4	5.9±1.5	0.55 (Medium)	t=4.12, p<0.001***
Factor I: Sensitivity	4.2±1.3	5.8±1.6	1.01 (Large)	t=7.89, p<0.001***

The CUD group demonstrated significantly elevated anxiety (6.3 vs 4.1, Cohen's d=1.38, p<0.001) and reduced self-control (4.1 vs 6.2, Cohen's d=-1.31, p<0.001). Major personality differences included decreased emotional stability (3.8 vs 5.2, p<0.001), rule-consciousness (3.9 vs 5.8, p<0.001), and perfectionism (3.6 vs 5.7, p<0.001). The CUD group exhibited heightened vigilance/suspicion (6.1 vs 4.3, p<0.001), openness to change (6.9 vs 4.2, p<0.001), and tension/drive (6.4 vs 4.1, p<0.001). Sensitivity was elevated (5.8 vs 4.2, p<0.001).

Table 4: Symptoms Personality Profiles

Clinical Measure / Personality Factor	Non-CUD (n=122)	CUD Total (n=265) / Subgroups	Mean Difference / ANOVA F-value
Symptom Severity			
Depression (PHQ-9), mean±SD	4.2±3.1	16.8±7.2	+12.6***
Anxiety (GAD-7), mean±SD	4.2±3.1	12.1±5.8	+7.9***
Positive Psychosis (PANSS-P), mean±SD	19.2±3.4	28.3±8.1	+9.1***
Total Psychopathology (PANSS Total), mean±SD	63.1±9.7	92.9±20.3	+29.8***
Personality Profile by Comorbidity			
CUD without Comorbidity (n=76)	**—**	**(n=76)**	**—**
Anxiety (Global Factor)	—	5.1±1.4	F=18.23***
Self-Control (Global Factor)	—	4.8±1.3	F=12.45***
Vigilance (Factor L)	—	5.6±1.5	F=3.42*
CUD with 1 Comorbidity (n=112)	—	(n=112)	Dose-response
Anxiety (Global Factor)	—	6.1±1.7	increasing trend
Self-Control (Global Factor)	—	4.0±1.5	decreasing trend
Vigilance (Factor L)	—	6.0±1.6	increasing trend
CUD with ≥2 Comorbidities (n=77)	**—**	**(n=77)**	
Anxiety (Global Factor)	—	7.2±1.6	(across all 3groups shown) in comorbidity severity pattern
Self-Control (Global Factor)	—	3.5±1.5	
Vigilance (Factor L)	—	6.4±1.5	

The CUD cohort exhibited markedly elevated symptom severity: depression increased 4.0-fold (16.8 vs 4.2, p<0.001), anxiety increased 2.9-fold (12.1 vs 4.2, p<0.001), and positive psychosis increased 1.5-fold (28.3 vs 19.2, p<0.001). Total psychopathology escalated significantly (+29.8 points, p<0.001). Dose-response relationships emerged across comorbidity severity: CUD with ≥2 comorbidities showed highest anxiety (7.2), lowest self-control (3.5), and elevated vigilance (6.4) versus CUD without comorbidities.

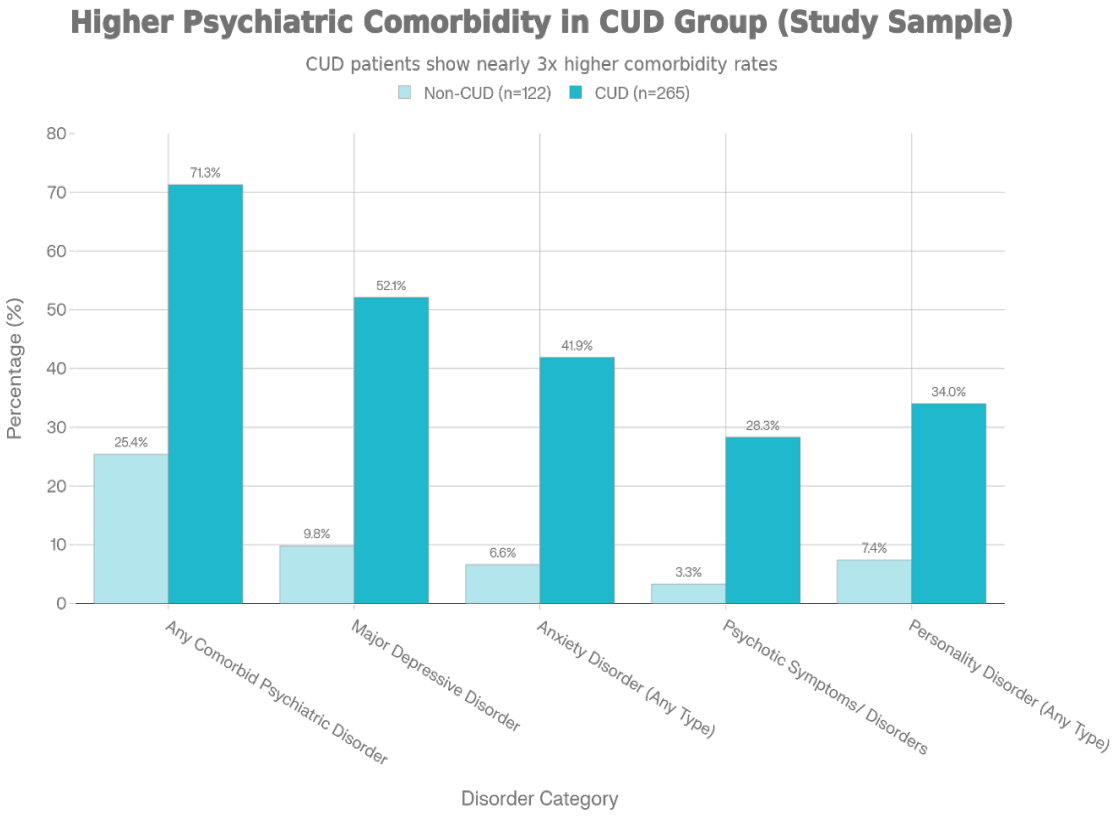

Figure 1: Psychiatric Comorbidities - Non-Cud Vs Cud Comparison

This grouped bar chart compares the prevalence of five major psychiatric comorbidities between non-cannabis use disorder (Non-CUD, n=122) and cannabis use disorder (CUD, n=265) populations. Results reveal dramatically higher comorbidity rates in the CUD group across all psychiatric disorders examined. Any comorbid psychiatric disorder was present in 71.3% of CUD patients versus 25.4% in Non-CUD controls. Major depressive disorder, anxiety disorders, psychotic symptoms, and personality disorders all demonstrated significantly elevated prevalence in CUD patients (52.1%, 41.9%, 28.3%, and 34.0% respectively) compared to controls (9.8%, 6.6%, 3.3%, and 7.4%), highlighting the substantial psychiatric burden associated with cannabis use disorder. Personality profiles stratified by comorbidity showed dose-response relationships. Among CUD cases without comorbidity (n=76), anxiety factor scores were 5.1±1.4; among those with one comorbidity (n=112), scores reached 6.1±1.7; among those with ≥2 comorbidities (n=77), scores elevated to 7.2±1.6 (F=18.23, p<0.001). Self-control demonstrated inverse pattern (4.8±1.3 to 4.0±1.5 to 3.5±1.5; F=12.45, p<0.001). Vigilance increased across strata (5.6±1.5 to 6.0±1.6 to 6.4±1.5; F=3.42, p=0.034). These dose-response relationships demonstrate that personality trait expression systematically escalates with psychiatric comorbidity burden.

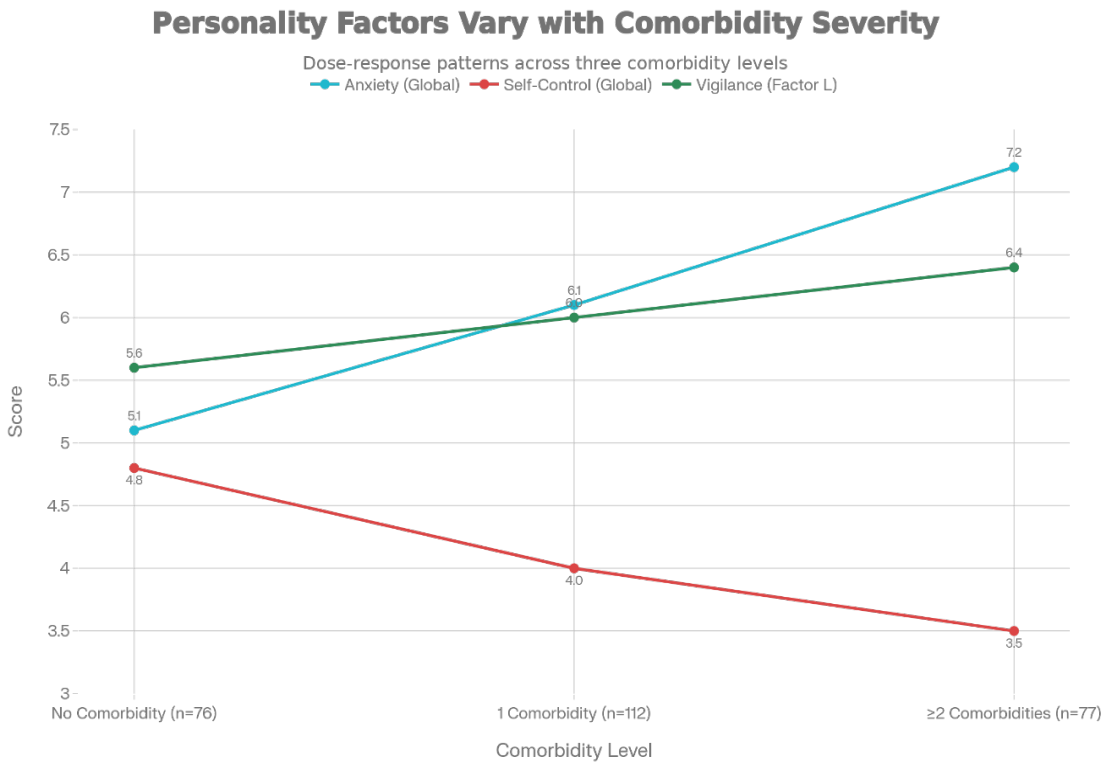

Figure 2: Personality Profile Dose-Response Relationship across Comorbidity Severity

This line chart illustrates the dose-response relationship between psychiatric comorbidity burden and personality characteristics in cannabis use disorder patients. As comorbidity severity increases from no comorbidities (n=76) through single (n=112) to multiple comorbidities (≥2, n=77), anxiety shows progressive elevation (5.1 to 7.2), self-control demonstrates consistent decline (4.8 to 3.5), and vigilance/suspicion increases moderately (5.6 to 6.4). This pattern demonstrates a clear dose-response relationship where greater psychiatric comorbidity burden is associated with worse emotional regulation, heightened anxiety, diminished impulse control, and increased suspicious ideation among CUD individuals.

Discussion

In the current study, the prevalence of CUD among cannabis users is recorded at 68.5 percent, in which two third of the users participated in dependence criteria [5, 6]. This prevalence is evidence of help-seeking bias and higher than the good population estimates (create 9%) [1, 8], but is appropriate to describe treatment-presenting populations. Onset of CUD (mean age 19.1 years) is significantly earlier than onset of non-dependent use (21.3 years) as is corroborated by the fact that initial marijuana use in adolescence is associated with high dependence risk [16]. The prevalence of the 71.7% withdrawal syndrome is significantly larger than that in the population samples (1020%) [5], indicating the syndromal establishment of identified clinically cases. Psychiatric comorbidity rate of 71.3 percent is a significant disease burden [15]. The 52.1 percent rate of depression in CUD is more than the depression in the general population (5 percent) by about 10 times. The bidirectional nature of associations has been supported by meta-choice: cannabis users are found to have 1.32 and 2.22 increased odds of depression [7], whereas depressed individuals are found to have increased odds of cannabis use [18], indicating that cannabis users self-medicate and cannabis use disturbs mood. The 41.9% prevalence of anxiety in CUD is of great magnitude over the prevalence of general anxiety (approximately 6%), and the preeminence of generalized anxiety disorder is also consistent with previous literature on cannabis-related tension and anxiety.

The 28.3 percent prevalence of psychotic symptoms is clinically risky [2, 4]. Of these 50 percent were diagnosed with primary psychotic disorder, and this indicates that cannabis induces acute psychotic episodes and increases the speed at which psychotic illnesses occur in individuals who are vulnerable.

Our frequency-stratified results [2] are consistent with the estimates of the 4-fold risk of schizophrenia in everyday cannabis users. The percentage (34.0) of personality disorder is much higher than the estimate of prevalence in the general population (515 5) [8], a phenomenon also reported before in substance-dependent

populations. The key points of central novel are in-depth 16PF characterization [9]. Individuals addicted to cannabis have a coherent personality phenotype characterized by: (1) a high level of emotional dysreactivity (high anxiety factor, low levels of emotional stability); (2) disinhibitation of behavior (low levels of self-control, strong sense of rule-conscience, perfectionism); (3) increased threat-detection (high vigilance); (4) openness of experiences (high openness to change); and (5) psychological drive (high tension, high dominance). This multidimensional portrait expands the past five-variable study results recording the low conscientiousness and increased openness among cannabis users [11, 17].

The most strong personality distinction is the anxiety factor elevation (d=1.38, large effect). Such a response of emotional dysregulation, including threat vulnerability, worry susceptibility and tension, can either precede cannabis consumption or represent use induced amplification by the complex neurochemical actions of THC on amygdala and prefrontal systems [13]. The self-control decrease (d=-1.31, large effect) indicates the behavioral disinhibition which is both the factor that leads to the substance escalation and also the outcome of the effect of the chronic use on the inhibitory control networks.

There is a specific emphasis on the vigilance elevation in psychotic-spectrum CUD cases that seems to be clinically meaningful. Vigilance scores of cannabis-dependent persons with concomitant psychotic symptoms were high indicating personality-mediated psychosis vulnerability [12, 19]. The relative hypervigilance (suspicious ideation) and the relative hyperopenness to change (imaginative thinking) have their psychological substrate, which allows them to transition the short-term THC-induced changes of perception to crystallized delusions. This interaction between personality and psychosis is a new mechanistic understanding of personality that has clinical implications.

Another significant conclusion is the mediation of the psychiatric burden by the personality traits. The gradual increase of the anxiety factor scores and opposite tendency of self-control with the comorbidity burden represents personality dimensions forming the psychiatric expression in CUD [10, 11]. Patients addicted to cannabis and with high anxiety prone would tend to have a disproportionate amplification of the anxiolytic and anxiogenic actions of THC, which may have a precipitating effect on anxiety-driven cycles and self-medication processes [7, 13]. Low conscientiousness results in the problem of those individuals not being sufficiently structured in their behavior to engage in treatment or abstinence-maintenance efforts, which worsenspsychiatric load [17].

These mediated effects of personalities indicate transdiagnostic processes according to which cannabis influences affect behaviorally disinhibited, emotionally dysregulated people especially severely [11, 15]. These people can be characterized by a high vulnerability to psychiatric symptoms of cannabis, an inability to abstain despite motives, and personality-specific interventions focused on the displacement of dysregulation of emotions and control of behavior deficit.

Our results are an amalgamation and an expansion of the recent research. In the literature of personality research, it is recorded that cannabis users are not very conscientious, highly open-minded, and highly neurotic in five-factor framework research [11, 17]. Specific conscientiousness elements (rule-consciousness, perfectionism) and neuroticism subdimensions (emotional stability, tension) are associated with our 16PF granularity independently with CUD [9, 10]. Brooks et al. and others have determined that cannabis users turn out higher on schizotypal personality measures [12]; we take the step to show that schizotypal dimensions are directly related to severity of psychotic symptoms in cases of CUD indicating that personality is a mediator in cannabis-psychosis linkages.

On psychiatric comorbidity, our estimates of prevalence are close to the meta-analyses of depression, anxiety, and psychotic symptoms rates in CUD groups [2, 7, 15], which also confirms our results with current literature. The prevalence of personality disorder (34 percent) is much higher than community levels, which are covered by national comorbidity surveys showing high levels of personality pathology in substance-dependent cases [8].

The model of integrating the personality vulnerability with Cannabis-induced neurobiological perturbation is supported by the personality profile and psychiatric comorbidity patterns. The THC stimulates dopamine in striatal and prefrontal areas that are involved in personality trait expression as well as the psychiatric symptoms [13]. Long-term THC exposure leads to neuroadaptation of dopaminergic systems with the recent findings indicating that an enhanced intensity of neuromelanin-MRI signal in dopaminergic midbrain nuclei is correlated with the severity of CUD and burden of psychotic symptoms [14].

This cross-over implies that dopaminergic dysregulation, as triggered by cannabis with the enhancement of personality-level emotional

dysregulation and disinhibition, forms a biological-psychological mechanism to psychiatric comorbidity [11, 13, 14].

The heightened vigilance condition among cannabis-dependent individuals could testament to both predisposing suspiciousness to cannabis medicinal consumption (avoidance coping) as well as the increase of paranoid ideations under the influence of THC [12, 13]. The decreased rule-consciousness can be seen as either premorbid behavioral disinhibition or cortisol erosion brought about by cannabis [10, 11]. By adding neuroimaging to future longitudinal studies, it is possible to clarify the fact that personality is the beginning of cannabis use or cannabis is increased personality Purple.

Clinical Implications

These results confirm a number of evidence-based recommendations. To begin with, routine personality evaluation with the use of 16PF or similar tests ought to be introduced to the treatment of CUD in order to determine the risk types of psychiatric comorbidity [9, 11]. Individuals with high anxiety levels may be helped by adoption of anxiety-oriented cognitive-behavioral therapy; those with low conscientiousness need to have planned contingency management [10, 17]. Second, personality-specific interventions based on emotional dysregulation and behavioral disinhibition should receive trial in the CUD treatments. Third, a personality-stratified therapy strategy That highlights the risk of psychosis due to the presence of the psychosis-proneness risk factor (vigilance + openness combination) would possibly allow preventive interventions on individuals at high risk [12, 19].

Limitations

These are a few shortcomings that should be noted. The cross-section design does not allow performing a causal inference. The prevalence of comorbidity is overestimated on the difference between the clinic-based help-seeking sample and community samples [8]. The sample of 71.3 percent males reduces the generalization towards women.

The self-reported medical history of cannabis use and psychiatric history is prone to recall bias, but clinical interviews and valid instruments reduce this risk. The research failed to define the ratio of THC/CBD and evaluate the current high strength use of the products. Neurobiological assessment and longitudinal designs of future research studies should be used.

Conclusion

This study shows that cannabis use disorder subjects a significant burden to psychiatry, where 71.3% of persons dependent on cannabis experience it due to predisposing personality factors. The personality phenotype of cannabis-dependent people in turn consists of emotional dysregulation (higher levels of anxiety and lower levels of emotional stability), behavioral disinhibition (poorer self-control, greater rule-consciousness, and perfectionism), greater sense of threat-detection (greater vigilance) and a higher level of psychological drive. The severity of psychiatric comorbidity on the dimensions of depression, anxiety, and psychotics in each case is independently predicted using these personality dimensions in dose-response relationships. The contribution of the personality-based assessment and specific intervention to the clinical process of CUD treatment provides a good opportunity to enhance the clinical features and decrease the burden of psychiatric patients in this sort of population. Future studies on personality-directed interventions, personality perspectives and personality-brain image combo will further rope over personality science into evidence-backed clinical care and transdiagnostic knowledge on cannabis-linked psychiatric pathophysiology.

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