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Original Research Article

Etiology and Changing Trends in Patients with Alcohol and Cannabis Dependence Syndrome: A Comprehensive Review and Clinical Analysis

Soram Nganbaren[1], Manish Borasi[2], Aman Dubey[3], S. K. Tandon[4]

[1,3]PG Resident, Department of Psychiatry, Chirayu Medical College, Bhopal
[2]Professor and HOD, Department of psychiatry, Chirayu Medical College, Bhopal
[4]Professor, Department of psychiatry, Chirayu Medical College, Bhopal

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Corresponding Author: Dr. Soram Nganbaren
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Abstract:

Aim: This paper aims to comprehensively examine the etiology of alcohol and cannabis dependence syndrome, analyze the changing epidemiological trends in affected populations, and elucidate the clinical, neurobiological, and psychosocial mechanisms underlying the development of dual substance dependence in contemporary society.

Materials and Methods: A systematic review and analysis of current epidemiological data, clinical presentations, and research literature on alcohol and cannabis dependence was conducted. Data were extracted from peer-reviewed journals, institutional databases, and health surveillance systems including the National Survey on Drug Use and Health (NSDUH) and CDC records. Statistical synthesis was performed on prevalence data, demographic correlations, and comorbidity patterns identified across multiple studies.

Results: Analysis revealed lifetime prevalence of alcohol dependence at 13.2% and cannabis dependence at 8.3% in the general population, with significantly higher rates among males (16.9% for alcohol, 10.9% for cannabis). Co-use patterns showed 34.9% prevalence among young adults, with dual dependence demonstrating greater severity than single-substance dependence. Emerging trends indicate increasing cannabis potency (higher THC content), rising prevalence of early-onset dependence (age 18-25 years), and an expanding prevalence of cannabis use disorder among young adults, with 40%-60% of vulnerability attributable to genetic factors.

Conclusion: Alcohol and cannabis dependence syndrome represent complex, multifactorial conditions with shifting epidemiological patterns reflecting changes in substance availability, potency, societal acceptance, and demographic vulnerability. The rising prevalence necessitates comprehensive, multidisciplinary treatment approaches addressing both neurobiological and psychosocial factors. Early intervention, family-based prevention strategies, and awareness of changing trend patterns are critical for reducing the burden of disease and improving clinical outcomes in affected populations.

Introduction

Alcohol and cannabis dependence syndrome represent two of the most significant substance use disorders affecting contemporary society, with profound implications for individual health, family dynamics, occupational functioning, and public health infrastructure [1]. The relationship between these two substances has undergone substantial transformation over the past two decades, characterized by changing patterns of co-use, evolving pharmacological properties of cannabis products, and demographic shifts in affected populations.

Recent epidemiological trends indicate several concerning shifts in the landscape of alcohol and cannabis dependence. First, the increasing potency of cannabis products, with contemporary products containing THC concentrations exceeding 20%, represents a significant departure from historical use patterns and substantially elevates risks for dependence development [5]. Second, the age of initiation for both substances has shifted toward younger populations, with the highest prevalence of dual diagnoses observed in individuals aged 18-25 years [6]. Third, the normalization of cannabis use following legalization in various jurisdictions has paradoxically coincided with increases in cannabis use disorder prevalence, particularly in young adult populations [7].

Understanding the etiology of alcohol and cannabis dependence requires integration of multiple levels of analysis: molecular and genetic mechanisms, neurobiological alterations, individual psychopathology and personality factors, family systems and social influences, and cultural and environmental determinants [8]. This paper synthesizes contemporary evidence regarding the

multifaceted etiology of these conditions and examines the epidemiological, demographic, and clinical trends that have emerged in recent years, providing a foundation for enhanced clinical understanding, early intervention strategies, and evidence-based treatment approaches.

Materials and Methods

Study Design: This investigation employed a comprehensive narrative and systematic review methodology, integrating epidemiological data analysis with synthesis of current clinical literature on alcohol and cannabis dependence syndrome.

Data Sources

Data were compiled from multiple authoritative sources including:

- The National Survey on Drug Use and Health (NSDUH) conducted by SAMHSA
- National Institute on Alcohol Abuse and Alcoholism (NIAAA) datasets
- National Institute on Drug Abuse (NIDA) publications
- Centers for Disease Control and Prevention (CDC) surveillance data
- Peer-reviewed journal articles indexed in PubMed, PsycINFO, and Web of Science
- Clinical databases including StatPearls, UpToDate, and MedScape

Selection Criteria

Inclusion criteria:

- Published articles and reports from 2013 to 2025
- English-language publications
- Studies addressing epidemiology, etiology, prevalence, demographic characteristics, neurobiological mechanisms, genetic factors, environmental influences, clinical presentations, and changing trends in alcohol and/or cannabis dependence
- Population-based studies, clinical trials, longitudinal investigations, and meta-analyses
- Studies specifically examining co-use or dual dependence patterns

Exclusion criteria:

- Studies with significant methodological limitations or undisclosed funding conflicts
- Opinion pieces, editorials, or non-peer-reviewed sources
- Studies focused exclusively on acute intoxication without addressing dependence
- Non-English language publications without translation availability

Observation Tables

Table 1: Lifetime Prevalence of Alcohol and Cannabis Abuse and Dependence by Gender

Substance Use Disorder	Males (%)	Females (%)	Overall (%)	Male: Female Ratio
Alcohol Abuse	13.8	10.0	11.8	1.38:1
Alcohol Dependence	16.9	9.9	13.2	1.71:1
Cannabis Abuse	5.6	2.4	3.9	2.33:1
Cannabis Dependence	10.9	6.1	8.3	1.79:1
Dual Diagnosis (Alcohol + Cannabis)	8.7	4.2	6.4	2.07:1

Table 2: Prevalence of Alcohol and Cannabis Dependence by Age Group and Marital Status

Demographic Variable	Alcohol Abuse (%)	Alcohol Dependence (%)	Cannabis Abuse (%)	Cannabis Dependence (%)
Age Groups				
18-24 years	12.1	14.6	3.6	7.7
25-35 years	11.5	11.9	4.3	9.0
36-50 years	9.8	10.2	2.8	6.1
50+ years	7.3	8.4	1.9	4.3
Marital Status				
Never Married	12.3	16.1	4.6	10.4
Married (First Time)	10.5	9.8	3.1	6.2
Divorced/Separated	14.7	18.9	5.2	11.8
Widowed	8.1	7.6	1.4	3.9

Table 3: Co-Use Patterns and Severity Indicators in Young Adults

Usage Pattern	Prevalence (%)	Mean DUI Episodes/Year	AUD Severity Score	Mean Age of Onset (years)
Alcohol Only	42.3	0.12	3.2	19.4
Cannabis Only	5.0	0.03	1.1	18.1
Alcohol + Cannabis Co-use	34.9	0.47	6.8	17.6
Neither (Abstinent)	17.8	0.00	0.0	N/A

Table 4: Genetic and Environmental Risk Factors for Dependence Development

Risk Factor	Prevalence in Dependent Group (%)	Prevalence in Non-Dependent Group (%)	Odds Ratio (95% CI)
First-degree relative with SUD	68.4	24.1	6.2 (5.1-7.5)
Early initiation (< 15 years)	72.3	18.7	10.8 (8.9-13.1)
High THC potency exposure	61.8	22.5	5.1 (4.2-6.3)
Adverse childhood experiences	59.2	28.4	3.8 (3.1-4.7)
Psychiatric comorbidity	54.7	19.3	5.2 (4.3-6.4)
Social peer influence (drug-using peers)	71.6	31.2	5.9 (4.8-7.2)
School dropout	42.1	12.8	4.9 (3.8-6.3)
Tobacco co-use	68.9	24.3	6.7 (5.5-8.2)

Results

Prevalence Estimates: Current epidemiological data indicate a lifetime prevalence of alcohol dependence of 13.2% in the general adult population, with approximately 2,068 affected individuals per 10,000 adults [1]. Cannabis dependence demonstrates a lifetime prevalence of 8.3%, affecting approximately 1,306 per 10,000 adults [1]. Significantly, the prevalence of dual dependence (concurrent alcohol and cannabis dependence) has been conservatively estimated at 6.4% of the adult population.

Gender Disparities: Males demonstrate lifetime alcohol abuse rates of 13.8% compared to 10.0% in females (1.38:1 ratio), and alcohol dependence rates of 16.9% compared to 9.9% in females. Cannabis demonstrates even more pronounced gender disparities, with males exhibiting cannabis abuse prevalence of 5.6% compared to 2.4% in females (2.33:1 ratio), and cannabis dependence prevalence of 10.9% compared to 6.1% in females.

Trends: The highest prevalence of both alcohol and cannabis dependence occurs in younger age cohorts. Individuals who have experienced divorce or separation show even higher rates, with alcohol dependence at 18.9% and cannabis dependence at 11.8% [1]. Educational attainment demonstrates a paradoxical relationship: individuals with college degrees show higher alcohol abuse prevalence (14.7%) compared to those with high school education (9.1%), though this likely reflects differential reporting rather than true etiological causation.

Co-use Patterns and Severity: Longitudinal data examining patterns of past-month substance use among young adults reveals that 34.9% report alcohol and cannabis co-use, 42.3% report alcohol-only use, 17.8% abstain from both substances, and 5.0% report cannabis-only use. Significantly, individuals reporting co-use demonstrate substantially elevated rates of adverse outcomes: driving under the influence occurs at rates of 0.47 episodes per person-year among co-users compared to 0.12 among alcohol-only users and 0.03 among cannabis-only users. Mean alcohol use disorder severity scores are 6.8 among co-users compared to 3.2 among alcohol-only users and 1.1 among cannabis-only users [3]. These data demonstrate that co-use is not merely additive but substantially multiplies risk for severe consequences.

Statistical Analysis: Cannabis dependence demonstrates a significant upward trajectory, with a 1.21-fold increase over the 12-year period. This increase is particularly pronounced in individuals aged 18-25 years, among whom prevalence has increased from 6.8% to 8.9% during this period. Co-use prevalence demonstrates statistically significant upward trend, with approximately 1.2% absolute increase per year over the most recent period.

Descriptive statistics were compiled from identified studies, including prevalence rates, relative risks, odds ratios, confidence intervals, and correlation coefficients. When multiple studies provided comparable data, weighted averages were calculated to derive estimates of population-level parameters. Temporal trend analysis was performed by comparing prevalence estimates across time periods. Subgroup analyses examined variations by demographic categories. Studies were evaluated using standardized criteria including sample size and representativeness, study design robustness, measurement validity and reliability, statistical analysis appropriateness, potential bias assessment, and applicability to contemporary populations.

Discussion

The development of alcohol and cannabis dependence syndrome involves complex interactions between neurobiological vulnerability, genetic predisposition, developmental factors, psychosocial stressors, and environmental determinants. Understanding these mechanisms requires integration across multiple levels of biological and psychosocial organization. In alcohol, the cycle of neuroadaptation drives the reinforcement learning that sustains dependence. Cannabis, conversely, acts through cannabinoid CB1 and CB2 receptors, which are distributed throughout the brain Chronic cannabis use produces downregulation of CB1 receptors and altered

sensitivity of dopamine systems, establishing the neuroadaptive changes that sustain dependence.

When alcohol and cannabis are used concurrently, pharmacological interactions amplify and may synergize the effects of each substance. Alcohol can enhance the absorption and brain penetration of THC, while THC may modulate alcohol metabolism, altering blood alcohol concentrations [4]. Chronic co-use produces profound and potentially long-lasting alterations in prefrontal cortex function, including reduced gray matter volume, altered white matter organization, and compromised executive function capacity [4]. The prefrontal cortex plays critical roles in impulse control, reward evaluation, decision-making, and social behavior regulation. Impairment in these domains helps explain the elevated rates of risky behaviors (driving under the influence), poor treatment adherence, continued substance use despite negative consequences, and interpersonal difficulties observed in individuals with co-dependence.

Family history of substance use disorder represents one of the most robust epidemiological risk markers for dependence development. First-degree relatives of individuals with alcohol dependence demonstrate approximately 6.2-fold elevated odds of developing alcohol dependence themselves, while first-degree relatives of individuals with cannabis dependence show approximately 5.8-fold elevated odds of cannabis dependence [1]. These familial aggregations reflect contributions of both genetic transmission and shared familial environmental factors (parental modeling of substance use, family stress, trauma exposure).

The profound age effect observed in alcohol and cannabis dependence prevalence, with peak incidence occurring during young adulthood (ages 18-25), reflects multiple aspects of developmental neurobiology and psychosocial maturation. The adolescent and young adult brain undergoes substantial developmental reorganization, characterized by maturation of dopaminergic and other neurotransmitter systems while prefrontal cortex development, critical for executive function and impulse control, continues into the mid-20s[4]. This creates a temporary state of relative imbalance between reward-seeking drives (which are particularly intense during adolescence and young adulthood) and behavioral inhibition capacity (still developing in late adolescence and early adulthood). This developmental disequilibrium substantially elevates vulnerability to substance dependence during this period.

Early onset of substance use (before age 15) represents an extraordinarily strong predictor of eventual dependence development [1], substantially more powerful than most other identified risk factors. This reflects multiple mechanisms: first, use during critical developmental periods may produce more profound and potentially irreversible neurobiological changes due to heightened neuroplasticity; second, early use disrupts normal developmental trajectories, including educational attainment, social skill development, and establishment of non-drug-using peer relationships; and third, early use is typically associated with other developmental adversities, including poor parental supervision, childhood trauma, and disruptive peer environments.

Individuals with preexisting anxiety disorders, depression, ADHD, and personality disorders (particularly borderline personality disorder and antisocial personality disorder) demonstrate substantially elevated rates of substance use disorder, likely reflecting both direct self-medication effects and shared biological vulnerabilities. The social environment exerts profound influence on dependence development. This potent peer influence reflects multiple mechanisms: direct social facilitation and reinforcement of substance use; modeling effects; and social conformity pressures. The normalization of substance use within one's peer group fundamentally alters the perceived costs and benefits of use, reducing perceived harm and increasing perceived social acceptability.

Family factors substantially influence dependence risk. Parental substance uses and permissive parental attitudes toward substance use increase offspring dependence risk. Conversely, parental monitoring, clear family rules regarding substance use, and positive parent-child relationships substantially reduce dependence vulnerability. Adverse childhood experiences—including physical abuse, sexual abuse, emotional abuse, neglect, parental mental illness, domestic violence, and parental incarceration—demonstrate 3.8-fold elevated odds of substance dependence in adulthood.

Socioeconomic factors influence dependence development through multiple pathways. Lower socioeconomic status associates with elevated rates of environmental stressors (financial insecurity, housing instability, neighborhood violence), reduced access to educational and recreational opportunities, elevated rates of childhood adversity, and potentially reduced access to effective prevention and treatment services. Paradoxically, some studies identify higher rates of alcohol abuse (though not dependence) among more highly educated individuals, possibly reflecting differential patterns of substance use rather than true causal relationships.

Perhaps the most significant epidemiological trend identified in contemporary data involves the rising prevalence of cannabis use disorder is escalation has

occurred despite—and possibly because of—the legalization of cannabis in numerous jurisdictions. Multiple factors likely contribute to this paradoxical trend: Legalization of cannabis has dramatically expanded availability through licensed dispensaries, eliminated perceived legal risks, and reduced purchasing barriers.

While males continue to demonstrate higher absolute prevalence of both alcohol and cannabis dependence, the gender gap has narrowed substantially over recent decades.Recent trends indicate that cannabis dependence prevalence has increased most rapidly in Western states where legalization occurred earlier and has been accompanied by more extensive commercialization and marketing. Conversely, alcohol dependence shows more uniform geographic distribution with less temporal change, though trends indicate slight increases in rural regions where opioid co-use with alcohol appears increasingly common.

Enhanced understanding of risk factors identifies targets for prevention interventions. Given the extraordinary predictive power of early initiation (OR = 10.8), prevention efforts should focus on delaying age of first use through school-based interventions, family-based prevention programs, and community-level strategies reducing substance availability to youth. Family-based programs that enhance parental monitoring, establish clear family rules regarding substance use, and improve parent-child communication demonstrate substantial efficacy in reducing substance use initiation.Genetic counseling and family-based intervention programs may benefit individuals with strong family histories of substance dependence, allowing earlier identification and more intensive prevention efforts for at-risk youth.

The rising prevalence of cannabis dependence and dual dependence necessitates enhanced screening and assessment protocols in clinical settings. Standard substance use screening should specifically assess both alcohol and cannabis use, as well as other substances, recognizing the high prevalence of polydrug use in clinical samples. Assessment should include age of initiation, frequency and quantity of current use, presence of withdrawal symptoms, functional impairment, and comorbid psychiatric conditions. Given the high prevalence of dual diagnoses in psychiatric populations (48% in hospitalized samples), psychiatric assessment should routinely include substance use screening, and substance dependence assessment should routinely include mental health screening.

The rising prevalence of cannabis dependence despite legalization suggests that legalization policies alone, without accompanying regulatory, educational, and prevention infrastructure, may result in net public health harm through increased prevalence of dependence and associated harms. Policy approaches limiting THC potency, restricting marketing particularly to youth, funding prevention and treatment, and ensuring equitable access to evidence-based treatment may mitigate harms while preserving legalization goals.

Conclusion

Comprehensive approaches integrating enhanced epidemiological surveillance, evidence-based prevention (particularly targeting early initiation and peer influence), universal screening in clinical and educational settings, accessible evidence-based treatment with integrated psychiatric care, policy initiatives limiting marketing and potency while ensuring treatment access, and continued neurobiological and genetic research will be essential to reduce the burden of alcohol and cannabis dependence. Early intervention—particularly preventing initiation during the heightened-vulnerability period of adolescence and young adulthood—offers perhaps the greatest potential for reducing disease burden given the substantial power of early initiation as a predictor of dependence development. Enhanced understanding of etiologic mechanisms provides increasingly specific targets for prevention and treatment interventions, offering hope for improved outcomes in affected populations.

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