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Executive Function and Externalizing Traits in Early and Late Onset Substance Use

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ABSTRACT

Introduction: Early substance use during this sensitive window likely disrupts maturation processes, resulting in persistent impairments in cognitive and behavioural regulation.
Aim: The present study aims to assess executive function and externalizing traits among individuals with early-onset and late-onset substance use.
Methods: The study is a cross-sectional study conducted at the inpatient psychiatry (de-addiction) ward of the Government Mental Health Hospital, Visakhapatnam, Andhra Pradesh, India, from October to December 2025. Sixty individuals aged 18 to 45 with ICD-11 diagnosed substance use disorder and abstinent for a minimum one week were recruited using purposive sampling. Executive function was measured using the Brown Executive Function/Attention Scale. Aggressive tendencies were measured using the Buss–Perry Aggression Questionnaire (BPAQ).
Results: Upon analysis of the composite T-score for all the individuals irrespective of the groups, 43 (71.7%) were found to have markedly atypical executive functioning, while 17 (28.3%) showed moderately atypical executive functioning, indicating that most individuals demonstrated significant impairment in executive functions. This indicates that substance use itself predispose to significant problems in executive function. The study also found that, higher executive dysfunction was associated with increased impulsivity and aggression in study groups.
Conclusions: Early identification and intervention targeting cognitive and behavioral dysregulation are critical to mitigating the enduring burden of adolescent substance dependence.
Key words: Substance use, Executive function, Externalizing traits, Buss–Perry Aggression Questionnaire, T-score.

INTRODUCTION

Adolescence is a critical neurodevelopmental period marked by substantial brain reorganization, particularly within the prefrontal cortex, which governs executive functions essential for decision-making, impulse control, and risk assessment [1,2]. It is also the phase during which most

individuals initiate substance use, with studies indicating that approximately half begin by late adolescence [3,4]. Early substance use during this sensitive window likely disrupts maturation processes, resulting in persistent impairments in cognitive and behavioural regulation [5].

Externalizing traits such as impulsivity, hyperactivity, aggression, and defiance frequently emerge during adolescence and confer heightened risk for substance experimentation and eventual dependence [6,7]. These traits may precede and potentiate substance use, establishing a bidirectional relationship wherein early use exacerbates behavioural dysregulation and vice versa [8]. Moreover, early substance use is linked with poorer clinical outcomes and greater cognitive impairments compared to later onset [4,9].

Given the public health significance of adolescent substance use and its neurobehavioral consequences, this study aims to comprehensively assess executive function and externalizing traits among individuals with early- and late-onset substance use. Understanding these cognitive-behavioral profiles informs targeted prevention and early intervention strategies. The present study aims to assess executive function and externalizing traits among individuals with early-onset and late-onset substance use.

METHODOLOGY

Study Design: This is a cross-sectional observational study.

Study Setting: The study was conducted at the inpatient psychiatry (de-addiction) ward of the Government Mental Health Hospital, Visakhapatnam, Andhra Pradesh, India, from October to December 2025.

Study participants: Sixty individuals aged 18 to 45 with ICD-11 diagnosed substance use disorder and abstinent for a minimum one week were recruited using purposive sampling. Exclusion criteria included prior major psychiatric diagnoses, neurological illnesses, or active withdrawal symptoms.

Assessment Scales: Executive function was measured using the Brown Executive Function/Attention Scale: a 57-item self-report questionnaire evaluating six domains/ clusters: activation, attention, effort, emotion regulation, working memory, and action [1]. Each domain/ cluster is scored separately and then a composite T-score is computed as per the instructions provided in the scale. T-scores of ≤54 indicate Typical executive function (unlikely significant problem), 55 – 59 indicate somewhat atypical executive function (possibly significant problem), 60 – 69 indicate moderately atypical executive function (significant problem) and ≥70 indicate markedly atypical executive function (very significant problem). Impulsivity was assessed with the Barratt Impulsiveness Scale (BIS-11), a validated 30-item measure capturing attentional, motor, and non-planning impulsivity [2]. Aggressive tendencies were measured using the Buss–Perry Aggression Questionnaire (BPAQ), a 29-item tool evaluating physical aggression, verbal aggression, anger, and hostility [3].

After informed consent, demographic and clinical data—including age of substance onset—were collected. Participants were stratified into Group A – early-onset users (<18 years) and Group B – late-onset users (≥18 years) groups. Psychometric scales were administered in a standardized setting.

Statistical analysis:

Data were presented as mean, standard deviation, frequency and percentage. Continuable variables were compared using the independent sample t-test, Spearman's rho Correlation and Mann Whitney test. Categorical variables were compared using the Pearson chi-square test. Significance was defined by P values less than 0.05 using a two-tailed test. Data analysis was performed using IBM-SPSS version 21.0 (IBM-SPSS Science Inc., Chicago, IL).

RESULTS

Demographics: Groups differed significantly in education levels (p=0.017) and occupational status (p=0.005), with group A (early-onset users) predominantly having primary education and are engaged in semi-skilled/ skilled occupations while none had professional occupation. Parental education and occupations showed low literacy and manual work common to both groups (Table 1).

Table 1: Analysis of Socio-demographic parameters

		Group				P value
		Group A		Group B		
		Count	Column N %	Count	Column N %	
Education	Illiterate	1	3.3%	0	0.0%	0.017
	Primary	14	46.7%	8	26.7%	
	Secondary	2	6.7%	12	40.0%	
	Tertiary	13	43.3%	10	33.3%	
Occupation	Manual	7	23.3%	8	26.7%	0.005
	Semi-skilled	13	43.3%	18	60.0%	
	Skilled	9	30.0%	0	0%	
	Professional	0	0%	4	13.3%	
Father's Education	Illiterate	12	40.0%	20	66.7%	0.038
	Primary	6	20.0%	4	13.3%	
	Secondary	2	6.7%	4	13.3%	
	Tertiary	10	33.3%	2	6.7%	

Executive Function: Group A (Early-onset users) scored significantly higher on the Brown Executive Function Scale (mean 77.73, SD 8.62) vs. late-onset (mean 73.20, SD 6.71; p=0.049), indicating greater executive dysfunction. Analysis of deficits in Subdomains showed statistical significance in activation (p=0.003), action (p=0.008). Despite the statistical difference was not significant, Group A also showed a trend towards greater memory impairment than group B (p=0.054). This reflects that group A (early-onset users) had significant difficulties in initiating tasks, regulating behaviour, attention and working memory (Table 2).

Table 2: Analysis of Brown Executive Function scale and sub-analysis of individual components

Brown Executive Function Scale	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
Total score	77.73	8.62	73.20	6.71	0.049
Activation	18.77	5.40	15.67	3.38	0.003
Focus	18.23	3.68	17.47	3.82	0.854
Effort	22.10	5.90	18.67	4.59	0.078
Emotion	22.87	5.64	21.60	5.44	0.938
Working memory	21.40	5.52	16.60	4.41	0.054
Action	21.40	5.52	20.13	3.42	0.008

Upon analysis of the composite T-score for all the individuals irrespective of the groups, 43 (71.7%) were found to have markedly atypical executive functioning, while 17 (28.3%) showed moderately atypical executive functioning, indicating that most individuals demonstrated significant impairment in executive functions. Interestingly, none of the participants showed somewhat atypical or typical executive function (Figure 1). This indicates that substance use itself predispose to significant problems in executive function.

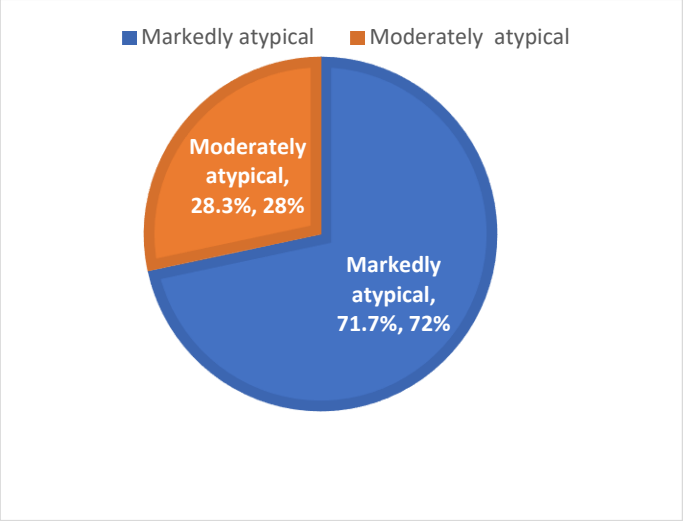

Figure 1: Brown executive function scale

We have further sub-analyzed if there is a statistical difference between both the groups with respect to the degree of executive dysfunction. In Group A, 23 (76.7%) participants demonstrated markedly atypical executive function, and 7 (23.3%) were moderately atypical. In Group B, 20 (66.7%) were markedly atypical, and 10 (33.3%) were moderately atypical. When comparing the subcategories (markedly vs. moderately atypical), the difference between Group A and Group B there was no statistically significant (p = 0.390) (Figure 2). However, when the overall Brown's Executive Function Scale scores were compared between the two groups without dividing into subcategories, the difference was statistically significant (p = 0.049). This indicates that while overall executive dysfunction is significantly higher in early-onset substance users (Group A), the distribution across the subcategories does not show a significant difference between the groups.

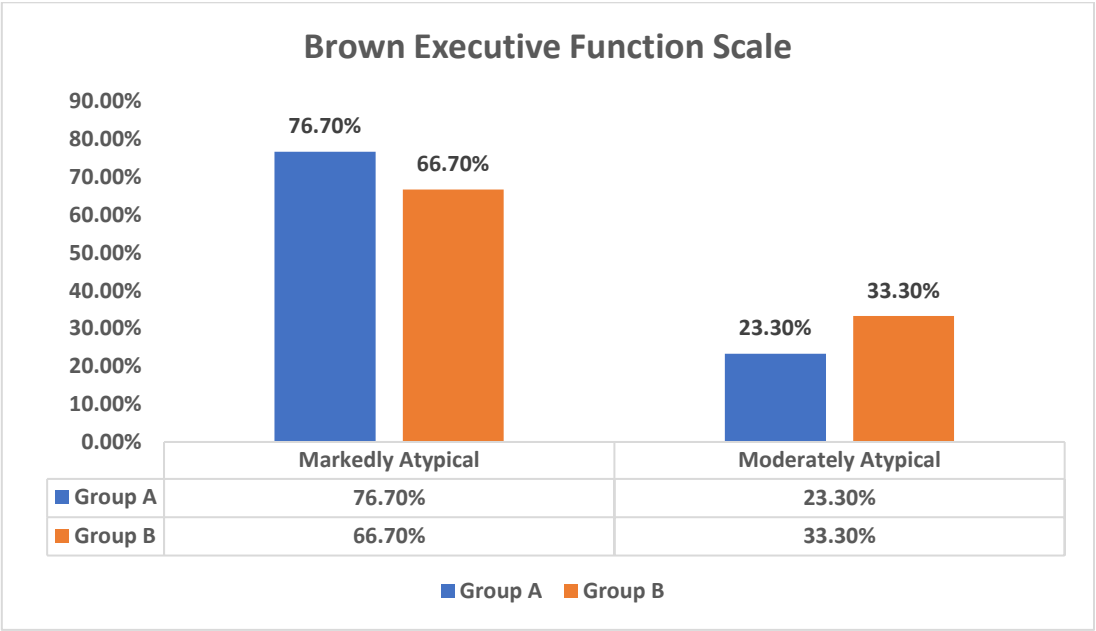

Figure 2: Degree of executive dysfunction

Impulsivity: BIS-11 total impulsivity scores were significantly higher in early-onset (mean 80.50, SD 11.46) compared to late-onset (mean 70.47, SD 11.73; p=0.001).

Upon sub-analysis of each component, attentional impulsivity scores were significantly higher in early-onset (21.7±4.32 Vs. 19.73±3.14, p=0.004) indicating greater impulsivity in group A, in particular attentional impulsivity (Table 3).

Table 3: Analysis of Barrats Impulsive Rating Scale and sub-analysis of individual components

Barrats Impulsive Rating Scale	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
Total score	80.50	11.46	70.47	11.73	0.001
Attentional Impulsiveness	21.70	4.32	19.73	3.14	0.004
Motor Impulsiveness	28.03	4.76	25.00	5.79	0.168
Non Planning Impulsiveness	30.77	4.70	25.73	5.38	0.183

Aggression: (Table 4) Group A scored higher on the Bus-Perry Aggression Scale (mean = 81.10, SD = 18.27) than group B (mean = 76.27, SD = 13.83; p = 0.366), indicating greater aggression, although the difference was not statistically significant. However, on subdomain analysis, group A has statistical significance with respect to physical aggression (p=0.012)

Table 4: Analysis of Buss-Perry Aggression Scale and Sub-analysis of individual components

Buss-Perry Aggression Scale	Group				P value
	Group A		Group B		
	Mean	Standard Deviation	Mean	Standard Deviation	
Total score	81.10	18.27	76.27	13.83	0.366
Physical Aggression	23.53	7.28	21.33	5.23	0.012
Verbal Aggression	16.37	3.42	13.87	3.19	0.709
Hostility	20.30	4.72	22.13	3.66	0.508
Anger	21.10	4.85	18.93	4.32	0.132

Correlations between impulsivity, aggression and executive dysfunction in both the groups: In Group A, the Brown Executive Function Scale showed a significant correlation with the Barratt Impulsiveness Scale ($\rho = 0.715$, $p < 0.0001$) and the Buss–Perry Aggression Scale ($\rho = 0.682$, $p < 0.0001$). Similarly, in Group B, significant positive correlations were observed with the Barratt Impulsiveness Scale ($\rho = 0.648$, $p < 0.0001$) and the Buss–Perry Aggression Scale ($\rho = 0.562$, $p = 0.001$), indicating that higher executive dysfunction was associated with increased impulsivity and aggression in both groups (Table 5, Figure 3, Figure 4, Figure 5, Figure 6).

Table 5: Correlations between impulsivity, aggression and executive dysfunction in both the groups

		Brown Executive Function Scale	
		Spearman's rho Correlation Coefficient	P value
Group A	Barrats Impulsive Rating Scale	0.715	< 0.0001
	Buss-Perry Aggression Scale	0.682	< 0.0001
Group B	Barrats Impulsive Rating Scale	0.648	< 0.0001
	Buss-Perry Aggression Scale	0.562	0.001

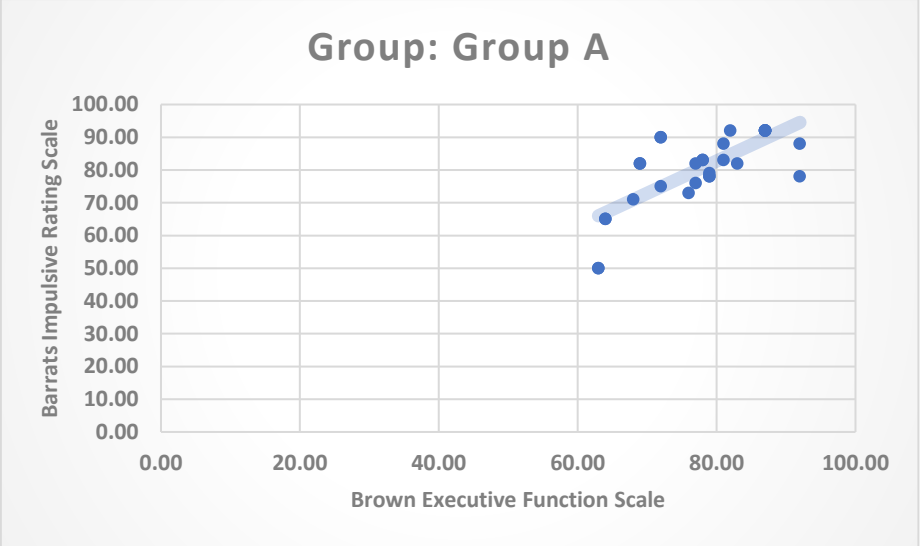

Figure 3: Correlations between impulsivity and executive dysfunction in group A

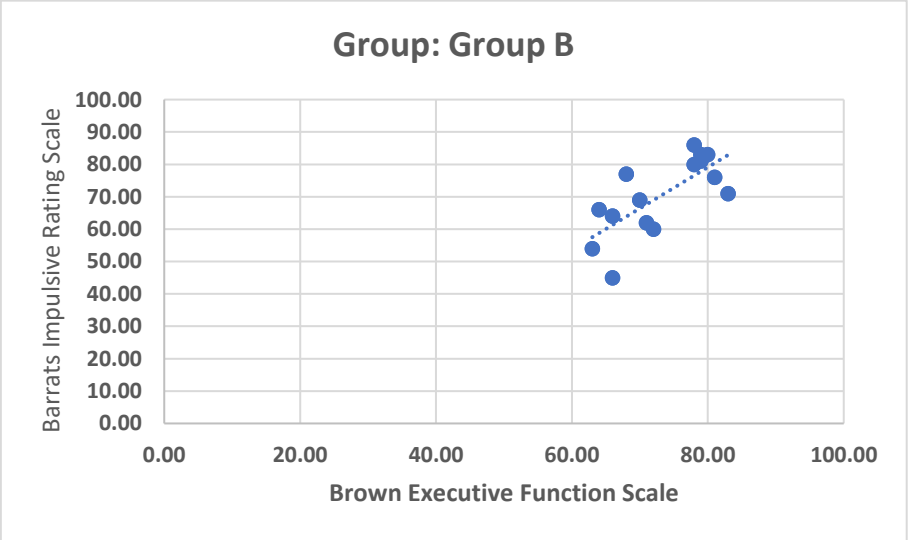

Figure 4: Correlations between impulsivity and executive dysfunction in group B

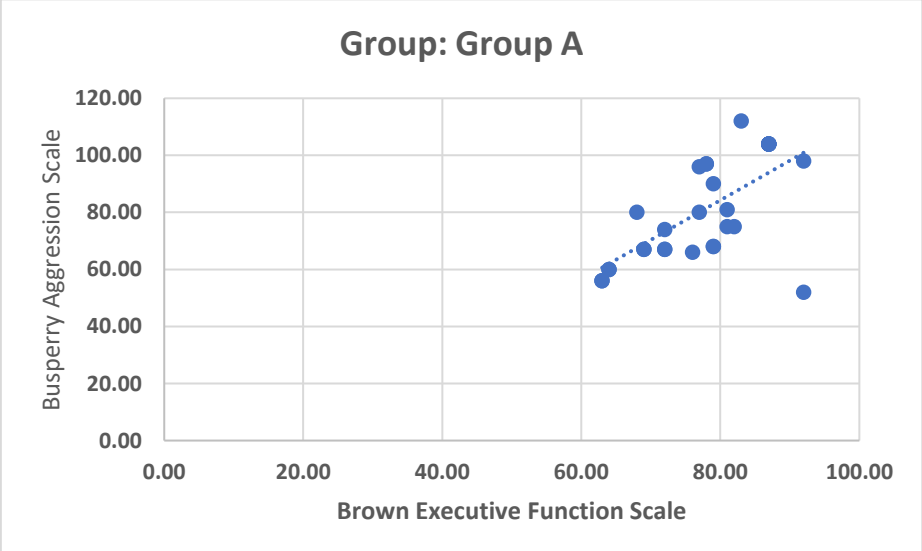

Figure 5: Correlations between aggression and executive dysfunction in group A

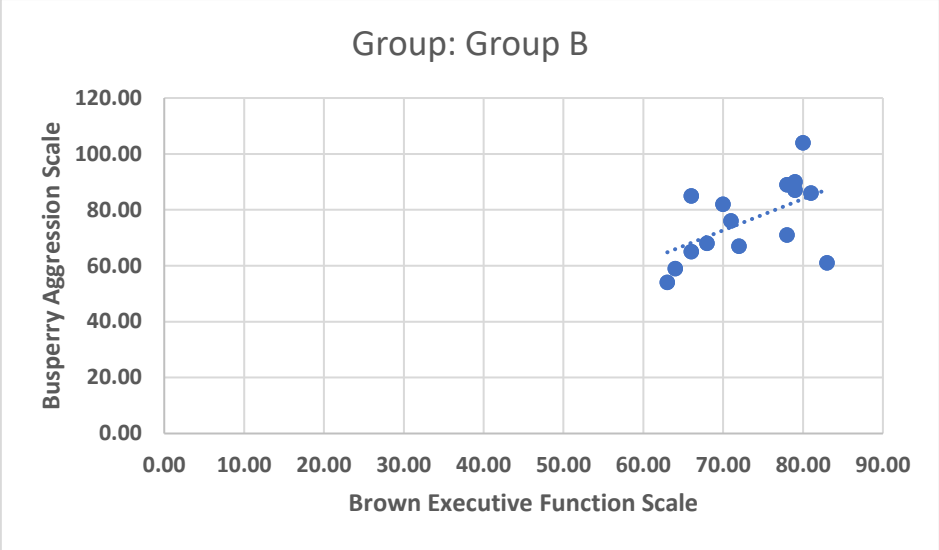

Figure 6; Correlations between aggression and executive dysfunction in group B

DISCUSSION

This study highlights that individuals initiating substance use in adolescence exhibit substantially greater executive function impairments and greater impulsivity and physical aggression compared to those with later onset. The Brown Executive Function/Attention Scale revealed pronounced deficits in initiation and behavioural regulation domains. These findings align with the neurodevelopmental research highlighting the heightened vulnerability of the adolescent prefrontal cortex to the neurotoxic effects of substances. [7,9,10,11] The prefrontal cortex, which governs executive functions such as planning, decision-making, inhibitory control, and goal-directed behaviour, continues to mature into the mid-20s. Early substance exposure during this critical period disrupts normative synaptic pruning and myelination processes, leading to persistent deficits in cognitive control and self-regulation. Disrupted hippocampal and prefrontal structures manifest cognitively as working memory and learning impairments—trends noted in this and previous studies [11-12].

Moreover, executive dysfunction in this population may create a vicious cycle: difficulties in goal-directed behavior and self-regulation exacerbate substance use severity, which further impairs neurocognitive function. Elevated scores on action and activation measures in Group A suggest challenges in initiating and sustaining purposeful behaviours, which are essential for adaptive functioning and recovery. Longitudinal evidence indicates that persistent substance use beginning in adolescence predicts broader and lasting neuropsychological decline into midlife. [13] Elevated attentional impulsiveness measured by BIS-11 corroborates the neurodevelopmental literature describing disruptions in prefrontal cortical maturation—especially in attentional regulation and inhibitory control pathways—that predispose youth to experimentation and maladaptive substance use trajectories [2,5,8,9]. Specifically, early substance use is associated with structural alterations including cortical thinning in prefrontal regions and impaired connectivity within frontoparietal networks critical for executive function. [11,14] These neurobiological changes underlie the higher impulsivity and executive dysfunction observed in adolescents who initiate substance use early. Impaired inhibitory control and increased attentional impulsiveness manifest behaviorally as difficulty resisting immediate rewards despite long-term negative consequences, thereby perpetuating risky behaviours and increasing vulnerability to substance dependence. [15,16]

The selective increase in physical aggression among early-onset users supports theories linking impaired executive control to disinhibited affect and violent behaviours [3,6,13,14]. The shared neurobiological substrates involving fronto-limbic circuits disrupted by early neurotoxic insult facilitate both behavioural dysregulation and increased risk behaviours that complicate clinical management. Sociodemographic factors provided partial context but did not fully explain early onset vulnerability, highlighting multifactorial influences including peer, genetic, and environmental stressors [17,18,19]. This highlights necessity for multi-level prevention approaches engaging families, schools, and communities.

Clinically, the findings underscore importance of incorporating screening for executive dysfunction and externalizing traits in adolescent substance use evaluations. Early, developmentally tailored interventions emphasizing cognitive-behavioural therapy to enhance self-regulation, decision-making, and social skills, alongside family engagement, show promise in altering trajectories and improving outcomes [20-21].

Limitations include modest sample size, cross-sectional design precluding causal inference, and reliance on self-report instruments subject to bias. Future longitudinal research integrating neuroimaging will clarify causal pathways and refine intervention targets.

CONCLUSION

Early-onset substance use is strongly associated with extensive executive dysfunction, increased impulsivity, and heightened physical aggression compared to later onset. While sociodemographic factors contribute, complex neurodevelopmental and environmental interactions underlie vulnerability. Early identification and intervention targeting cognitive and behavioral dysregulation are critical to mitigating the enduring burden of adolescent substance dependence.

Declaration by Authors

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Conflict of Interest: Nil

REFERENCES

1. Brown TE. Brown Attention-Deficit Disorder Scales. San Antonio: Psychological Corporation; 2001.
2. Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt impulsiveness scale. J Clin Psychol. 1995 Nov;51(6):768-74.
3. Buss AH, Perry M. The aggression questionnaire. J Pers Soc Psychol. 1992 Mar;63(3):452-9.
4. Das A, Kar SK, Dalal PK, Gupta PK. Externalizing psychopathology and cognitive functions in patients with early-and late-onset alcohol dependence. Indian J Psychiatry. 2021 May;63(3):233-9.
5. Lawrence AJ, Luty J, Bogdan NA, Sahakian BJ, Clark L. Impulsivity and response inhibition in alcohol dependence and problem gambling. Psychopharmacology (Berl). 2009 Nov;207(1):163-72.
6. Bácskai E, Czobor P, Gerevich J. Heavy drinking as a differential predictor of physical aggression in clinical and general populations. Prog Neuropsychopharmacol Biol Psychiatry. 2008 Apr 1;32(3):668-72.
7. Miech RA. Monitoring the future national survey results on drug use, 1975-2019. Institute for Social Research; 2020.
8. Tarter RE, Kirisci L, Mezzich A, Cornelius JR, Pajer K, Vanyukov M, Gardner W, Blackson T, Clark D. Neurobehavioral disinhibition in childhood predicts early age at onset of substance use disorder. Am J Psychiatry. 2003 Jun;160(6):1078-85. doi: 10.1176/appi.ajp.160.6.1078. PMID: 12777265.
9. Squeglia LM, Jacobus J, Tapert SF. The influence of substance uses on adolescent brain development. Clin EEG Neurosci. 2009 Jan;40(1):31-8.
10. Casey BJ, Jones RM, Hare TA. The adolescent brain. Ann N Y Acad Sci. 2008 Mar; 1124:111-26. doi: 10.1196/annals.1440.010. PMID: 18400927; PMCID: PMC2475802.
11. Jacobus J, Tapert SF. Neurotoxic effects of alcohol in adolescence. Annu Rev Clin Psychol. 2013; 9:703-21.

12. Steinfeld MR, Torregrossa MM. Consequences of adolescent drug use. Transl Psychiatry. 2023 Oct 6;13(1):313. doi: 10.1038/s41398-023-02590-4. PMID: 37802983; PMCID: PMC10558564.
13. Meier MH, Caspi A, Ambler A, Harrington H, Houts R, Keefe RS, et al. Persistent cannabis users show neuropsychological decline from childhood to midlife. Proc Natl Acad Sci U S A. 2012 Oct 16;109(40): E2657-64.
14. Whelan R, Watts R, Orr CA, Althoff RR, Artiges E, Banaschewski T, et al. Neuropsychosocial profiles of current and future adolescent alcohol misusers. Nature. 2014 Aug 14;512(7513):185-9. doi: 10.1038/nature13402.
15. Goldstein RZ, Volkow ND. Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. Nat Rev Neurosci. 2011; 12(11):652-69.
16. Verdejo-García A, Lawrence AJ, Clark L. Impulsivity as a vulnerability marker for substance-use disorders: review of findings from high-risk research, problem gamblers, and genetic studies. Neurosci Biobehav Rev. 2008;32(4):777-810.
17. Giedd JN. The teen brain: insights from neuroimaging. J Adolesc Health. 2008;42(4):335-43.
18. Luna B, Padmanabhan A, O'Hearn K. What has fMRI told us about the development of cognitive control through adolescence? Brain Cogn. 2010 Jul;72(1):101-13.
19. Blair RJ. The Neurobiology of Impulsive Aggression. J Child Adolesc Psychopharmacol. 2016 Feb;26(1):4-9. doi: 10.1089/cap.2015.0088. Epub 2015 Oct 14. PMID: 26465707; PMCID: PMC4779272.
20. Palmer RH, Young SE, Corley RP, Hopfer CJ, Stallings MC, Hewitt JK. Stability and change of genetic and environmental effects on the common liability to alcohol, tobacco, and cannabis DSM-IV dependence symptoms. Behav Genet. 2013 Sep;43(5):374-85. doi: 10.1007/s10519-013-9599-5. Epub 2013 Jun 13. PMID: 23760788; PMCID: PMC3794456.
21. Waldron HB, Turner CW. Evidence-based psychosocial treatments for adolescent substance abuse. J Clin Child Adolesc Psychol. 2008 Jan;37(1):238-61.

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