RESEARCH ARTICLE

Sleep architecture disturbances and their impact on daytime cognitive function in early-stage amphetamine-type stimulant abusers undergoing compulsory isolated detoxification: A retrospective study

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Amphetamine-type stimulant (ATS) use is frequently accompanied by persistent sleep disturbance during early abstinence. However, objective changes in sleep architecture and their relationship with daytime cognition remain insufficiently characterized in compulsory detoxification settings. This study aimed to characterize sleep architecture abnormalities in early-stage ATS users undergoing compulsory isolated detoxification and examine their associations with daytime cognitive function. In this retrospective cross-sectional study, 86 male participants with ATS use disorder and 78 age-matched healthy male controls underwent overnight polysomnography (PSG). Subjective sleep was assessed using the Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale (ESS), while cognition was evaluated with the Montreal Cognitive Assessment (MoCA), Stroop Color-Word Test, Trail Making Test (TMT), and Digit Symbol Substitution Test (DSST). Group differences were examined using appropriate between-group tests and effect sizes, and sleep-cognition relationships within the ATS group were evaluated using correlation analyses and multivariable linear regression. Compared with controls, ATS participants had longer sleep onset latency and wake after sleep onset, lower sleep efficiency and total sleep time, a higher proportion of N1 sleep, lower N3/slow-wave sleep and rapid eye movement (REM) sleep proportions, prolonged REM latency, and a higher arousal index (all $P < 0.001$). They also had lower MoCA and DSST scores and slower Stroop and TMT performance (all $P < 0.001$). Within the ATS group, N3/slow-wave sleep percentage correlated positively with MoCA (r = 0.412) and DSST (r = 0.367) and negatively with TMT-B completion time (r = -0.358). In adjusted models, N3/slow-wave sleep percentage remained independently associated with MoCA, TMT-B, and DSST outcomes. These findings indicated that early abstinence from ATS use was characterized by shallow, fragmented sleep with reduced restorative sleep and that these objective sleep abnormalities were associated with poorer daytime cognitive performance. Objective assessment of sleep architecture may therefore help identify clinically relevant targets for cognitive rehabilitation during compulsory detoxification.

Keywords: amphetamine-type stimulants; sleep architecture; polysomnography; cognitive function; compulsory detoxification; slow-wave sleep.

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Introduction

Amphetamine-type stimulants (ATS), particularly methamphetamine, remain among the most widely used non-opioid illicit drug classes worldwide. The World Drug Report 2023 estimated that approximately 36 million people used amphetamines during the preceding year

with East and Southeast Asia accounting for a substantial proportion of the global burden [1]. In China, methamphetamine has become the dominant registered drug of abuse, accounting for more than half of registered drug users by the end of 2022 [2]. For individuals with recurrent illicit drug use, compulsory isolation detoxification remains an important treatment and rehabilitation setting that combines physical recovery, psychological counseling, vocational training, and relapse-prevention education. This institutional environment removes immediate access to illicit drugs and provides relatively stable conditions in which persistent sleep and cognitive abnormalities can be evaluated [3].

Current evidence indicates that chronic methamphetamine exposure can adversely affect neural systems involved in arousal regulation and cognition. Postmortem human data demonstrate oxidative stress, altered neurotrophic signaling, and cellular injury in the dorsolateral prefrontal cortex of chronic methamphetamine users [4]. Experimental transcriptomic evidence further indicates that chronic methamphetamine exposure disrupts prefrontal cellular and metabolic pathways [5]. These frontostriatal, prefrontal, and limbic systems are central to executive control, memory, reward processing, and sleep-wake regulation, and cognitive dysfunction is a recognized feature of addictive disorders [6]. Clinical and pharmacological reviews of methamphetamine use additionally describe persistent neuropsychiatric, cognitive, and sleep-wake consequences that may continue beyond acute intoxication [7]. Sleep disturbance is one of the most common complaints during methamphetamine withdrawal and early abstinence. In individuals with cocaine or methamphetamine use disorders, poorer self-reported sleep has been associated with clinically relevant drug-use characteristics and greater daytime sleepiness [8]. Polysomnographic research in patients with methamphetamine dependence during remission has demonstrated abnormalities in sleep continuity and sleep architecture [9]. More broadly, disturbed sleep is prevalent across substance use disorders and may persist during recovery [10]. Longitudinal observations of methamphetamine withdrawal also indicate that sleep-related symptoms change substantially across different stages of abstinence [11]. Nevertheless, objective polysomnography (PSG) evidence remains limited, particularly in compulsory detoxification settings. It remains unclear whether early abstinence is characterized primarily by insomnia-like continuity disturbance, abnormal rapid eye movement (REM) regulation, loss of deep non-rapid eye movement (NREM) sleep, or a broader pattern of sleep fragmentation. The relationship between sleep architecture and cognition is especially relevant in this population. Slow-wave sleep supports memory consolidation and hippocampal-neocortical communication [12], while homeostatic sleep processes contribute to synaptic recovery and cortical plasticity [13]. REM sleep also contributes to emotional and procedural memory processing [14]. Disturbance of these restorative sleep processes could coexist with, and potentially intensify the functional consequences of, ATS-related neural injury. Sleep disturbance has also been proposed as a factor associated with relapse vulnerability in addictive disorders [15], and systematic evidence across substance-use populations supports the clinical importance of sleep assessment during recovery [16].

The specific PSG markers most closely associated with daytime cognitive performance in early-stage ATS abstinence have not been well defined. This study retrospectively analyzed clinical, PSG, subjective sleep, and neuropsychological data from men with ATS use disorder undergoing compulsory isolation detoxification to characterize objective sleep architecture abnormalities relative to healthy controls and determine whether sleep-stage distribution and sleep-continuity measures were associated with daytime cognitive performance. Full-night attended PSG, standardized sleep questionnaires, a multidomain cognitive battery, correlation analysis, and covariate-adjusted linear regression were used to evaluate these

relationships. By identifying objective sleep features associated with cognitive performance, this study would clarify clinically relevant sleep phenotypes that might inform sleep-focused assessment and cognitive rehabilitation during ATS recovery.

Materials and methods

Study design, setting, and participants

This retrospective cross-sectional study examined sleep architecture and daytime cognitive function in individuals with ATS use disorder undergoing compulsory isolation detoxification at the Guangzhou Compulsory Isolation Detoxification Center (Guangzhou, Guangdong, China). The center routinely performs PSG and neuropsychological evaluations during standardized intake and rehabilitation monitoring, providing a consistent clinical data source. Clinical records collected between January 2023 and December 2025 were retrospectively reviewed. All procedures of this study were approved by the Ethics Committee of the Criminal Investigation Police University of China (Shenyang, Liaoning, China). The ATS group consisted of 86 male participants aged 18 - 50 years who met the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria for ATS use disorder with methamphetamine as the primary abused substance (American Psychiatric Association, Washington, DC, USA). Eligible participants were those who had completed acute detoxification and entered the early rehabilitation phase, generally 30 - 90 days after admission, had negative urine toxicology results at the time of assessment, and completed overnight PSG and cognitive assessment within the same week. Exclusion criteria were primary dependence on opioids, benzodiazepines, or alcohol, moderate-to-severe obstructive sleep apnea defined as an apnea-hypopnea index >15 events/h, restless legs syndrome, narcolepsy, schizophrenia, bipolar disorder, major depressive disorder, current use of psychotropic medications, hypnotics, or other agents known to affect sleep architecture, traumatic brain injury, epilepsy, other neurological disease, or major uncontrolled medical illness. The control group consisted of 78 age-matched healthy male volunteers recruited from the local community during the same period. Controls had no history of illicit substance use, major psychiatric illness, primary sleep disorder, or major medical disease and had a Pittsburgh Sleep Quality Index (PSQI) global score ≤ 5. All controls completed the same PSG and cognitive protocols at the Sleep Medicine Center of the Affiliated Brain Hospital.

Polysomnographic assessment

Full-night attended PSG was performed using a Grael HD-PSG system (Compumedics Ltd., Abbotsford, Victoria, Australia). The PSG montage included six-channel electroencephalography (EEG), bilateral electrooculography (EOG), submental electromyography (EMG), electrocardiography (ECG), nasal-oral airflow, thoracoabdominal respiratory effort, pulse oximetry, bilateral anterior tibialis EMG, and body-position monitoring. Signals were digitized at a sampling frequency of 256 Hz, and electrode impedance was maintained below 5 kOhm before recording. Participants avoided caffeine and vigorous exercise on the study day and slept in a controlled laboratory environment from their habitual bedtime, approximately 22:00, until spontaneous awakening or 07:00. Recordings shorter than 7 h were excluded. Sleep stages including NREM stages N1, N2, and N3, REM sleep, and arousals were visually scored in 30-s epochs according to the American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events, Version 2.6 [17] (American Academy of Sleep Medicine, Darien, IL, USA) by two certified technologists. To assess inter-scorer reliability, 20% of PSG records were randomly selected for independent rescoring by the second technologist, yielding a Cohen kappa coefficient of 0.87, indicating excellent agreement. Extracted PSG variables were total sleep time (TST), total recording time (TRT), sleep onset latency (SOL), REM latency, wake after sleep onset (WASO), sleep efficiency (SE),

percentages of N1, N2, N3/slow-wave sleep (SWS), and REM sleep relative to TST, arousal index, and periodic limb movement index. These variables were selected to characterize sleep initiation, sleep continuity, sleep-stage distribution, REM timing, deep-sleep preservation, and sleep fragmentation.

Subjective sleep measures

Subjective sleep was assessed on the morning after PSG. Sleep quality over the previous month was measured with the validated Chinese version of the Pittsburgh Sleep Quality Index (PSQI) (https://www.sleep.pitt.edu/psqi) to evaluate sleep latency, duration, efficiency, disturbances, and daytime dysfunction. Global scores > 7 were interpreted as poor sleep quality according to Chinese validation data [18, 19]. Daytime sleepiness was measured with the Epworth Sleepiness Scale (ESS) (https://eprovide.mapi-trust.org/instruments/epworth-sleepiness-scale) with scores ≥ 11 being interpreted as excessive daytime sleepiness [20].

Cognitive function assessment

A standardized cognitive battery was administered between 09:00 and 11:00 by trained psychologists who were blinded to group assignment. The battery assessed global cognition, selective attention, inhibitory control, visuomotor speed, set-shifting, processing speed, and sustained attention, which were domains that were vulnerable to both ATS-related neural dysfunction and sleep disruption. Morning testing was used to reduce between-participant circadian variability. Global cognition was assessed with the Chinese version of the Montreal Cognitive Assessment (MoCA) (MoCA Test Inc., Greenfield Park, Quebec, Canada) [21]. Scores ranged from 0 to 30, and one point was added for participants with ≤ 12 years of education according to the standard scoring protocol. Scores < 26 were considered suggestive of mild cognitive impairment. Selective attention and inhibitory control were assessed using the Stroop Color-Word Test according to the standard administration procedure described by Scarpina and Tagini [22]. The principal outcome was completion time for the interference condition with longer times indicating weaker suppression of an automatic reading response. Visuomotor speed, visual scanning, and cognitive set-shifting were assessed with the Trail Making Test (TMT) according to the standardized administration and interpretation procedure described by Bowie and Harvey [23]. Part A primarily assessed psychomotor speed and attention, while Part B additionally required alternating numeric and alphabetic sequences and therefore imposed greater executive demand. Longer completion times indicated poorer performance. Processing speed and sustained attention were assessed with the Digit Symbol Substitution Test (DSST) [24]. Participants transcribed as many correct symbol-digit pairs as possible within 90 s; higher scores indicated better speeded attention and visuomotor coordination.

Statistical analysis

SPSS version 26.0 (IBM, Armonk, NY, USA) and R version 4.3.1 (https://www.r-project.org) were employed for the statistical analysis of this study. Continuous variables were summarized as mean ± standard deviation (SD) and assessed for distributional characteristics using Shapiro-Wilk tests and Q-Q plots. Between-group comparisons used independent-samples t-tests or Mann-Whitney U tests as appropriate, while categorical variables were compared using chi-square tests. Cohen d was reported for continuous between-group comparisons. Within the ATS group, associations between PSG and cognitive variables were evaluated using Pearson or Spearman correlations according to data distribution. Multiple linear regression models examined independent sleep predictors of cognitive outcomes after adjustment for age, education, duration of ATS use, and abstinence duration, which were selected a priori because of their potential relationships with cognition or recovery during abstinence. Regression results were reported using unstandardized coefficients (B), standard errors (SE), standardized beta coefficients, 95% confidence intervals (CI), *P* values, and adjusted R-squared values. All tests

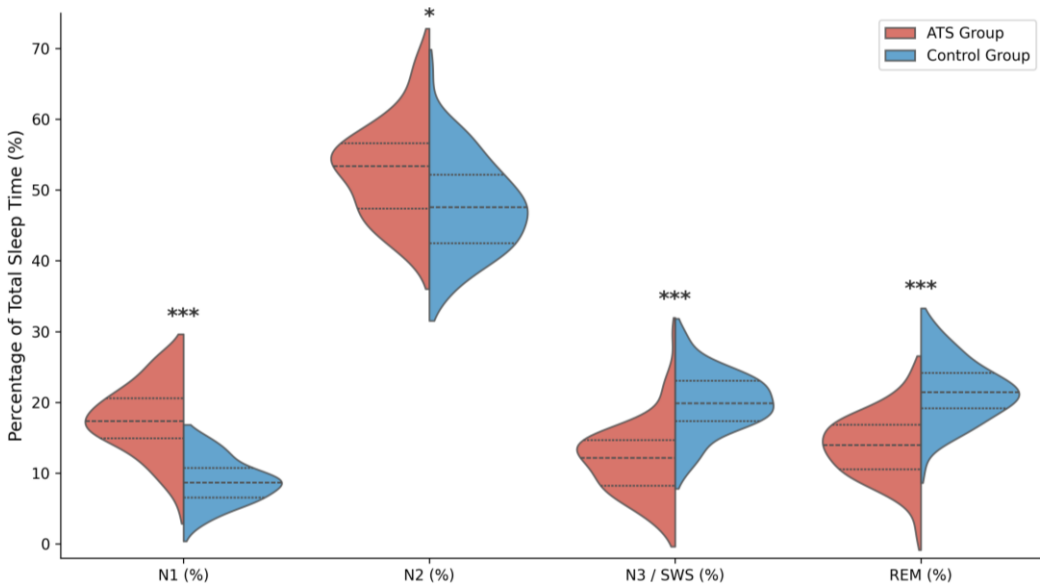


Figure 1. Split violin plot showing the distribution of sleep stage percentages in the ATS group (red) and control group (blue). Horizontal lines within violins indicated quartiles. *: $P < 0.05$. ***: $P < 0.001$.

were two-sided, and $P < 0.05$ was considered statistically significant.

Results

Demographic and clinical characteristics

The ATS group and the healthy control group demonstrated no statistically significant differences in age (33.7 ± 6.8 *vs*. 32.9 ± 7.1 years), body mass index (22.8 ± 3.1 *vs*. 23.4 ± 2.9 kg/m^2), or years of education (10.2 ± 2.6 *vs*. 10.9 ± 3.0 years). Cigarette smoking was significantly more prevalent among ATS participants than controls (82.6% *vs*. 43.6%) ($P < 0.001$). Among ATS participants, the mean duration of ATS use was 5.8 ± 3.4 years, the mean abstinence duration at assessment was 52.3 ± 18.6 days, and estimated cumulative methamphetamine exposure was 148.6 ± 89.2 g. Smoked administration was the predominant route of ATS use (76.7%) followed by nasal insufflation (14.0%) and intravenous injection (9.3%). ATS participants also reported poorer subjective sleep quality with higher PSQI scores (11.4 ± 3.8 *vs*. 3.6 ± 1.5) ($P < 0.001$) and greater daytime sleepiness reflected by higher ESS scores (12.8 ± 4.2 *vs*. 5.3 ± 2.4) ($P < 0.001$).

Polysomnographic sleep parameters

Compared with healthy controls, ATS participants exhibited marked impairment in sleep initiation and maintenance. Total sleep time was significantly shorter (348.2 ± 52.6 *vs*. 402.8 ± 32.4 min) (Cohen d = -1.25), whereas sleep onset latency (28.4 ± 14.6 *vs*. 14.2 ± 7.8 min) (d = 1.22) and wake after sleep onset (58.7 ± 22.3 *vs*. 28.4 ± 12.6 min) (d = 1.67) were significantly greater. Sleep efficiency was substantially reduced in the ATS group (72.4 ± 10.8% *vs*. 89.6 ± 4.3%) (d = -2.10) (all $P < 0.001$). Sleep-stage analysis demonstrated a shift toward lighter and more fragmented sleep (Figure 1). ATS participants had higher proportions of N1 sleep (18.2 ± 5.1% *vs*. 8.5 ± 3.2%) (d = 2.28) and N2 sleep (52.8 ± 7.3% *vs*. 48.3 ± 6.1%) (d = 0.67), accompanied by marked reductions in N3/SWS (11.4 ± 4.8% *vs*. 20.7 ± 4.5%) (d = -1.99) and REM sleep (13.6 ± 5.2% *vs*. 21.2 ± 4.0%) (d = -1.63). REM latency was prolonged (128.6 ± 38.4 *vs*. 86.3 ± 22.1 min) (d = 1.35), and the arousal index was elevated (22.4 ± 8.6 *vs*. 10.2 ± 3.8 events/h) (d = 1.83) (all $P < 0.001$). These findings indicated substantial disruption of sleep quantity, continuity, and restorative sleep-stage distribution.

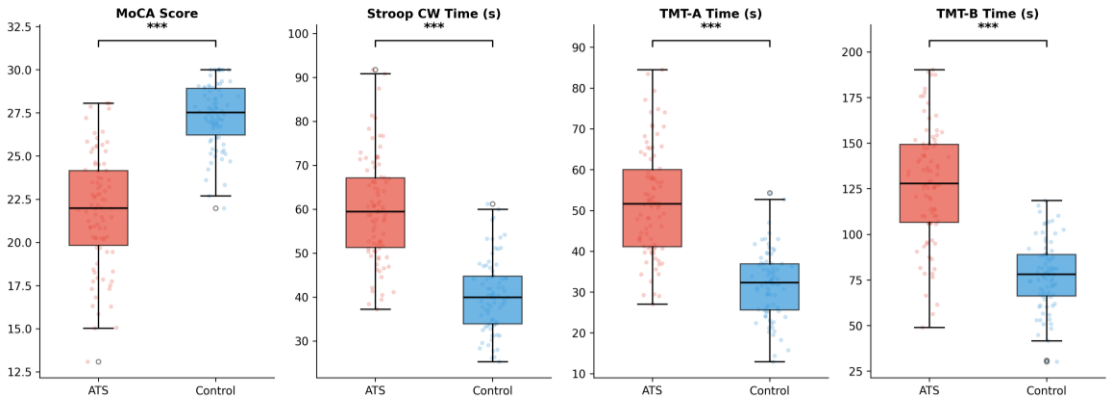


Figure 2. Box plots comparing cognitive function scores between the ATS group (red) and control group (blue). Individual data points were overlaid. ***: $P < 0.001$.

Cognitive function outcomes

ATS participants demonstrated significantly poorer global cognitive performance than controls. Mean MoCA score was lower in the ATS group (22.1 ± 3.4 *vs*. 27.2 ± 1.8) (Cohen d = -1.87) ($P < 0.001$), and 50 of 86 ATS participants (58.1%) scored below the MoCA threshold of 26 compared with 6 of 78 controls (7.7%) ($P < 0.001$). MoCA visuospatial/executive scores (3.2 ± 1.1 *vs*. 4.5 ± 0.6) (d = -1.47), attention scores (4.3 ± 1.3 *vs*. 5.6 ± 0.7) (d = -1.25), and delayed-recall scores (2.4 ± 1.4 *vs*. 4.1 ± 0.9) (d = -1.44) were also lower in ATS participants (all $P < 0.001$). Executive, attention, and processing speed measures showed similar large group differences (Figure 2). ATS participants required longer completion times on the Stroop interference condition (58.3 ± 12.7 *vs*. 39.5 ± 8.4 s) (d = 1.75), TMT-A (48.6 ± 14.2 *vs*. 32.1 ± 8.9 s) (d = 1.39), and TMT-B (128.5 ± 32.8 *vs*. 78.4 ± 18.6 s) (d = 1.88). DSST performance was lower in the ATS group (42.7 ± 9.8 *vs*. 58.3 ± 8.1 correct items) (d = -1.73) (all $P < 0.001$), indicating prominent impairment in inhibitory control, set-shifting, processing speed, and sustained attention.

Correlations between sleep parameters and cognitive function

Sleep-cognition correlations in the ATS group demonstrated that N3/SWS percentage showed the most consistent relationships with cognitive performance, correlating positively with MoCA (r = 0.412, $P < 0.001$) and DSST scores (r = 0.367, $P < 0.001$) and negatively with TMT-B completion time (r = -0.358, $P < 0.01$) and Stroop interference time (r = -0.324, $P < 0.01$). Sleep efficiency was positively associated with MoCA (r = 0.298, $P < 0.01$) and DSST (r = 0.341, $P < 0.01$) and negatively associated with TMT-B (r = -0.287, $P < 0.01$). REM percentage showed weaker positive associations with MoCA and DSST, whereas higher arousal index and greater WASO were associated with poorer executive and processing-speed performance (Table 1). SOL demonstrated comparatively limited associations, suggesting that sleep maintenance and sleep depth were more closely related to daytime cognitive performance than sleep initiation alone.

Multiple regression analysis

After adjustment for age, education, duration of ATS use, and abstinence duration, N3/SWS percentage remained independently associated with MoCA score (B = 0.25, SE = 0.07, standardized beta = 0.347, 95% CI 0.12 to 0.38, $P < 0.001$), while arousal index was inversely associated with MoCA score (B = -0.09, SE = 0.04, standardized beta = -0.218, 95% CI -0.16 to -0.02, $P = 0.014$). The adjusted R-squared for the MoCA model was 0.326. For TMT-B, higher N3/SWS percentage was associated with shorter completion time (B = -2.04, SE = 0.64, standardized beta = -0.298, 95% CI -3.32 to -0.76, $P = 0.002$), whereas greater WASO was associated with longer completion time (B = 0.33, SE = 0.14, standardized beta = 0.224, 95% CI 0.06

Table 1. Pearson correlation coefficients between PSG sleep parameters and cognitive function scores in the ATS group (n = 86).

Sleep Parameter	MoCA	Stroop CW	TMT-A	TMT-B	DSST
SE (%)	0.298**	-0.243*	-0.218*	-0.287**	0.341**
N1 (%)	-0.234*	0.212*	0.187	0.246*	-0.219*
N3/SWS (%)	0.412***	-0.324**	-0.268*	-0.358**	0.367***
REM (%)	0.278**	-0.198	-0.176	-0.246*	0.263*
Arousal index	-0.275*	0.289**	0.234*	0.312**	-0.297**
WASO (min)	-0.256*	0.267*	0.198	0.302**	-0.284**
SOL (min)	-0.187	0.156	0.142	0.198	-0.213*

Note: *: $P < 0.05$. **: $P < 0.01$. ***: $P < 0.001$. SE: sleep efficiency. SWS: slow-wave sleep. REM: rapid eye movement. WASO: wake after sleep onset. SOL: sleep onset latency.

to 0.60, $P = 0.018$). The adjusted R-squared was 0.284. For DSST, higher sleep efficiency (B = 0.29, SE = 0.08, standardized beta = 0.312, 95% CI 0.13 to 0.45, $P = 0.001$) and higher N3/SWS percentage (B = 0.54, SE = 0.20, standardized beta = 0.267, 95% CI 0.14 to 0.94, $P = 0.008$) were independently associated with better performance. The adjusted R-squared for the DSST model was 0.301. These models indicated that objective measures of sleep depth and continuity accounted for a meaningful proportion of cognitive variation beyond demographic and ATS-use covariates.

Discussion

This study identified substantial objective sleep architecture abnormalities and broad daytime cognitive deficits in men with ATS use disorder during early compulsory isolation detoxification. The predominant sleep phenotype comprised prolonged sleep initiation, reduced sleep efficiency and total sleep time, greater wakefulness after sleep onset, increased light N1 sleep, reduced N3/SWS and REM sleep, prolonged REM latency, and increased arousal burden. These abnormalities co-occurred with poorer global cognition, executive function, attention, and processing speed. Within the ATS group, deeper and more efficient sleep was consistently associated with better cognitive performance, and N3/SWS remained an independent correlate of MoCA, TMT-B, and DSST outcomes after adjustment for relevant demographic and drug-use characteristics. These findings extended the research on ATS-related sleep disturbance to the compulsory detoxification setting and identified objective sleep features that might be clinically relevant during cognitive rehabilitation. The observed sleep profile was consistent with prior clinical and experimental evidence, although the available research reflected different aspects of sleep disturbance. Mahoney *et al*. reported that individuals with cocaine or methamphetamine use disorders commonly exhibited poor self-reported sleep and greater daytime sleepiness, linking subjective sleep problems to clinically relevant drug-use characteristics [8]. Rezaei-Ardani *et al*. provided polysomnographic evidence of altered sleep architecture during remission from methamphetamine dependence [9]. Conroy and Arnedt described persistent sleep disturbance as a common feature across substance use disorders rather than a problem unique to methamphetamine [10]. McGregor *et al*. demonstrated that withdrawal symptoms including sleep-related symptoms evolved across the time course of methamphetamine abstinence [11]. In the present study, the large increase in N1 sleep together with marked reductions in N3/SWS and REM sleep suggested a shift toward lighter and less restorative sleep rather than an isolated complaint of insomnia. Experimental work in rhesus monkeys has

likewise shown methamphetamine-related reductions in N3 and REM sleep and subsequent rebound effects, supporting a biological basis for disruption of restorative sleep stages [25]. Increased arousal burden may additionally reflect persistent dysregulation of neural circuitry governing wakefulness and sleep-state stability [26].

Several biological mechanisms may contribute to persistent sleep disturbance after ATS cessation. Human postmortem evidence indicated that chronic methamphetamine exposure was associated with oxidative stress, altered neurotrophic signaling, and cellular injury in the prefrontal cortex [4]. Single-nucleus transcriptomic findings further demonstrated methamphetamine-related disruption of cellular, myelin, mitochondrial, and metabolic pathways in the prefrontal cortex [5]. Dopaminergic signaling had a well-established role in regulating wakefulness and transitions between sleep and waking states [27]. Serotonergic pathways also contributed to sleep-wake regulation and REM-related processes [28]. Beyond monoamine signaling, methamphetamine could dysregulate central and peripheral immune responses [29], while epigenetic mechanisms might contribute to persistent neuroadaptation during addiction and abstinence [30]. The persistence of marked PSG abnormalities after a mean abstinence duration of approximately 52 days therefore suggested that disturbed sleep during compulsory detoxification was not limited to acute withdrawal. The cognitive findings also accorded with established evidence on methamphetamine-related neurocognitive dysfunction. Verdejo-Garcia *et al*. emphasized that addiction was accompanied by disruption of executive, attentional, learning, and decision-making processes that were central to treatment engagement and behavioral control [6]. Zhong *et al*. specifically reported broad cognitive impairment in methamphetamine-dependent patients relative to healthy controls with evidence of improvement in some domains during abstinence [31]. Scott *et al*. found, in a critical review and meta-analysis, that methamphetamine use was associated with deficits across episodic memory, executive function, information-processing speed, motor skills, language, and visuoconstruction [32]. More recent works in abstinent methamphetamine users identified selective abnormalities in response inhibition and related behavioral control [33]. Independently of substance use, short-term sleep deprivation impaired attention and processing speed across cognitive domains [34]. The large differences in Stroop interference, TMT-B, MoCA, and DSST performance in the present sample were therefore consistent with the combined presence of ATS-related neurocognitive vulnerability and substantial sleep disruption. The strongest sleep-cognition relationship involved N3/SWS. Slow-wave sleep supported memory consolidation through coordinated hippocampal-neocortical communication [12]. The synaptic homeostasis framework further proposed that sleep contributed to neural renormalization and restoration after waking plasticity [13]. A broader synthesis of sleep and memory research also identified slow-wave physiology as central to the reorganization and stabilization of newly acquired information [35]. In the present adjusted models, higher N3/SWS percentage was independently associated with better global cognition, faster executive set-shifting, and better processing speed after adjustment for age, education, ATS-use duration, and abstinence duration. In healthy adults, greater slow-wave activity had been prospectively associated with better working-memory performance [36]. In older community-dwelling adults, the relationship between SWS and cognition had also been shown to vary with cognitive reserve [37]. Together, these findings supported N3/SWS as an objective marker that might help identify ATS patients with greater daytime cognitive vulnerability, while the cross-sectional design precluded conclusions about directionality. Sleep continuity also appeared functionally important. Experimental and clinical evidence indicated that sleep fragmentation could impair daytime alertness and cognitive efficiency [38]. In the present sample, lower sleep efficiency, greater

WASO, and a higher arousal index were associated with poorer DSST and TMT-B performance, and sleep efficiency and WASO remained independent correlates in adjusted models. Brower and Perron proposed sleep disturbance as a potentially generalizable factor associated with relapse risk across addictive disorders [15]. Angarita *et al*. reviewed clinically important sleep abnormalities across several substance-use populations and highlighted the need to address sleep during recovery [16]. These observations supported routine sleep assessment in ATS rehabilitation. Interventions that improve sleep continuity or increase restorative sleep may have value as adjuncts to cognitive rehabilitation, but prospective interventional studies are required before any causal or therapeutic benefit can be established.

Several limitations should be considered in this research. The retrospective cross-sectional design did not permit causal inference regarding whether sleep disruption contributed to cognitive impairment, cognitive or neural dysfunction contributed to sleep disturbance, or both reflected shared ATS-related mechanisms. Further, all participants were male, which limited generalizability to women, who might differ in patterns and responses related to methamphetamine use [39]. Furthermore, residual confounding from premorbid cognitive ability, childhood adversity, personality traits, cigarette smoking, and detailed polydrug exposure could not be fully excluded. Single-night PSG might not capture night-to-night variability, although the standardized institutional environment might have reduced some sources of variability. In addition, longitudinal follow-up was unavailable, preventing assessment of within-person recovery of sleep and cognition or evaluation of whether baseline sleep architecture predicted later functional outcomes. Prior longitudinal evidence suggested that neurocognitive function could improve with sustained methamphetamine abstinence [40], underscoring the need for prospective studies integrating repeated PSG and cognitive assessment.

Conclusion

Men with ATS use disorder undergoing early compulsory isolation detoxification showed marked objective disruption of sleep architecture, characterized by impaired sleep initiation and continuity, increased light sleep, reduced N3/SWS and REM sleep, prolonged REM latency, and increased arousal burden. These sleep abnormalities co-occurred with deficits in global cognition, attention, executive function, and processing speed. N3/SWS percentage was the most consistent independent sleep correlate of cognitive performance, while measures of sleep continuity also contributed to adjusted cognitive models. These findings supported objective sleep architecture as a clinically relevant dimension of ATS recovery and identified restorative sleep depth and continuity as potential targets for future longitudinal and interventional research. Prospective studies are needed to determine whether improvement in sleep architecture precedes or accompanies cognitive recovery and whether sleep-focused interventions can strengthen rehabilitation outcomes during sustained abstinence.

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