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Neural dysfunction underlying divergent thinking and its shared mechanisms with protracted withdrawal symptoms in heroin use disorder

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Abstract

Background. Drug use may induce transient bursts of inspiration but is also associated with increasing cognitive rigidity. How neurological damage induced by drug addiction affects higher-order thinking processes in humans remains elusive. This gap limits our understanding of broader cognitive impairment in addiction and prolonged maladaptation during withdrawal.
Methods. The study investigated divergent thinking, a key component of flexible behavior, in heroin use disorder (HUD). Behavioral performance was assessed using the Realistic Presented Problem-Solving Task. Task-related neural signatures were recorded via functional near-infrared spectroscopy (fNIRS) in three groups of individuals: (1) HUD short withdrawal; (2) HUD long withdrawal; and (3) healthy controls (HC).
Results. Both HUD groups exhibited poorer divergent thinking performance than the HC group. HUD participants showed hyperactivation in the prefrontal cortex (PFC) and temporoparietal junction (TPJ), accompanied by reduced functional connectivity within the PFC and between the PFC and TPJ. Network analysis further revealed reduced topological properties of PFC nodes and increased topological properties of TPJ nodes, indicating disrupted PFC-mediated top-down regulatory control. PFC subregional activation and functional connectivity with the bilateral TPJ served as shared neural features that predicted divergent thinking impairment and protracted withdrawal symptoms.
Conclusions. These findings reveal a sustained compensatory pathological mechanism and an imbalance in neural resource allocation during complex cognition in HUD individuals. The identified signatures of cortical activity by fNIRS offer reliable biomarkers for the rapid detection of both higher-order cognitive deficits and withdrawal symptoms, and further offer precise cortical targets for their integrated intervention.

Introduction

In cultural narratives, drugs are often portrayed as catalysts for inspiration and imagination, yet also regarded as poisons that erode genius. Drug-induced neurotoxic alterations promote compulsive drug-seeking behavior, which may impair divergent thinking and lead to cognitive rigidity (Cole et al., 2020; Gangal et al., 2023). Such cognitive limitations may undermine adaptive responses to environmental demands, constituting a critical factor underlying persistently high relapse rates (Carrión et al., 2026). Previous studies have primarily focused on the effects of drug addiction on general cognitive functions (Ceceli et al., 2023; Domínguez-Salas, Díaz-Batanero, Lozano-Rojas, & Verdejo-García, 2016; Verdejo-Garcia et al., 2015). However, how long-term drug use and withdrawal affect higher-order thinking processes, such as divergent thinking, and their association with withdrawal symptoms remains elusive. This gap limits a deeper understanding of adjustment difficulties during abstinence and hinders the acceptance and effectiveness of withdrawal treatment (Goldstein, 2022; Vidrine et al., 2013).

As a core component of higher-order thinking and flexible behavior, divergent thinking enables individuals to generate novel and appropriate solutions through cognitive shifting when habitual strategies fail to resolve a problem (Ovando-Tellez et al., 2022). The prefrontal cortex (PFC) and temporoparietal junction (TPJ) are the key neural substrates for divergent thinking. Previous neuroimaging studies have consistently revealed structural abnormalities and functional dysregulation in the PFC and TPJ among individuals with drug addiction (Herlinger &

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Lingford-Hughes, 2022; Liu et al., 2009; Sun et al., 2017; Tolomeo & Yu, 2022; Wollman et al., 2017). The PFC regulates top-down cognitive control to maintain goal-directed behavior, while the TPJ updates problem context representation and regulates spontaneous reflection processes (Beaty et al., 2021; Benedek et al., 2014; Chen et al., 2014, 2018; Huang, Tang, Sun, & Luo, 2018; Ovando-Tellez et al., 2022). During divergent thinking, the TPJ is involved in idea generation, while the PFC supports idea evaluation and integration (Dosenbach et al., 2007). Therefore, functional abnormalities in the TPJ and PFC could disrupt key cognitive processes underlying divergent thinking. Lower efficiency in neural information transmission and higher cognitive demands may induce compensatory mechanisms, manifested by increased activation to mitigate neural functional damage (Huhn et al., 2019; Jansen et al., 2015; Mayer et al., 2013; McConnell et al., 2020).

More efficient regional coupling is essential for sustaining goal-directed behavior and adaptive control (Menon & D'Esposito, 2022). Individuals with high divergent thinking typically exhibit strong connectivity between PFC and TPJ (Beaty et al., 2021; Chen et al., 2018). The PFC serves as a core hub of the executive control network (ECN), whereas portions of the TPJ constitute key nodes of the default mode network (DMN) (Davey, Pujol, & Harrison, 2016). This PFC–TPJ coupling may therefore reflect functional interactions between executive control and internally oriented processing systems that support divergent thinking. Disruptions in ECN–DMN connectivity have been observed in individuals with substance use disorders (Ceceli, Bradberry, & Goldstein, 2022; Zhang & Volkow, 2019). During withdrawal, chronic opioid users with pain may reflect a shift in neural resources from the ECN to the DMN, potentially leading to impaired flexible goal-directed control (McConnell et al., 2020). Reduced top-down control may facilitate unconstrained thinking processes, such as mind-wandering and spontaneous associations, which support the generation of divergent ideas (Christoff et al., 2016). However, excessive decoupling of executive control could disrupt the goal-directed evaluation, integration, and refinement of ideas, ultimately impairing divergent thinking performance (Fu et al., 2026; Menon & D'Esposito, 2022).

Impairment in divergent thinking reduces the ability to generate flexible and adaptive responses during abstinence, thereby leading to cognitive rigidity (Simon & Bock, 2016; Zabelina & Ganis, 2018). Such rigidity often increases a repetitive focus on negative internal states and limits effective coping strategies, which may exacerbate protracted withdrawal symptoms after the acute physiological phase, including persistent physical discomfort and negative emotional states (Demirbas, Ilhan, & Dogan, 2012; Gangal et al., 2023; Watkins & Roberts, 2020). However, the relationship between impaired divergent thinking and withdrawal symptoms remains to be clarified. Moreover, the shared neural signatures of these processes need to be identified, which could facilitate the diagnosis of high-risk populations and provide new intervention targets for the concurrent improvement of withdrawal outcomes and higher-order cognitive function.

In conclusion, the present study aims to examine divergent thinking deficits in heroin use disorder (HUD), characterize their underlying neural correlates, and explore how these impairments co-occur with protracted withdrawal symptoms. In the study, we mainly focused on the PFC and TPJ and detected their cortical neural responses using functional near-infrared spectroscopy (fNIRS). fNIRS has been widely used in monitoring abnormal neural indicators of mental disorders, demonstrating its efficacy in assessing cortical activity and functional connectivity (Duan, Van Dam, Ai, & Xu, 2020; Liu et al., 2019). Due to its portability and cost-effectiveness, fNIRS has demonstrated significant potential and advantages in the rapid diagnosis and classification of brain diseases (Albrecht, Kvist, & Franzén, 2025; Butters et al., 2023). A detailed investigation into fNIRS neural signatures can provide key biomarkers for the rapid diagnosis of withdrawal states and cognitive impairments, and offer direct cortical targets for non-invasive brain stimulation interventions. Using sparse regression-based feature identification, key neural features jointly associated with impairments in divergent thinking and the severity of withdrawal symptoms were precisely extracted. Additionally, although previous studies have demonstrated that some of the structural brain abnormalities related to addiction are restored as withdrawal time increases, cognitive impairments such as decision-making remain after 1.5 years of abstinence in drug users (Biernacki et al., 2016; Zhang et al., 2019). To further investigate the lasting impact of heroin dependence on divergent thinking, this study also examines differences among HUD patients at varying abstinence periods. We hypothesized that HUD individuals have impaired divergent thinking, which is associated with hyperactivation and impaired network connectivity. Such behavioral impairment and abnormal cortical activity patterns could reliably predict the severity of protracted withdrawal symptoms.

Methods

Participants

The present study recruited 70 heroin users aged 35 to 55 years from multiple drug rehabilitation centers who were under isolation treatment. 35 healthy control participants were recruited from the local schools and communities, who were matched to the HUD group in terms of age, gender, educational attainment, smoking, and drinking habits. Selection criteria for participants with HUD included: (1) meeting the diagnostic criteria for severe substance use disorder outlined in the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5); (2) consuming an average of 0.1 grams or more of heroin per day, with a positive result of a urine test before entering the rehabilitation center; (3) having a history of drug use for 10 years or more; (4) absence of other substance use disorders, such as methamphetamine, cannabis, and so forth. All participants had no symptoms of mental illness or a family history of mental illness, no intake of hormonal contraceptives, anesthetics, or caffeine-containing foods in the three days before the experiment, and no recent surgery. At the rehabilitation center, all HUD participants underwent standardized rehabilitation procedures, including daily physical exercise and supportive therapy to prevent relapse.

These participants were in a continuous abstinence period of 6–18 months (Francis, 2020; Laudet & White, 2008). Evidence from animal experiments and neuroimaging studies suggests that approximately 12 months after abstinence represents a critical stage during which neural plasticity, cognitive function, and emotion regulation show significant recovery (Martinelli et al., 2020; Xu et al., 2015). To further examine whether divergent thinking shows significant improvement with increasing duration of abstinence, the HUD participants were further divided into two subgroups based on drug withdrawal duration: one group with a drug abstinence period of 6–12 months (HUD1), and another with a period of 12–18 months (HUD2). Eight participants were excluded from the study due to compromised data resulting from sampling interruptions and poor signal quality. The final sample consisted of 34 participants in the HUD1 group (age: M = 49.23 years, SD = 6.63), 30 participants in the HUD2 group (age: M = 46.33 years, SD = 9.94), and 33 participants in the HC group (age: M = 46.39 years, SD = 7.06). Detailed demographic and addiction characteristics are presented in Supplementary Table S1.

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Task and procedure

The Realistic Presented Problem-solving Task (RPP) was employed to assess divergent thinking (Cheng, Zhang, Xie, & Hao, 2026; *Duan et al., 2022*; Xue, Lu, & Hao, 2018; Yuan, Lu, Yang, & Hao, 2021). Participants were required to verbally generate as many solutions as possible for realistic social problems commonly encountered in daily life. The RPP task was adapted from the Alternative Uses Task (AUT) paradigm by replacing abstract object-based problems with realistic social scenarios. Naturalistic contexts with greater ecological validity minimize potential behavioral differences attributable to general cognitive abilities between the HUD and HC groups. Open-ended problems without predefined correct answers can effectively assess divergent thinking, reflecting the processes of flexible idea generation and adaptive problem-solving.

In this study, three socially relevant problem scenarios from the Means-Ends Problem-solving Task (MESP) were selected as test items (Madore & Schacter, 2014). All scenarios were randomly presented from a third-person perspective. For example, one scenario described a hypothetical individual who had recently moved to a new neighborhood and did not know anyone there. Participants were instructed to generate possible ways to help him/her establish new social connections. Each item was presented for 2 minutes, followed by a 20-second fixation interval ("+"). fNIRS data were recorded throughout task completion.

Protracted withdrawal symptoms scale

Protracted withdrawal syndrome was assessed by the Rating Scale for Protracted Withdrawal Symptoms of heroin addiction (Jie et al., 2009). It contains 19 items in four dimensions: physical symptoms, emotional symptoms, craving symptoms, and insomnia symptoms. The items were scored on a 5-point scale. Higher scores indicated more severe protracted withdrawal symptoms.

fNIRS data acquisition

Near-infrared signals of three wavelengths of 780 nm, 805 nm, and 830 nm were recorded by the NirSmatII-200pro system (HuiChuang, China) with a sampling rate of 11 Hz. In all, 24 emitters and 24 detectors were spaced three centimeters apart to form 68 recording channels (Figure 1a,b). Referencing the international 10–20 system, the placement of the optodes covering the PFC was referred to as the Fpz electrode, while the placement of the optodes covering the bilateral parietal and temporal cortex was referred to as the P6 electrode and P5 electrode (Zhao et al., 2022). The spatial positions of all optodes and channels were recorded by a 3D magnetic space digitizer (FASTRAK; Polhemus, USA). The Brodmann partition, based on MNI coordinates, was further converted using the NIRS-SPM MATLAB package (Supplementary Table S8). Seven subregions were identified, including the dorsolateral prefrontal cortex (dlPFC, BA9, BA46), frontopolar area (FPC, BA10), inferior prefrontal gyrus (IFG, BA47), orbitofrontal area (OFC, BA11), angular gyrus (AG, BA39), supramarginal gyrus (SMG, BA40), superior parietal lobule (SPL, BA7), and superior temporal gyrus (STG, BA22) (for a detailed description, see Supplementary Table S7). The AG and SMG overlap with the TPJ (Igelström & Graziano, 2017).

Behavioral data analysis

The overall analysis workflow is illustrated in Figure 1c. Following the standardized scoring method of the classic AUT for assessing divergent thinking (Wang et al., 2022, 2023), responses generated in the RPP task were evaluated across three key dimensions: fluency, flexibility, and originality. Additionally, appropriateness and persistence were assessed to further gauge participants' abilities in idea evaluation and sustained exploration. The fluency score was the total number of answers given by a participant, with 1 point for each reasonable answer. The flexibility score was the number of categories in a participant's answers, with one point for each category. The originality score was determined by the frequency of a reported answer in the overall answer pool, which was composed of the answers from all participants. Two points were given when the percentage was less than 1%. One point was given when the percentage was between 1% and 5%. No points were given when the percentage exceeded 5%. The appropriateness scores were rated on a 5-point scale, with 5 being "strongly appropriate" and 1 being "strongly inappropriate." The appropriateness scores of each answer were averaged to get the final appropriateness score of one participant. Two professionals in the field of psychology assessed each participant's responses. The average scores of the two raters were used as the overall results for each of the three dimensions. The persistence scores were ultimately calculated by dividing flexibility scores by fluency scores (Nijstad, De Dreu, Rietzschel, & Baas, 2010; Wang, Lu, Runco, & Hao, 2018). It was confirmed that the Internal Consistency Coefficient (ICC) between the two raters was reliable (fluency ICC = 0.994, flexibility ICC = 0.964, originality ICC = 0.919, appropriateness ICC = 0.809). The final score for each dimension is the total of the three items.

fNIRS data analysis

The NirSpark v1.8.1 (HuiChuang, China) was used to preprocess the fNIRS data. Motion artifacts were removed using a spline interpolation method by calculating the moving standard deviation. A band-pass filter with a frequency of 0.01 Hz–0.1 Hz was then applied to diminish both noise and interference signals, such as low-frequency drift, heartbeats, and breathing (Zhang et al., 2022). The concentration changes of HbO, HbR, and HbT were converted from the raw density signals by the modified Beer–Lambert Law. The subsequent analysis utilized the HbO signal as the primary indicator, given its superior signal-to-noise ratio and greater sensitivity to cerebral blood flow changes (Yennu, Tian, Gatchel, & Liu, 2016). The fNIRS data were then smoothed with the hemodynamic response function (HRF) to correct the temporal autocorrelation (Friston et al., 2000). A block range was set to 0–120 s with a pre-baseline range of -20s. Based on the general linear model (GLM), the beta values for the 68 channels were analyzed to reflect the level of neural activation. The beta values of all channels within each ROI were averaged.

The task-related functional connectivity (FC) strengths between channels were calculated using Pearson correlation coefficients. To achieve a stable FC strength, the total duration of three blocks excluding the fixation was used for analysis, yielding a 68 × 68 correlation matrix representing FC strength over the time series (Aarabi & Huppert, 2019). Fisher's r-to-z transformation was applied to normalize coherence values into a Gaussian distribution variable. Based on the channel-based localization of brain regions, we averaged all channel-to-channel pairs within the regions of interest (ROIs) to determine both inter-ROI and intra-ROI connectivity.

Gretna toolbox was used to conduct the main network analysis based on binary brain networks (Wang et al., 2015). Since negative correlations also reflect signal synchronization between cortical regions, the absolute value of functional connectivity was incorporated into the calculation. To minimize bias from using a single

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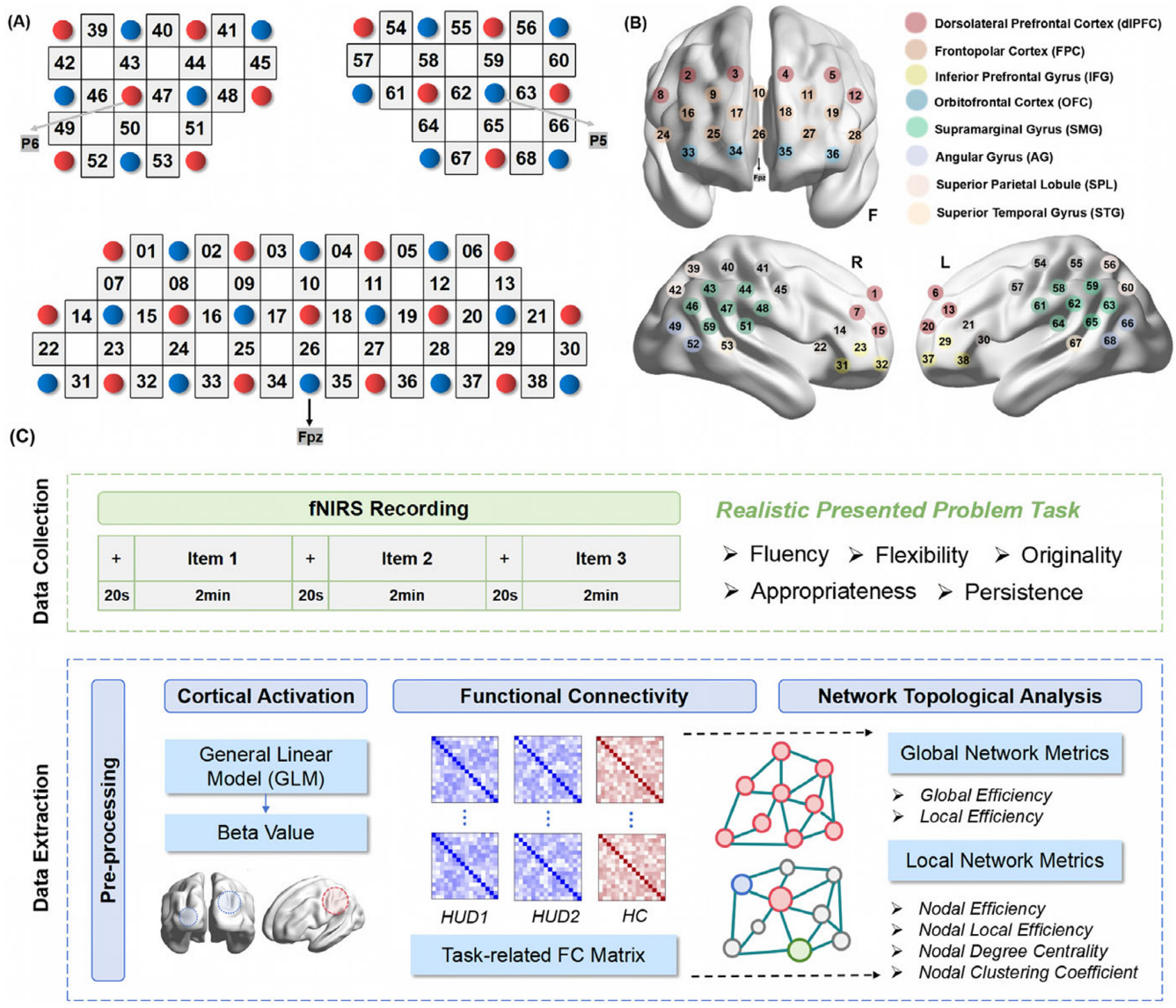


Figure 1. Diagram of the fNIRS optode probes and analysis workflow. (a) 68 fNIRS channels composed of 24 emitters and 24 detectors. The center point of the bottom row of optode probes was located at Fpz. The center point of the bilateral parietal and temporal cortex was located separately in P6 and P5. (b) Regions of interest. The configuration of the regions of interest was located according to the MNI coordinates. (c) Overall analysis workflow.

threshold, a sparsity of 10% - 50% (Step size = 1%) was applied to extract the brain networks' topological features (Wang et al., 2022; Zhang et al., 2022). To assess overall brain network connectivity, the area under the curve (AUC) of each network metric was calculated across different sparsity levels. In this study, global network and regional node metrics were compared between the addiction and control groups (Latora & Marchiori, 2001). Global network metrics included global efficiency (E_{glob}) and local efficiency (E_{loc}), while nodal network metrics encompassed nodal efficiency, nodal local efficiency, nodal degree centrality, and nodal clustering coefficient (detailed in Method S2).

Statistical analysis

Statistical significance testing

An F-test with Group (HUD1, HUD2, HC) as the between-participants factor was conducted using SPSS 25.0 for the between-group statistical comparison. *F*-test results for the fNIRS data were corrected using the False Discovery Rate (FDR) method, and post hoc tests were adjusted using the Bonferroni method to control for multiple comparison errors.

Sparse regression-based features identification

To further examine the shared neural features between divergent thinking deficits and protracted withdrawal symptoms, sparse regression-based feature identification was applied (Zhang et al., 2025). In Sparse Regression Prediction, let $Y \in R^{n \times q}$ be the matrix of fNIRS neural signatures with n subjects and q features, and z be the diagnosis status of protracted withdrawal symptoms; the regression model:

$$\min_{v} \frac{1}{2}\|\mathbf{z} - \mathbf{Y}\mathbf{v}\|_2^2 + \lambda_v \Omega(\mathbf{v}),$$

which could reveal diagnosis-related features. If $\Omega(v)$ is the L1-norm, this model is the LASSO. LASSO promotes sparsity by shrinking the coefficients of less informative features toward zero,

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thereby selecting a compact subset of predictive biomarkers and potentially improving model generalization. Owing to the sparsity-inducing nature of the subgradient-optimized LASSO, the resulting coefficient vector contains a small number of large-magnitude weights and many near-zero values. Features associated with high-magnitude coefficients are interpreted as predictive features, forming the final selected feature set for downstream analysis.

Results

Lower behavioral performance in the HUD groups relative to the HC group

The results showed a significant main effect of Group in fluency [F (2, 94) = 62.28, p < 0.001, η_p^2 = 0.570], flexibility [F (2, 94) = 41.96, p < 0.001, η_p^2 = 0.472], originality [F (2, 94) = 38.16, p < 0.001,η_p^2 = 0.448] and persistence [F (2, 94) = 35.50, p < 0.001, η_p^2 = 0.430] (Figure 2a–c, Supplementary Table S2). Bonferroni-adjusted pairwise comparison tests indicated that RPP scores in the two HUD groups were lower than those in the HC group on the dimensions of fluency (HUD1: p < 0.001; HUD2: p < 0.001), flexibility (HUD1: p < 0.001; HUD2: p < 0.001), originality (HUD1: p < 0.001; HUD2: p < 0.001) and persistence (HUD1: p < 0.001; HUD2: p < 0.001). For appropriateness scores, no significant difference was found between the HUD and HC groups [F (2, 94) = 0.12, p = 0.886, η_p^2 = 0.003]. There were also no significant differences in RPP performance between the HUD1 and HUD2 groups.

Higher cortical activation in the HUD groups relative to the HC group

The main effect of Group was significant on the OFC [F (2, 94) = 8.78, p_{FDR} < 0.001, η_p^2 = 0.158], the FPC [F (2, 94) = 4.35, p_{FDR} =

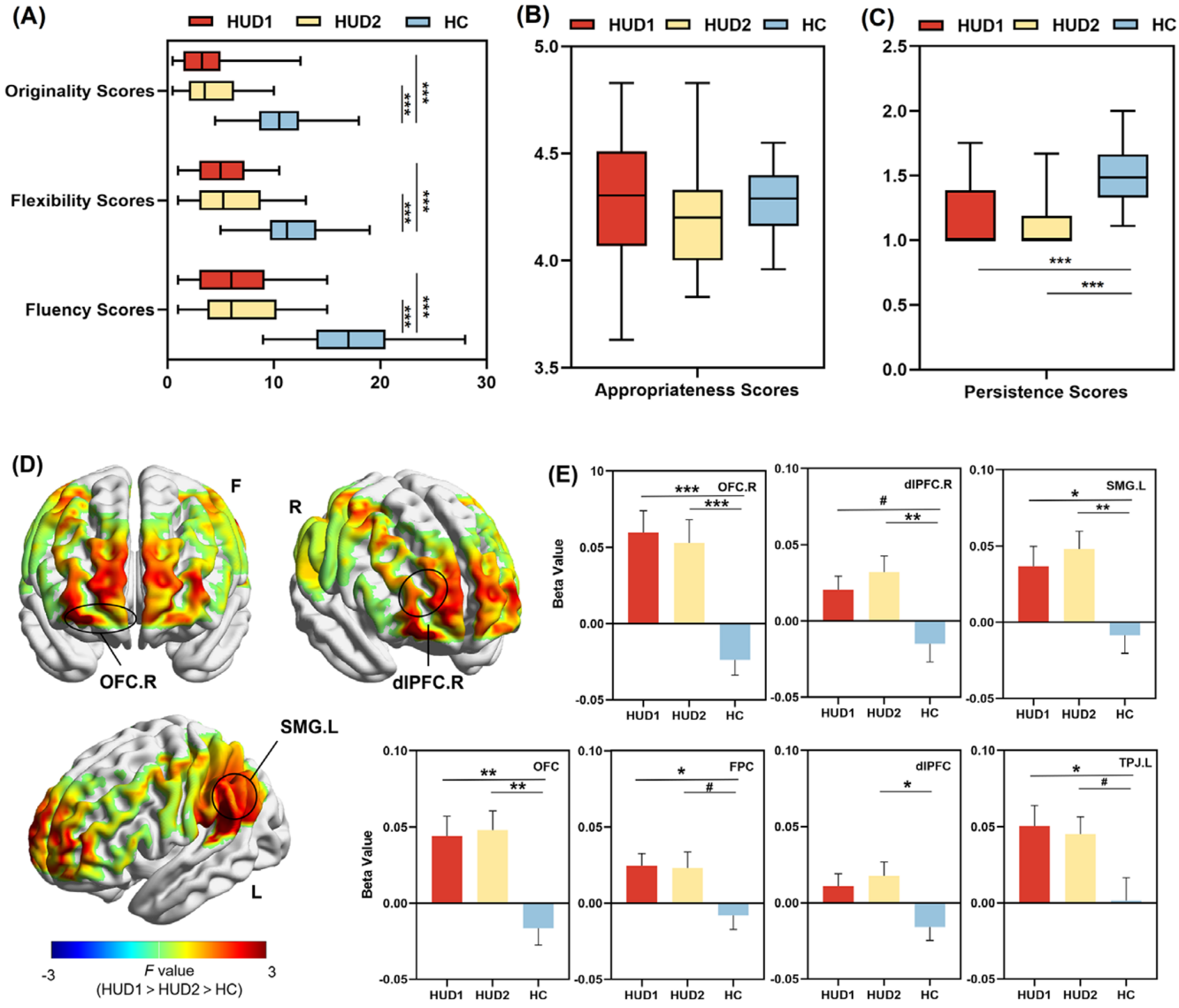


Figure 2. Differences in task performance and cortical activation among three groups. (a) Scores on originality, flexibility, and fluency dimensions among three groups. (b) Scores on appropriateness dimensions among three groups. (c) Scores on the persistence dimension among three groups. (d) The *F*-map illustrates the differences in *F*-values across brain regions among the three groups. Significant subcortical regions delineated through FDR correction are highlighted. (e) Beta values across significant ROIs after FDR correction among the three groups. Error bars represent standard errors of the mean. *Note:* L, 'left sagittal plane'; R, 'right sagittal plane'; F, 'coronal plane'. HUD1: 'heroin use disorder group with short duration of abstinence'; HUD2: 'heroin use disorder group with long duration of abstinence'; HC: 'healthy control group'. $^{\#}p$ < 0.10, $^{*}p$ < 0.05, $^{**}p$ < 0.01, $^{***}p$ < 0.001.

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0.029, $\eta_p^2 = 0.157$], the dlPFC [F (2, 94) = 4.11, $p_{FDR} = 0.029$, $\eta_p^2 = 0.080$], and the left TPJ [F (2, 94) = 4.18, $p_{FDR} = 0.029$, $\eta_p^2 = 0.082$] (Figure 3, Supplementary Table S3). Subsequently, the Bonferroni-adjusted pairwise comparison tests showed that the two HUD groups exhibited greater activation in the OFC (HUD1: $p = 0.001$; HUD2: $p = 0.002$) and dlPFC (HUD1: $p = 0.076$; HUD2: $p = 0.029$) compared to the HC group. Activation on the FPC (HUD1: $p = 0.015$; HUD2: $p = 0.154$) and left TPJ (HUD1: $p = 0.021$; HUD2: $p = 0.110$) was greater in the HUD1 group than in the HC group.

The left and right hemispheres of the ROI were further distinguished to localize regions of variance more accurately. The main effect of Group was significant on the right OFC [F (2, 94) = 12.64, $p_{FDR} < 0.001$, $\eta_p^2 = 0.212$], the right dlPFC [F (2, 94) = 5.31, $p_{FDR} = 0.033$, $\eta_p^2 = 0.102$], and the left SMG [F (2, 94) = 5.82, $p_{FDR} = 0.028$, $\eta_p^2 = 0.110$]. Subsequent Bonferroni-adjusted pairwise comparison tests revealed that beta values of the right OFC (HUD1: $p < 0.001$; HUD2: $p = 0.001$), right dlPFC (HUD1: $p = 0.046$; HUD2: $p = 0.009$), and left SMG (HUD1: $p = 0.014$; HUD2: $p = 0.011$) in the HUD groups were significantly higher than those in the HC group. The differences in cortical activation in these regions between the HUD1 and HUD2 groups were not significant.

Alterations in functional connectivity in HUD

As seen in Figure 3a–c, the results of functional connectivity revealed weaker intra-ROI connections within the left dlPFC, left

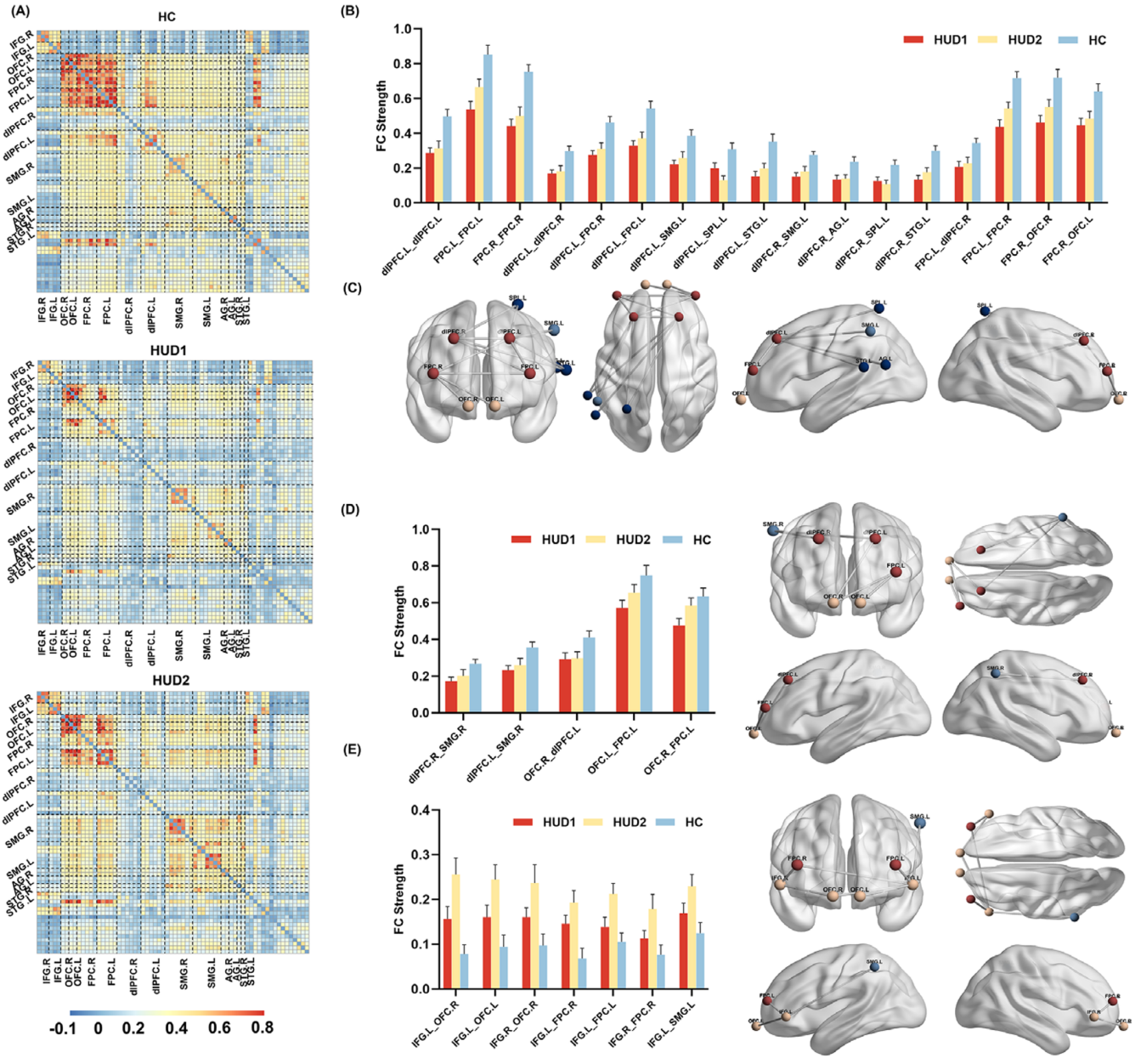


Figure 3. The difference in the functional connectivity among the three groups. (a) Group-averaged connectivity matrix map for HUD1, HUD2, and HC groups. The Pearson correlation coefficient *z-value* was presented in the correlation matrix maps corresponding to channel pairs from each ROI. (b) Group-averaged functional connectivity strengths of significant ROI pairs after *F*-tests with FDR correction. (c) The visualization of spatial information through ROI connectivity. Error bars represent standard errors of the mean. *Note:* HUD1: 'heroin use disorder group with short duration of abstinence'; HUD2: 'heroin use disorder group with long duration of abstinence'; HC: 'healthy control group'.

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FPC, and right FPC in the HUD groups compared to the HC group (ANOVA tests, all *ps* < 0.05, FDR corrected) (Supplementary Table S4). Compared to the HC group, both HUD groups exhibited significantly reduced short-range FC strengths between ROIs within the PFC, including dlPFC.L_dlPFC.R, dlPFC.L_FPC.R, dlPFC.L_FPC.L, FPC.L_dlPFC.R, FPC.L_FPC.R, FPC.R_OFC.R, and FPC.R_OFC.L (ANOVA, all *ps* < 0.05, FDR corrected). Regarding the long-range FC strengths between the PFC and bilateral temporoparietal regions, both HUD groups demonstrated weaker FC strengths compared to the HC group, specifically between the bilateral dlPFC and temporoparietal regions, including SMG, SPL, and STG (ANOVA tests, all *ps* < 0.05, FDR corrected).

Additionally, the two HUD groups also exhibited partially differentiated FC patterns (Supplementary Tables S5–S6). The HUD1 group showed weaker FC strengths on ROI pairs between the bilateral dlPFC and right temporoparietal region (*t-tests*, all *ps* < 0.05), as well as ROI pairs between the OFC and other subregions of the PFC (*t-tests*, all *ps* < 0.02) (Figure 3d). The HUD2 group showed stronger FC strengths related to the IFG, particularly the connections of the IFG with the OFC, FPC, and the left temporoparietal region (*t-tests*, all *ps* < 0.05) (Figure 3e).

Alterations in the global and nodal network metrics in HUD

For global network properties, there was no significant difference in E_{glob} or E_{loc} between HUD and HC groups (Figure 4a). However, as shown in Figure 4b,c, both HUD groups exhibited significantly reduced nodal efficiency (HUD1: $p = 0.040$; HUD2: $p < 0.001$), nodal local efficiency (HUD1: $p < 0.001$; HUD2: $p < 0.001$), nodal degree centrality (HUD1: $p < 0.001$; HUD2: $p < 0.001$), and nodal clustering coefficient (HUD1: $p < 0.001$; HUD2: $p < 0.001$) in the left dlPFC compared to the HC group (ANOVA tests, all *ps* < 0.05, FDR corrected) (Supplementary Table S7). Additionally, the HUD1 group further showed reduced nodal local efficiency ($p = 0.002$) and nodal degree centrality ($p = 0.001$) in the left FPC compared to the HC group (ANOVA tests, all *ps* < 0.08, FDR corrected).

Conversely, compared with the HC group, both HUD groups showed higher nodal efficiency (HUD1: $p = 0.014$; HUD2: $p = 0.026$) and nodal degree centrality (HUD1: $p = 0.018$; HUD2: $p = 0.013$) in the left IFG, as well as higher nodal degree centrality in the right TPJ (HUD1: $p = 0.009$; HUD2: $p = 0.015$). In addition, the HUD1 group exhibited higher nodal efficiency in the right TPJ ($p < 0.001$) (ANOVA tests, all *ps* < 0.015, FDR corrected).

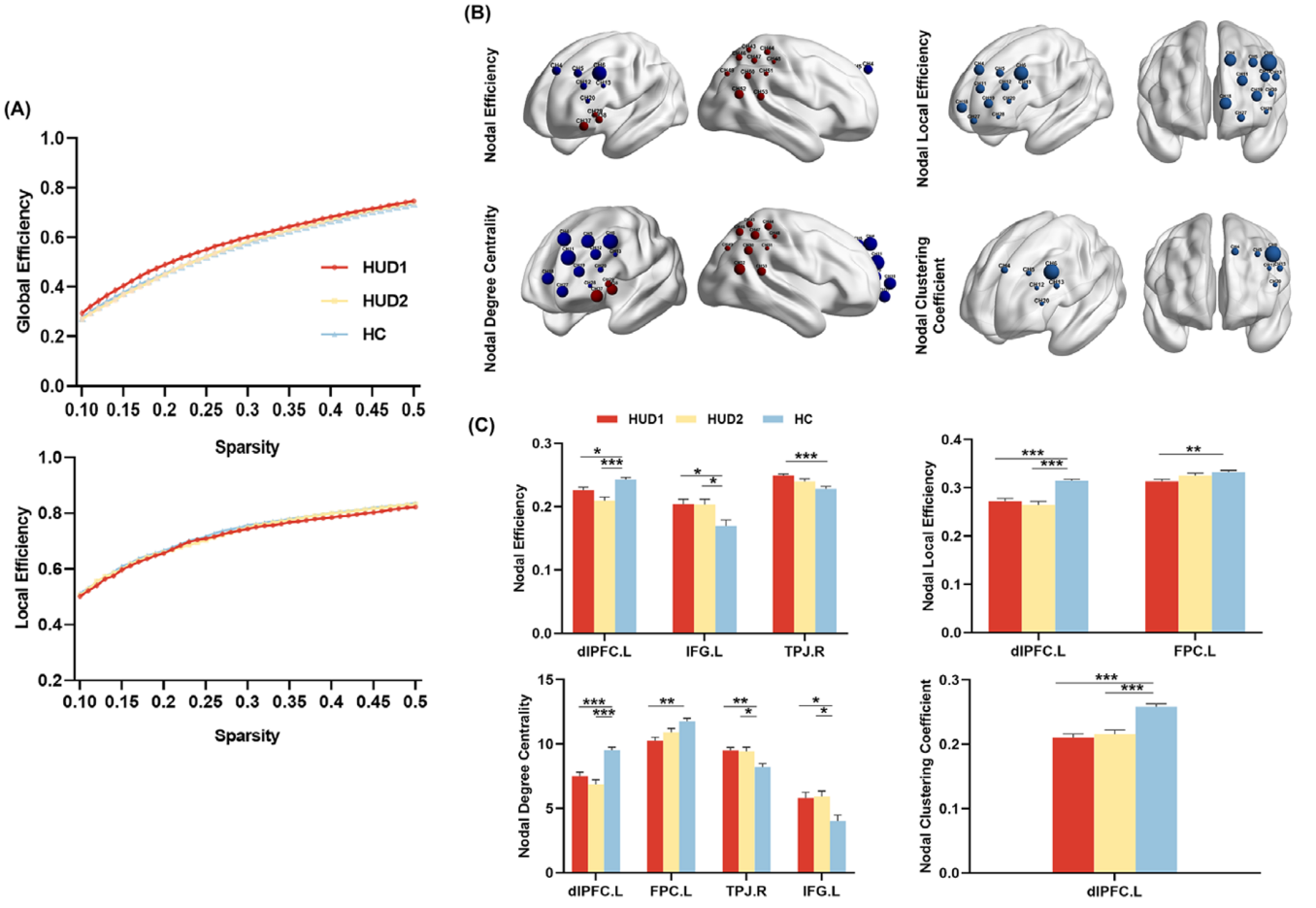


Figure 4. Differences in global and nodal network topological characteristics among three groups. (a) Global network properties in the sparsity range of 10%—50% (Step size = 1%). (b) Nodal distributions within ROIs show significant differences across three groups in nodal network properties. The nodes are shown with different node sizes, indicating the *F* values of ANOVA. The nodes within disrupted ROIs with significantly decreased or increased values related to HUD are respectively shown in blue and red. (c) Group-averaged values of nodal network properties among disrupted ROIs. Error bars represent standard errors of the mean. *Note:* L, 'left sagittal plane'; R, 'right sagittal plane'; F, 'coronal plane'. HUD1: 'heroin use disorder group with short duration of abstinence'; HUD2: 'heroin use disorder group with long duration of abstinence'; HC: 'healthy control group'. $^{\#}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

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Prediction of protracted withdrawal symptoms by task-related brain responses

Pearson's correlation analysis indicated that most of the brain response metrics were significantly correlated with scores on the RPP task (Figure 5a). Notably, in the HUD group, RPP task performance in fluency, flexibility, and originality showed significant negative correlations with emotional and insomnia symptoms of protracted withdrawal (Figure 5b). Moreover, multiple abnormal neural features related to network connectivity were also found to associate with both emotional and insomnia symptoms.

Task-related neural features during divergent thinking significantly predicted emotional, physical, and insomnia symptoms (Emotional: RMSE = 0.34, r = 0.78; Physical: RMSE = 0.59, r = 0.59; Insomnia: RMSE = 0.37, r = 0.83) (Figure 5c). In contrast, the predictive performance for craving-related withdrawal symptoms was poor (RMSE = 1.53, r = 0.20). Among the selected features, FC strength of dlPFC.R_SMG.R, beta values of the OFC.R, and FC strength of dlPFC.R_SMG.L exhibited the highest relative weights in the prediction of emotional, physical, and insomnia symptoms, respectively (Figure 5d). Mediation analyses were further conducted to examine the relationships among the highest-weighted neural features, task performance, and withdrawal symptoms. The results indicated significant indirect effects linking FC strength of dlPFC.R_SMG.R to emotional symptoms via divergent thinking performance (Fluency, 95% CI = [−0.2007, −0.0148]; Flexibility, 95% CI = [−0.2215, −0.0244]; Originality, 95% CI = [−0.1893, −0.0096]) (Figure 5e). Similarly, the association between FC strength of dlPFC.R_SMG.R and insomnia severity was also statistically mediated by divergent thinking performance (Fluency, 95% CI = [−0.2657, −0.0463]; Flexibility, 95% CI = [−0.2635, −0.0450]; Originality, 95% CI = [−0.1988, −0.0341]). In contrast, physical symptoms were not significantly associated with task performance. However, the beta value of the OFC.R was significantly related to both physical symptoms and task performance.

Discussion

This study revealed deficits in divergent thinking in real-world situations and altered fNIRS neural signatures in individuals with HUD. Behavioral results demonstrated that both HUD groups exhibited significantly lower scores than the HC group across four dimensions of fluency, flexibility, originality, and persistence. fNIRS findings revealed significantly higher task-related activations in the right OFC, right dlPFC, and left TPJ in the HUD groups compared to the HC group. In contrast, the HUD groups exhibited significant reduction in functional connectivity strength within the PFC and between the PFC and bilateral TPJ. Additionally, significant reductions in local topological features were observed in the left dlPFC and left FPC in the HUD groups, whereas significant increases were noted in the right TPJ and left IFG. Aberrant brain features during divergent thinking effectively predicted the severity of protracted withdrawal symptoms. Functional connectivity between the right dlPFC and TPJ was identified as a shared neural feature underlying divergent thinking deficits and protracted withdrawal symptoms.

Previous studies have focused more on cognitive functions related to self-control involving drug stimuli and drug craving. The present study provides solid evidence of drug-related impairment in higher-order social cognitive function from the perspective of divergent thinking. In the context of heroin addiction, we observed widespread cortical hyperactivation during the creative problem-solving task, accompanied by disrupted intercommunication within core functional areas. These findings support the hypothesis of compensatory network dysfunction patterns, suggesting functional impairments in brain regions associated with chronic heroin use. Hyperactivation is indicative of neural inefficiency and elevated cognitive load (Wang et al., 2015). Prolonged brain hypoxia may impair the capacity of neural circuits in individuals with HUD to allocate resources effectively (Solis, Cameron-Burr, Shaham, & Kiyatkin, 2017; Younger et al., 2011), resulting in inadequate support for tasks requiring high cognitive demands such as creative problem-solving. As a consequence, increased recruitment of neural resources elevates cortical activation as a compensatory response to neural damage. The reduction in connectivity reflects impaired functional signal transmission between regions, resulting in aberrant functional integration of information required for creative problem-solving. Hypo-connectivity within and between core cognitive control areas has been demonstrated in multiple behavioral addictions (*Chen et al., 2024*). This pattern of hyper-activation and hypo-connectivity caused by network dysfunction has also been observed in typical brain disorders, such as schizophrenia (Wadehra, Pruitt, Murphy, & Diwadkar, 2013).

Opioid drugs induced excessive signaling of m-opioid receptors within the PFC, thereby altering its responsiveness to excitatory drive (Anand et al., 2005; Baldo, 2016). Building on previous research, the PFC is the central region impaired by drug addiction. We identified task-related abnormal activation predominantly within specific subregions of the PFC, particularly the right dlPFC and the right OFC. These findings are consistent with numerous fMRI studies demonstrating reduced gray matter volume in the right PFC among heroin users (Lin et al., 2018; Liu et al., 2009). Moderate activation of the right dlPFC could facilitate goal-directed behavior and support successful exploration within problem spaces (Kleibeuker et al., 2013). However, excessive activation of the right dlPFC may be associated with compulsive behaviors (Khedr et al., 2022). The activity in the OFC may represent the conflict between behavioral options independently of the task (Mansouri, Buckley, & Tanaka, 2014; Stalnaker, Takahashi, Roesch, & Schoenbaum, 2009). The OFC regulates compulsive behaviors, modulating reward-related preferences and seeking behaviors in HUD patients (Guillem, Brenot, Durand, & Ahmed, 2018; Li et al., 2020; Maltby et al., 2005; Seifert et al., 2015). In HUD, heightened OFC activation may indicate impaired regulation between goal-directed and habitual behaviors, promoting drug-seeking tendencies and consequently constraining adaptive problem-solving behaviors under high cognitive demands (Hu et al., 2019; Mansouri et al., 2014; Walter et al., 2015).

In the functional connectivity within the PFC, both HUD groups were observed to have reduced internal short-distance connectivity centered on the dlPFC and FPC. Previous studies have also noted reduced intrinsic functional connectivity within the PFC during rest in HUD patients (Chen et al., 2021; Lin et al., 2018). The dlPFC and FPC are pivotal nodes in the ECN involved in monitoring and regulation, further confirming impaired executive control in HUD patients (Dosenbach et al., 2007). Reduced intra-PFC functional connectivity impairs sustained attention and the inhibition of task-irrelevant distractors, thereby limiting the simultaneous maintenance and manipulation of candidate ideas during idea generation (Benedek et al., 2014). Furthermore, this connectivity deficit limits the integration of goal-directed information, preventing high-order evaluation and systematic optimization, which ultimately leads to diminished performance in divergent thinking tasks (Beaty et al., 2018; Zabelina & Andrews-Hanna, 2016).

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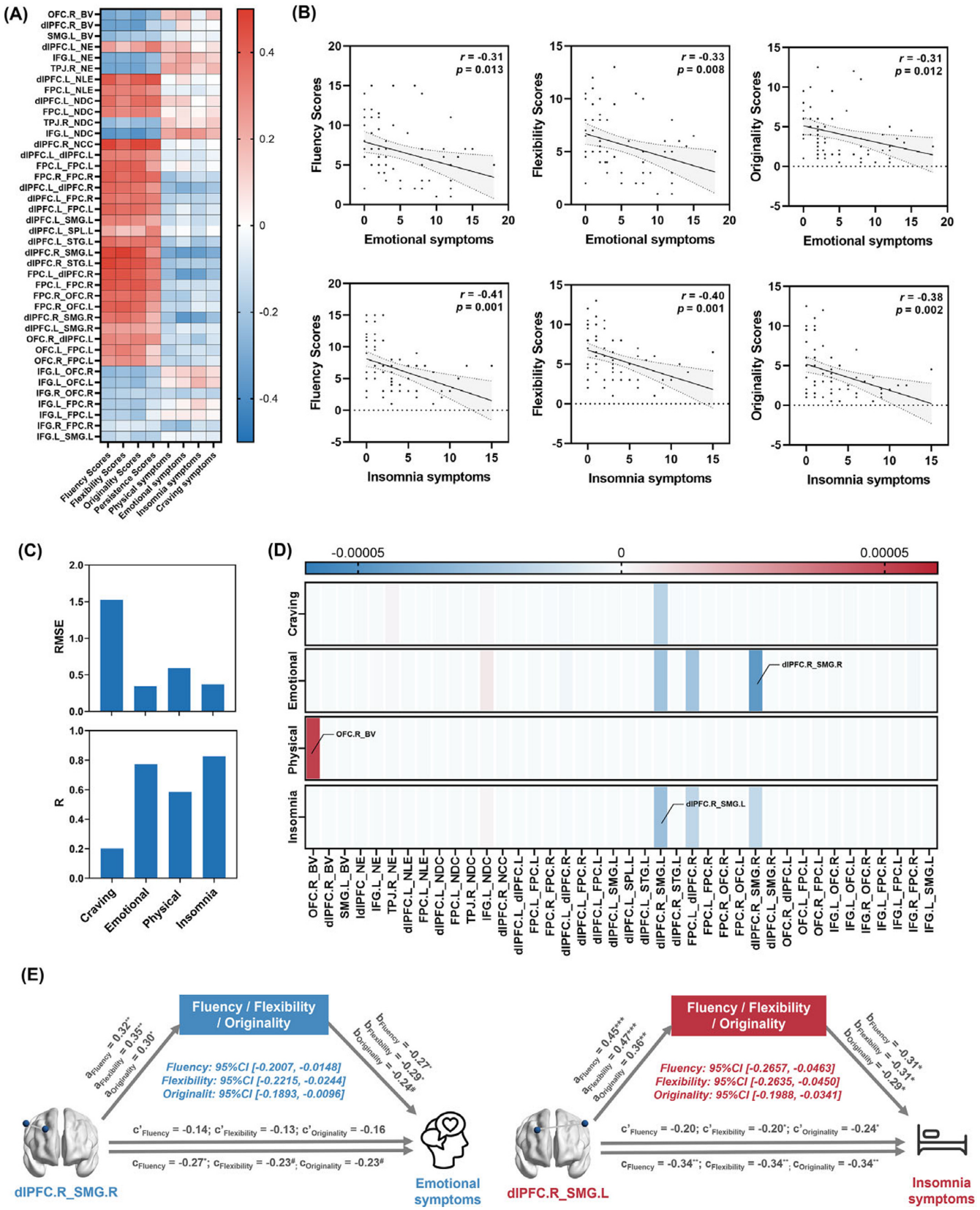


Figure 5. The prediction of protracted withdrawal symptoms by key features from cortical activity. (a) Correlation between all extracted neural features, task performance, and protracted withdrawal symptoms. In addition to the appropriateness dimension, four dimensions of the RPP task that showed significant differences across three groups were included in the analysis. (b) Correlations between the three dimensions of task performance and emotional and insomnia withdrawal symptoms. (c) Prediction performance for the four dimensions of protracted withdrawal symptoms. (d) Prediction weights of neural features. (e) Mediation model of neural features, task performance, and withdrawal symptoms. *Note:* BV: 'beta value'; NE: 'nodal efficiency'; NLE: 'nodal local efficiency'; NDC: 'nodal degree centrality'; NCC: 'nodal clustering coefficient'. $^{\#}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

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The TPJ mainly regulates the processing of divergent thinking, including conceptual updating, schema violation, and recombination of problem representations (Benedek et al., 2014). Abnormal activation in the TPJ was observed in this study. However, decreased functional connectivity was not found in short-range connections within the TPJ, but in the long-range connection between the FPC/dlPFC and the bilateral TPJ. Further network analysis revealed that these nodes exhibit significantly reduced efficiency, local efficiency, degree centrality, and clustering coefficient. Meanwhile, the nodal efficiency and centrality of the right TPJ were significantly increased. These findings further support an imbalance in the allocation of neural resources within the cognitive control network, indicating a strategic shift in cognitive control from top-down to bottom-up processing. This decreased long-range connectivity is also reflected in ineffective core nodal hubs (Y. Liu et al., 2014). These findings further support a decoupling between the ECN and the DMN, accompanied by a reallocation of neural resources from the ECN toward the DMN (Ceceli et al., 2022; Zhang & Volkow, 2019). These network-level alterations may be driven by chronic drug-induced impairments in prefrontal dopaminergic signaling and executive control. This reduces the capacity of the ECN to suppress internally oriented processing, thereby weakening behavioral control while enhancing drug-related reward processing (Koob & Volkow, 2016; Zhang & Volkow, 2019). While the heightened TPJ activity might support spontaneous associative processes (Benedek et al., 2014), this 'unfettered' bottom-up input is unfiltered and poorly integrated due to the weakened PFC–TPJ coupling. Individuals with HUD may manifest as an inability to suppress task-irrelevant introspective states (e.g., mind-wandering and distraction) (Christoff et al., 2016), which makes HUD individuals more prone to prematurely abandon active problem-solving efforts. As evidence, the behavioral data revealed diminished task persistence in HUD individuals.

Machine-learning-based biomarker discovery frameworks can further identify disease-sensitive and interpretable neural features (Zhang et al., 2025, 2026). Functional dysconnectivity between ECN and DMN has also been recognized as a crucial neural basis underlying craving persistence and relapse vulnerability (Meng et al., 2026; Zhang & Volkow, 2019). Consistent with this, our machine-learning predictive models identified weakened rdlPFC–TPJ coupling as a significant predictor of insomnia and emotional withdrawal symptoms. These findings suggest the common dysregulation of multiple systems, such as individual cognition, emotion, and physiological arousal, in HUD caused by PFC dysfunction. The rdlPFC is a key region involved in cognitive control, suppression of irrelevant information, and emotional regulation (Cieslik et al., 2013; Feeser et al., 2014; Jackson et al., 2021). Reduced rdlPFC–TPJ coupling may indicate impaired regulation of emotion-related and self-referential cognitive processing by the executive control system, thereby increasing the persistence of negative affect and rumination. When confronted with negative emotions and ruminative thoughts during abstinence, individuals with HUD may lack effective cognitive regulation and alternative behaviors, thereby exacerbating emotional distress and insomnia symptoms (Demirbas et al., 2012; Gangal et al., 2023; Wang et al., 2026; Watkins & Roberts, 2020). During the sustained recovery period following the acute withdrawal phase, such intensified psychological and physical withdrawal symptoms constitute key contributors to relapse and the development of psychiatric disorders. Furthermore, although no correlation was found between divergent thinking and somatic symptoms, a significant predictive effect of high task-state rOFC activation levels on somatic symptoms was discovered. This result further demonstrates the crucial role of OFC in behavioral and physiological control during drug addiction. In conclusion, the identified prefrontal cortical hubs and their functional alterations provide novel insights into the neural links between cognitive dysfunction and relapse vulnerability, suggesting that targeted modulation of rdlPFC and rOFC functions may represent promising strategies for facilitating cognitive recovery and maintaining long-term abstinence.

In this study, deficits in divergent thinking did not improve with longer abstinence periods, suggesting that these sustained cognitive impairments require more attention in detoxification treatments to help reduce heroin relapse rates. Notably, the left IFG showed significantly higher activity in the HUD group, especially in those with longer withdrawal times. Specifically, the nodal characteristics of the left IFG were significantly higher in HUD2 compared to the control group. The HUD2 group with longer abstinence also showed stronger functional connectivity between the left IFG and the OFC. Previous research has consistently shown an inhibitory effect of the left IFG on creativity scores (Hertenstein et al., 2019; Khalil, Karim, Kondinska, & Godde, 2020; Kleinmintz et al., 2018), and disconnecting the left IFG from the right TPJ through brain stimulation has been found to improve divergent thinking (Huang et al., 2024). Therefore, the increased local network connectivity efficiency in the IFG partially indicates a maladaptive brain-network development pattern related to divergent thinking in individuals with HUD following abstinence.

There are several limitations to the present study. First, while heroin-dependent individuals did not show differences in task performance or brain damage based on abstinence duration, a longitudinal follow-up study is needed to provide additional evidence of developmental changes over time. Accordingly, although the mediation model suggests a potential mechanism linking altered neural activity, divergent thinking impairment, and withdrawal symptoms, longitudinal studies are required to clarify the causal relationships among these factors. Second, this study identified abnormal activation and functional connectivity patterns in the cerebral cortex. However, it would be worthwhile to further investigate the damaged pathways in the neural circuits, including the deep brain nuclei and their impact on social cognitive function. Additionally, impairments in divergent thinking may act as a predisposing factor for addiction. Further research is needed to understand and clarify the causal relationship between drug addiction and cognitive impairment through the exploration of vulnerable groups or the establishment of animal models. Finally, this study found neural pathways similar to those associated with relapse behavior (Li et al., 2018; Zhang & Volkow, 2019). Confirming the link between impaired realistic divergent thinking and high relapse behavior in HUD is critical for improving long-term abstinence outcomes.

Conclusion

In conclusion, the study demonstrated the detrimental impact of HUD on divergent thinking, and such impairment persists despite sustained abstinence. A pathological compensatory mechanism and an imbalance in neural resource allocation were elucidated, highlighting issues in flexible and sustained control of goal-directed behavior in chronic heroin users. The study deepens our understanding of advanced high-order cognitive deficits induced by neurological damage in individuals with drug addiction. Moreover, the results offer reliable neural biomarkers for identifying emotional and insomnia symptoms, paving the way for non-invasive

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brain stimulation as a potential treatment for drug withdrawal and cognitive dysfunction in individuals with drug addiction.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1017/S003329172610614X.

Data availability statement. The datasets generated and/or analyzed and codes during the current study are available from the corresponding author on reasonable request.

Author contribution. Conceptualization: Y.W. and H.D.; Data curation: Y.W., W.F. and W.L.; Formal analysis: Y.W., W.F. and W.L.; Funding acquisition: H.D.; Methodology: Y.W., W.F., W.L. and A.C.; Project administration: H.D.; Resources: C.Y. and H.D.; Supervision: H.D.; Validation: Y.L., K.Y. and A.C.; Visualization: Y.W.; Writing – original draft: Y.W.; Writing – review and editing: Y.W., Y.L., K.Y., C.Y., L.B., A.C. and H.D.

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Competing interests. The authors declare no competing interests.

Ethical standard. All participants provided informed consent, and monetary compensation was given upon completing the experiment. This study adhered to the ethical guidelines outlined in the Helsinki Declaration (World Medical Association, 2013) and received approval from the Ethics Committee (No. L20231117–02). All participants provided written informed consent to participate in this study.

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