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Dissociable ventral and dorsal sensorimotor functional circuits linking the hypomanic personality traits to aggression via behavioral inhibition system

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ABSTRACT

Hypomanic personality traits (HPT) are susceptibility markers for psychiatric disorders, particularly bipolar disorder, and are strongly associated with aggressive behaviors. However, the neuropsychological mechanisms underlying this association remain unclear. This study utilized psychometric network analysis and *Inter-Subject Representation Similarity Analysis* (IS-RSA) to explore the neuropsychological circuits that link HPT to aggression in a large non-clinical population. Psychometric network analysis (n = 716) identified two key nodes: the Behavioral Inhibition System (BIS) and mood volatility, a core dimension of HPT. We observed a positive correlation between mood volatility and aggression, with BIS serving as a mediating factor. Task-based functional imaging (n = 53) further revealed a double dissociation between the dorsal (dSMC) and ventral (vSMC) sensorimotor cortices to HPT, specifically during the processing of reward magnitude and delay in a delayed reward paradigm. Functional patterns within these regions mediated the relationship between individual differences in mood volatility and aggression, with BIS acting as a mediator through parallel pathways. Resting-state functional imaging (n = 505) replicated this functional segregation and revealed distinct integrative patterns: the dSMC was functionally connected to the frontoparietal network (FPN) and the vSMC to the sensorimotor network (SMN). These circuits collectively mediated the associations among mood volatility, aggression, and BIS. These findings highlight the critical role of sensorimotor circuits and BIS in understanding the neuropsychological pathways linking HPT-related mood volatility to aggression.

Introduction

Hypomanic personality traits (HPT) are common risk factors for developing bipolar disorder (BD). Eckblad and Chapman (1986) developed the Hypomanic Personality Scale (HPS) to measure HPT across three key dimensions: mood volatility, social vitality, and excitement. Individuals with prominent HPT exhibit a range of behaviors and positive characteristics, including heightened self-confidence (Stanton et al., 2019), increased energy (Terrien et al., 2014), enhanced creativity (Kim & Kwon, 2017), and improved *Theory of Mind* (ToM) abilities (Terrien et al., 2014). However, these individuals are also at greater risk for psychiatric conditions (Klaus et al., 2020; Miller et al., 2011), irritability (McCarthy-Jones et al., 2012), addiction, prejudice (Meyer & Hautzinger, 2003), circadian rhythm disruptions (Hensch et al., 2019), and increased aggression (King et al., 2020). Notably, the tendency for aggression among individuals with HPT can lead to social

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maladaptation, resulting in interpersonal conflicts, impaired social functioning, and, in severe cases, criminal behavior (Krumm-Merabet & Meyer, 2005; Savitz et al., 2008). Although past studies have linked HPT with aggression (Schalet et al., 2011; Zhu et al., 2023), the neuropsychological mechanisms underlying this association among non-clinical populations remain underexplored. Clarifying these mechanisms can be beneficial for advancing conceptual understanding of manic episodes, aiding in early BD diagnosis and prevention, and informing the development of effective behavioral interventions.

Extensive behavioral research has suggested two key motivational systems—the Behavioral Activation System (BAS) and the Behavioral Inhibition System (BIS)—as psychological mechanisms underlying the HPT and its association with aggression. These systems regulate individual responses to rewards, punishments, and conflicts (Fox et al., 2005; Henderson et al., 2015) and explain personality traits and their behavioral expressions (Farrell & Walker, 2019; Sovereign & Walker, 2021). Dysregulation in BAS activity has consistently been implicated in the development of BD (Hayden et al., 2008; Katz et al., 2021), manifesting in heightened sensitivity, especially during manic episodes (Johnson et al., 2012). However, the role of BIS in mania and hypomania is less clear, with mixed evidence in the literature. Some studies have identified a negative correlation between BIS and manic symptoms among BD patients (Elliot, 2008) and HPT (Jones & Day, 2008). In contrast, longitudinal studies indicate that high BIS sensitivity predicts major depressive episodes but not bipolar spectrum disorders (Alloy et al., 2008), and a meta-analysis has suggested that BIS is not associated with manic episodes in BD (Katz et al., 2021). Given the lack of consistency in BIS's function in hypomania, we aimed to conceptually replicate and validate the association between BIS and HPT in a non-clinical sample. Additionally, based on the established link between BIS and aggression (Martin et al., 2023; Pederson et al., 2018), we explored whether BIS mediates the association between HPT and aggression.

Converging neuroimaging evidence stresses the fundamental role of the sensorimotor cortex (SMC) in HPT and associated impulsive or aggressive behaviors. For example, studies in BD have shown increased functional activation in the precentral gyrus (Ahmed et al., 2023), reduced amplitude of low-frequency fluctuations (ALFF) in the postcentral gyrus and enhanced functional connectivity between the supplementary motor area and other brain regions (Brady et al., 2017). Recent findings indicate that HPT is linked to sex-specific gray matter volume (GMV) patterns in the postcentral and precentral gyri, as well as to structural and functional connectivity between the motor system and other networks, such as the frontoparietal (FPN) and limbic networks (Zhu et al., 2023). Additionally, activity in the motor cortex has been associated with impulsivity and aggression in individuals with prominent HPT (Etkin et al., 2015; Frank et al., 2014; Terrien et al., 2015). Neural responses to immediate and delayed losses during intertemporal decision-making have been shown to predict individual differences in HPT, particularly in motor and frontoparietal areas (Zhu et al., 2023). Further research has consistently highlighted distinctions between the ventral and dorsal regions of the SMC, integrating structural, functional, and connectivity data (Breshears et al., 2015; Syed et al., 2017). This study builds on these findings by investigating whether HPT is differentially associated with the ventral and dorsal sensorimotor circuits and whether these circuits have distinct roles in mediating the relationship between HPT and aggression, alongside the role of BIS as a mediator.

Compared to traditional univariate approaches, *Inter-Subject Representational Similarity Analysis* (IS-RSA) offers a well-established multivariate pattern analysis technique that provides greater sensitivity to subtle individual differences (Finn et al., 2020; Jiang et al., 2024; Li et al., 2024; Tang et al., 2024). This method combines two advances in neuroimaging: the geometric mapping of associations among stimulus features, as proposed in *Representational Similarity Analysis* (RSA) (Kriegeskorte et al., 2008), and the computation of brain-region similarity across subjects, as introduced in inter-subject connectivity analysis (Hasson et al., 2004). This framework posits that individuals who are more similar in their behavioral responses, psychological processing, or personality traits exhibit greater similarity in neural response patterns. Consequently, IS-RSA enables mapping the complex geometric structure of personality traits and behavioral responses within a multidimensional model space onto neural activity patterns, testing whether individuals similar in HPT also display similar brain activity patterns in both resting-state and task-based regions.

This study aims to systematically explore the neuropsychological mechanisms underlying the association between HPT and aggression using psychometric network analysis and IS-RSA in a large, non-clinical sample. We hypothesize that: 1) dispositional HPT is associated with increased aggression; 2) BIS mediates this association; and 3) the ventral and dorsal sensorimotor circuits have distinct functions related to HPT and its associations with aggression.

Materials and methods

Participants

A total of 762 college students participated in this study. Forty-six participants were excluded for the following reasons: First, to ensure the validity of responses, we implemented attention checks. Thirty-four participants failed these checks and were excluded from the analysis due to invalid data. Second, as this study utilized the HPS, BAS/BIS, and BWAQ, it was essential to have complete data for all three measures. Specifically, 5 participants were excluded due to missing BAS_Fun-Seeking scores, 3 due to missing BIS questionnaire scores, and 4 due to missing BWAQ_Indirect scores. After excluding 46 participants due to incomplete data, the final sample consisted of 716 individuals (67.5% female; mean age = 19.85, SD = 1.56 years). The study was structured with three distinct participant groups, each assigned to specific components of the study. All 716 participants completed the required questionnaires, collectively referred to as the *Total* group. Of these, 505 participants (67.5% female; mean age = 19.85, SD = 1.53 years) also underwent resting-state MRI (rs-fMRI) scanning, forming the *rs-fMRI* subset. Additionally, a separate group of 53 participants (60.3% female; mean age = 20.96, SD = 1.63 years) completed an intertemporal choice task, during which task-based functional MRI (t-fMRI) data were collected; this group is referred to as the *t-fMRI* subset. The design allowed for analysis of the questionnaire data across all participants while enabling focused neuroimaging analyses within the rs-fMRI and t-fMRI subsets. **Fig. S1A** illustrates the relationships among the *Total* group and these subsets. None of the participants reported a history of neurological or psychiatric conditions, and all provided written informed consent before participation. This study was approved by the Institutional Review Boards of Tianjin Normal University (No. XL2020-27), China.

Hypomanic personality scale (HPS)

The Hypomanic Personality Scale (HPS) is a 48-item binary (true-or-false) instrument developed to assess tendencies toward HPT (e.g., "There have often been times when I had such an excess of energy that I felt little need to sleep at night"). The HPS demonstrates strong test-retest reliability over fifteen weeks (Eckblad & Chapman, 1986) and is organized into three core subscales: 1) Social Vitality (22 items; α = 0.81), 2) Mood Volatility (15 items, α = 0.80), and 3) Excitement (8 items; α = 0.77). A total HPS score is calculated by summing the responses across all three dimensions, where higher scores reflect elevated levels of HPT.

Buss-warren aggression questionnaire (BWAQ)

The Buss-Warren Aggression Questionnaire (BWAQ), an extension of the Aggression Questionnaire (Buss & Perry, 1992), was designed by

Buss and Warren (2000) to measure individual differences in anger and aggression. BWAQ includes 34 items across five dimensions of aggression: 1) Physical (8 items; α = 0.78; e.g., "I may hit someone if he or she provokes me."), 2) Verbal (5 items; α = 0.67; e.g., "My friends say that I argue a lot."), 3) Anger (7 items; α = 0.64; e.g., "Some of my friends think I am a hothead."), 4) Hostility (8 items; α = 0.73; e.g., "I wonder what people want when they are nice to me."), and 5) Indirect (6 items; α = 0.60; e.g., "I like to play practical jokes."). Higher BWAQ scores indicate greater levels of anger and aggressive tendencies.

Behavioral inhibition/activation scale (BIS/BAS)

The Behavioral Inhibition/Activation (BIS/BAS) Scale is a 24-item instrument developed by Carver and White (1994) based on the *Reinforcement Sensitivity Theory* to measure individual differences in motivation systems. The BIS subscale consists of 7 items (α = 0.77; e.g., "Even when faced with unfortunate events, I rarely feel afraid or nervous."), capturing sensitivity to potential punishment and tendencies to inhibit behavior. The BAS is further divided into three subcomponents: Fun Seeking (4 items; α = 0.69; e.g., "I am always ready to try new things as long as I find them interesting."), Reward Responsiveness (5 items; α = 0.68; e.g., "If things go smoothly, I would be very happy to continue."), and Drive (4 items; α = 0.77; e.g., "To achieve what I desire, I will spare no effort."). The four remaining items are not coded. Higher scores on the BIS/BAS reflect a stronger propensity toward approach motivation (BAS) or inhibition (BIS).

Intertemporal choice task

Participants chose between an immediate reward and a delayed reward set at six months (for additional methodological details, see Wang et al., 2021). The values of the immediate and delayed rewards were adjusted independently, spanning 16 increments (immediate rewards ranged from CNY 25 to 100 in CNY 5 increments; delayed rewards ranged from CNY 28 to 112 in CNY 5.5 increments). These values were established through a pilot study with 14 independent participants (6 males; aged 18–26 years), matched in age and gender.

The task involved 256 trials covering all possible combinations of immediate and delayed options, divided into three runs. The event-related design for stimulus presentation was optimized for estimation efficiency using optseq2 (Dale, 1999). During each trial, both reward options were displayed simultaneously on either side of the screen (see Wang et al., 2023). Participants chose from four response options, ranging from "*strongly prefer the immediate gain*" to "*strongly prefer the delayed gain*," and were instructed to respond as quickly as possible within a 3-second window. The selected choice was highlighted in yellow as immediate feedback. At the end of the task, rewards were assigned based on participants' actual choices (refer to Wang et al., 2023 for details).

Psychometric network analysis

Initial descriptive analyses were performed in JASP v.0.18.0.0, followed by a network analysis to explore the associations among variables. We employed the EBICglasso Networks estimator (Foygel & Drton, 2010; Tibshirani, 2014) using the `glasso` package, which applies a graphical lasso to estimate a sparse Gaussian graphical model (Friedman et al., 2008). The extended Bayesian Information Criterion (EBIC) guided the selection of the tuning parameter. Due to the high number of nodes, the analysis could include "false" correlations; therefore, partial correlation coefficients were used to better represent true interrelations among nodes. Based on these partial correlations, the glasso algorithm regularized the covariance matrix by selecting a penalty parameter through EBIC, thus restricting spurious connections to zero and resulting in a more concise network structure.

Key node indicators in the network included centrality, predictability, and clustering metrics. We calculated betweenness, degree, and closeness centrality, with the tuning parameter (α; Opsahl et al., 2010) set to 0.5. Network robustness was assessed through node centrality stability and edge stability. Node centrality stability was analyzed using subset bootstraps to generate the centrality stability coefficient (CS-coefficient), which quantifies the proportion of data that can be removed while preserving the correlation between the new network and the original one. According to established guidelines, the CS coefficient should ideally exceed 0.5 and not drop below 0.25 (Epskamp et al., 2018). Edge stability was evaluated using 1,000 subset bootstrap samples via the bootnet package in *R*, with edge stability reflected by 95% confidence intervals (CIs); a narrow CI range that excludes zero signifies strong stability. Most analyses conducted in JASP relied on the *R* `bootnet` package (Epskamp et al., 2017), while network diagrams generated in JASP were based on the *R* `qgraph` package (Epskamp et al., 2012).

Mediation analysis

To examine mediation pathways, we conducted both simple and serial mediation analyses using the SPSS PROCESS macro (v3.5; SPSS v26.0), a widely used regression-based mediation analysis tool (Hayes & Rockwood, 2017). Stepwise regression was applied to assess the following associations: 1) between core HPT dimensions (e.g., mood volatility) and aggression ($Y = cX + \varepsilon 1$); 2) between mood volatility and the BIS ($M = aY + \varepsilon 2$); and 3) the mediation of mood volatility and aggression through BIS ($Y = c'X + bM + \varepsilon 3$) (Wen, 2004). In these equations, Y represents the outcome, X represents the predictor, and M serves as the mediator. The indirect effect was computed as a×b. Additionally, we tested a serial mediation model to explore the pathway from mood volatility → BIS → internalized emotional aggression → externalized behavioral aggression. To estimate total and specific indirect effects, we applied bootstrapping with 5000 resamples (Efron, 1994) at 95% Confidence Interval (CI). Effects are statistically significant if zero is not included in the confidence interval (Preacher & Hayes, 2008). Covariates included age, gender, and parental education level to account for potential confounding effects. We applied similar mediation techniques to investigate the associations between mood volatility, BIS, aggression, and associated neural activity indicators (e.g., activation, regional homogeneity [ReHo], and ALFF). Multiple comparisons were corrected using the Bonferroni method.

Brain imaging data acquisition

Neuroimaging data were collected at the Center for MRI Research, using a Siemens 3.0 T Prisma scanner equipped with a 64-channel head coil. Participants laid supine with their heads stabilized using foam padding to minimize movement, and earplugs were provided to reduce noise interference from the scanner. The experimental design was programmed in MATLAB and Psychtoolbox 3.1 (www.psychtoolbox.org) on a Windows machine, with stimuli presented using a mirror system. Responses were recorded using MRI-compatible response buttons. Both resting-state and task-based MRI data were obtained using Siemens' simultaneous multi-slice (SMS) sequences. Scanning parameters for these sequences included: Repetition Time (TR) = 2000 ms, Echo Time (TE) = 30 ms, GRAPPA factor = 2, multi-band acceleration factor = 2, flip angle = 90°, field of view (FOV) = 224 mm × 224 mm, slice thickness = 2 mm, inter-slice gap = 0.3 mm, and voxel size = 2 × 2 × 2 mm^3. The task-based scan spanned 8 minutes and 4 seconds, producing 242 volumes per participant, while the resting-state scan lasted 10 minutes and 13 seconds, yielding 300 volumes per participant. Additionally, a high-resolution T1-weighted image was acquired using a magnetization-prepared rapid acquisition gradient echo (MP-RAGE) sequence (192 slices, TR = 2530 ms, TE = 2.98 ms, multi-band factor = 2, flip angle = 7°, FOV = 224 mm × 256 mm, voxel size = 0.5 × 0.5 × 1 mm^3).

Functional MRI preprocessing and statistical analysis

Task-based fMRI

The Functional MRI Expert Analysis Tool (FEAT, version 5.98), part of the FSL suite (http://www.fmrib.ox.ac.uk/fsl), was used for preprocessing and statistical analyses of the task-based fMRI data. Preprocessing steps involved slice-timing correction, motion correction, filtering, registration, and smoothing, following the methods established in past studies (Wang et al., 2014, 2021). At the first level of analysis, a generalized linear model (GLM) was applied to the decision-making phase, defined from the onset of stimulus presentation until the participant's response. Four parametric regressors were included: 1) a general task regressor (assigned a value of 1 for each trial), 2) the magnitude of the immediate reward, 3) the magnitude of the delayed reward, and 4) reaction time (RT). Each regressor, except for the task regressor, was standardized and convolved with a double-gamma hemodynamic response function. Regressors of non-interest included trials with no response and six motion parameters. At the second level, a fixed-effects model combined the data from all three runs for each participant. Group-level analyses were then conducted using FLAME1 models to assess whether neural responses to immediate and delayed rewards explained individual differences in HPT and its components. To address head motion effects, framewise displacement (FD) was included as a covariate in the main GLM. Cluster-based thresholding was applied to the images, with a height threshold of $z > 3.1$ and a cluster-level $p < 0.05$, corrected for multiple comparisons across the whole brain using Gaussian Random Field Theory.

Resting-state fMRI

Preprocessing of resting-state fMRI data was conducted with fMRIPrep 23.1.3 (Esteban et al., 2019), a Nipype-based tool (Gorgolewski et al., 2011). The preprocessing pipeline, based on the standard fMRIPrep boilerplate (CC0 license), included: 1) correction for susceptibility distortion via fMRIPrep's fieldmap-based (FMB) approach; 2) co-registration of the BOLD reference image to the T1-weighted (T1w) high-resolution anatomical image using boundary-based registration in FreeSurfer's bbregister (Greve & Fischl, 2009), conFig.d with six degrees of freedom; 3) motion correction and extraction of motion parameters using MCFLIRT (FSL, Jenkinson & Smith, 2001); 4) slice-timing correction with AFNI's 3dTshift (Cox & Hyde, 1997, RRID); 5) processing of the T1w images using the "recon all" pipeline in FreeSurfer (version 7.3.2, Dale, 1999); and 6) resampling of all images in a single interpolation step, combining transformations for head motion, susceptibility distortion, and co-registration to anatomical and output spaces. These steps ensured thorough preprocessing, reducing artifacts and enhancing data quality.

The outputs generated by fMRIPrep were further processed with the eXtensible Connectivity Pipeline (XCP) (Ciric et al., 2018; Satterthwaite et al., 2013). The first five volumes from the BOLD data and nuisance regressors, known as 'dummy scans,' were removed to ensure a steady-state signal. The 36P strategy was used for nuisance regression, incorporating 36 confounding regressors: six motion parameters, global signal, mean signals from white matter and cerebrospinal fluid, along with their temporal derivatives and quadratic expansions (Ciric et al., 2017; Satterthwaite et al., 2013). Before denoising, linear trends and intercept terms were added to enhance the removal of nuisance signals. The BOLD data underwent despiking with AFNI's 3dDespike, followed by linear regression in Nilearn 0.10.0 (Abraham et al., 2014) to remove nuisance signals. Any previously censored volumes were interpolated in the residual time series, which was then band-pass filtered with a second-order Butterworth filter to retain frequencies within 0.01-0.1 Hz. The filtered and interpolated time series were re-censored to eliminate high-motion artifacts. The cleaned functional time series were extracted using Nilearn's NiftiLabelsMasker for the MNI atlas, and whole-brain seed-based functional connectivity was calculated.

The ALFF was computed by transforming the preprocessed BOLD time series to the frequency domain, extracting the power spectrum within the 0.01-0.1 Hz band, and calculating the mean square root of the power spectrum for each voxel to produce voxel-wise ALFF maps (Zou et al., 2008). These maps were then smoothed with a Gaussian kernel (FWHM=6.0 mm) in Nilearn. ReHo was computed using AFNI's 3dReHo (Taylor & Saad, 2013), without spatial smoothing to ensure individual variation in the IS-RSA analysis was preserved.

Inter-subject representation similarity analysis (IS-RSA)

IS-RSA was performed using custom MATLAB scripts. Activity maps for each participant under each task condition were derived from unsmoothed GLM beta maps. These beta maps were then divided into 200 distinct parcels based on a NeuroSynth meta-analysis of co-activation patterns (http://neurovault.org/images/39711/). The process for resting-state data was analogous, with ALFF, ReHo, and functional connectivity maps used in place of beta maps. Using a parcellation scheme offers several benefits over traditional searchlight methods. Parcellation is computationally efficient, with each parcel representing bilateral regions aligned with functional neuroanatomy, as opposed to searchlight methods that rely on local spheres, which may not fully capture functionally distinct cortical regions.

Following this, a dissimilarity matrix (or parcel dissimilarity matrix) was created for each parcel, with correlation distance serving as the metric. This distance metric is particularly suitable for comparing data across different scales, such as beta maps or resting-state measures, across participants. A corresponding dissimilarity matrix (behavioral dissimilarity matrix) was also constructed from questionnaire data, including the HPS, BAS/BIS, and BWAQ, using Euclidean distance to quantify differences between each participant pair. This matrix represents individual differences in personality traits, motivation, and aggressive behavior. After vectorizing the lower triangle of both dissimilarity matrices, we calculated the Spearman rank-order correlation between each parcel's dissimilarity matrix and the behavioral dissimilarity matrix.

To assess the statistical significance of the resulting Spearman rho values, we conducted a permutation test by shuffling the rows of one matrix 10,000 times and calculating the proportion of permutations where the randomized rho value exceeded the true rho. These Monte Carlo-derived *p*-values were adjusted for multiple comparisons by multiplying by the number of parcels (200) using the Bonferroni correction. Any *p*-values remaining below 0.05 after correction indicated a significant relationship between behavioral distance and parcel representation distance, suggesting a meaningful association between HPT and multivariate brain activity patterns within the specified parcel.

Results

Demographics

Table 1 provides the sample demographic information and scores for each scale with their comparisons. There was only a minor difference in age across the three subsets ($F_{(2,1271)} = 13.08$, $p < 0.001$, $\eta_p^2 = 0.02$), suggesting that the subsets were homogeneous and valid for cross-validation.

For HPT, gender differences were observed in the *Total* group and *rs-fMRI* subset in social vitality (*Total* group: $t_{(714)} = 2.58$, $p = 0.01$; *rs-fMRI* subset: $t_{(503)} = 2.33$, $p = 0.02$), mood volatility (*Total* group: $t_{(714)} = 3.03$, $p = 0.003$; *rs-fMRI* subset: $t_{(503)} = 2.70$, $p = 0.007$), and total HPT (*Total* group: $t_{(714)} = 2.74$, $p = 0.006$; *rs-fMRI* subset: $t_{(503)} = 2.37$, $p = 0.02$). No significant gender differences were found in excitement (*Total* group: $t_{(714)} = 0.34$, $p = 0.73$; *rs-fMRI* subset: $t_{(503)} = -0.08$, $p = 0.94$). There were no gender differences across any HPT dimension in the *t-fMRI* subset (all $p > 0.72$).

Furthermore, the social vitality dimension was not correlated with age across all subsets (all $p > 0.13$) but was positively correlated with

Table 1
Sample demographics.

Variables	Total Dataset (n = 716)	rs-fMRI Subset (n = 505)	t-fMRI Subset (n = 53)	F / χ^2	p
Gender (Males/ Females)	232/484	185/320	21/32	3.02	0.22
Age (M ± SD)	19.85 ± 1.56	19.85 ± 1.53	20.96 ± 1.63	13.08	<0.001
Paternal education level (%)				0.92	0.40
Less than primary school	13.6	13	20.7		
Junior high school	37.8	37.6	33.9		
Vocational high school	15.5	16.4	16.9		
Senior high school	10.1	10.8	15		
Junior college education	10.1	10.6	5.6		
Undergraduate level	11.1	10.2	7.5		
Graduate or above	1.3	0.9	0		
Maternal education level (%)				0.74	0.48
Less than primary school	19.1	19.4	28.3		
Junior high school	34.4	33.8	28.3		
Vocational high school	15	13.2	15		
Senior high school	12.1	13.2	13.2		
Junior college education	8.5	9.5	9.4		
Undergraduate level	9.7	9.9	5.6		
Graduate or above	0.8	0.7	0		
BWAQ					
Physical aggression	16.28 ± 5.51	16.44 ± 5.44	17.23 ± 5.93	0.76	0.47
Verbal aggression	12.80 ± 3.31	12.74 ± 3.24	13.06 ± 3.67	0.23	0.79
Anger	16.20 ± 3.94	16.11 ± 3.97	16.85 ± 3.81	0.83	0.43
Hostility	20.46 ± 4.79	20.46 ± 4.88	21.13 ± 4.68	0.49	0.61
Indirect aggression	14.24 ± 3.77	14.18 ± 3.83	14.57 ± 3.90	0.25	0.78
Total aggression	79.99 ± 16.40	79.94 ± 16.52	82.83 ± 17.59	0.77	0.47
HPS					
Social Vitality	68.95 ± 12.44	69.60 ± 12.24	70.49 ± 11.65	0.68	0.50
Mood Volatility	50.25 ± 9.17	50.50 ± 8.93	50.75 ± 9.03	0.16	0.85
Excitement	25.81 ± 6.17	25.91 ± 5.97	25.77 ± 6.31	0.04	0.96
Total score	145.01 ± 22.70	146.01 ± 22.03	147.02 ± 22.45	0.42	0.66
BAS					
Fun Seeking	7.48 ± 2.16	7.49 ± 2.02	8.02 ± 1.89	1.64	0.19
Reward Responsiveness	7.87 ± 2.10	7.89 ± 2.03	8.25 ± 2.11	0.82	0.44
Drive	8.10 ± 2.27	8.14 ± 2.23	8.32 ± 2.17	0.28	0.76
BIS	14.47 ± 3.40	14.51 ± 3.37	14.64 ± 3.40	0.08	0.92

Note: M = Mean; SD = Standard Deviation; BWAQ = Buss-Warren Aggression Questionnaire; BIS/BAS = Behavioral Inhibition/Behavioral Activation Scale; HPS = Hypomanic Personality Scale.

paternal (*Total* group: $r = 0.13$, $p < 0.001$; *rs-fMRI* subset: $r = 0.11$, $p = 0.01$) and maternal education level (*Total* group: $r = 0.14$, $p < 0.001$; *rs-fMRI* subset: $r = 0.14$, $p = 0.002$). Mood volatility negatively correlated with age (*Total* group: $r = -0.10$, $p = 0.009$; *rs-fMRI* subset: $r = -0.10$, $p = 0.03$) and positively correlated with paternal education level (*Total* group: $r = 0.10$, $p = 0.006$; *rs-fMRI* subset: $r = 0.12$, $p = 0.006$). However, we did not observe a similar pattern for the excitement dimension regarding age or parental education levels (all $p > 0.32$).

Finally, total HPT scores were positively correlated with both paternal (*Total* group: $r = 0.11$, $p = 0.004$; *rs-fMRI* subset: $r = 0.11$, $p = 0.01$) and maternal education levels (*Total* group: $r = 0.10$, $p = 0.01$; *rs-fMRI* subset: $r = 0.11$, $p = 0.02$), while showing a negative correlation with age only in the *Total* group ($r = -0.08$, $p = 0.03$). However, no significant correlations for any dimensions with age or parental education levels were found in the *t-fMRI* subset (all $p > 0.72$).

Core nodes in the personality-motivation-aggression network

To systematically examine the pathways through which HPT may influence aggression via the behavioral motivation system, we first conducted a psychometric network analysis to identify key nodes within this framework. In this model, dimensional variables were represented as nodes, while correlations between these variables were treated as edges, producing a symptom network model that outlines the interrelationships among HPT, BIS/BAS, and aggression (Fig. 1**A**). Betweenness centrality was chosen as the primary metric for identifying core mediating variables, with results indicating that the BIS serves as the central mediating variable in the Personality-Motivation-Aggression Network (Fig. 1**B**).

Additionally, degree centrality (or strength) was used as a secondary measure to evaluate the principal components of HPT that contribute to aggressive behavior. Results showed that mood volatility had the highest degree of centrality (Fig. 1**B**), suggesting its prominent role in the network. Hostility was consistently positioned as central to aggressive behavior across all centrality measures—betweenness, degree, and closeness centrality—highlighting that internalized emotional aggression may be a critical target for intervention (Fig. 1**B**, Supplementary **Fig. S1C**).

BIS mediates the impact of mood volatility on aggression

This study aimed to investigate whether and how mood volatility could trigger various forms of aggression through BIS. After controlling for gender, age, and parental education, BIS mediated the effects of mood volatility on total aggression (indirect effect = 0.012, 95% CI [0.002, 0.028]), anger (indirect effect = 0.018, 95% CI [0.003, 0.035]), and hostility (indirect effect = 0.030, 95%, CI [0.005, 0.057]) (Fig. 1**C**).

Results also showed a significant serial mediation effect of mood volatility → BIS → anger → physical aggression (indirect effect = 0.008, 95% CI [0.001, 0.015], Fig. 1**D**, right), verbal aggression (indirect effect = 0.006, 95% CI [0.001, 0.012], Fig. 1**D**, middle), and indirect aggression (indirect effect = 0.007, 95% CI [0.001, 0.015], Fig. 1**D**, left). Similarly, significant serial mediation effects were found for mood volatility → BIS → hostility → physical aggression (indirect effect = 0.012, 95% CI [0.002, 0.023], Fig. 1**E**, right), verbal aggression (indirect effect = 0.011, 95% CI [0.002, 0.022], Fig. 1**E**, middle), and indirect aggression (indirect effect = 0.012, 95% CI [0.002, 0.024], Fig. 1**E**, left).

Dissociable ventral and dorsal sensorimotor circuits supporting the mood volatility

We used IS-RSA to investigate the underlying neural mechanisms in a subset of participants (n = 53) from the *t-fMRI* subset. Our analyses revealed a double dissociation effect between the ventral and dorsal sensorimotor cortices concerning HPT during the processing of time and amount related to delayed rewards. Specifically, inter-subject variations in mood volatility were associated with neural activation patterns in the ventral SMC (vSMC; Spearman's $\rho = 0.132$; $p_{permutation} = 0.02$) but not in the dorsal SMC (dSMC; Spearman's $\rho = 0.071$; $p_{permutation} = 0.99$) when evaluating the delay time of future rewards. Conversely, when assessing the quantity of future rewards, no significant association was found in the vSMC (Spearman's $\rho = 0.075$; $p_{permutation} = 0.59$), while a significant

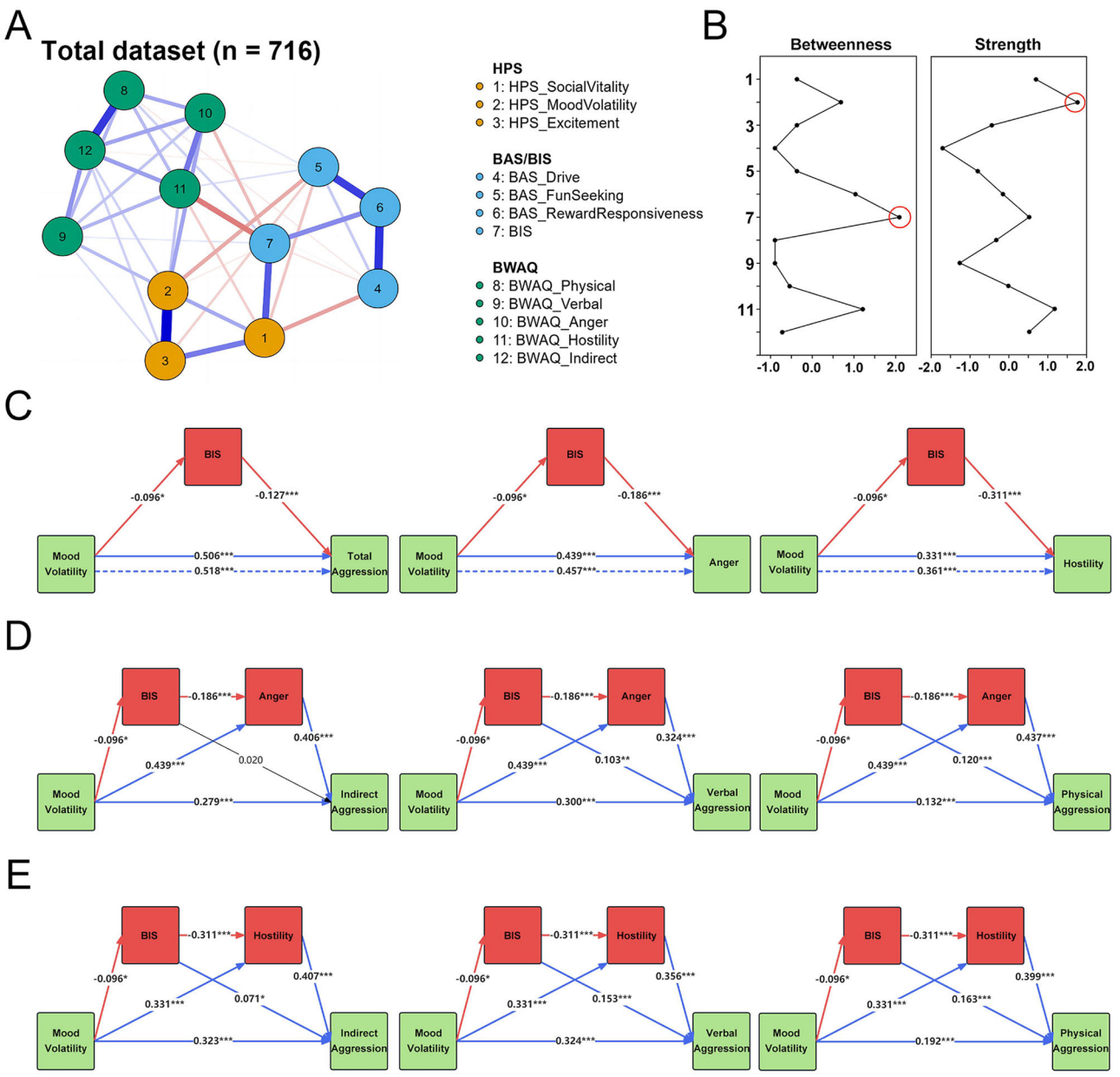

Fig. 1. Two critical hubs from network analysis and potential mediation effects in the Total group (n = 716).
(A) The symptom network was constructed, including HPS (orange), BAS/BIS (blue), and BWAQ (green). Nodes represent psychological variables, and edges indicate correlations between any pair of nodes, where blue denotes positive correlations and red denotes negative correlations, with line thickness indicating strength. (B) Betweenness centrality and strength centrality of each variable in the symptom network are displayed. The red circle highlights the highest centrality positions, where BIS exhibits the highest betweenness centrality, and mood volatility shows the highest strength centrality. (C) BIS mediated the associations between mood volatility and total aggression (left) and internalized emotional aggression (anger: middle; hostility: right). Serial mediation effects of BIS through anger (D) and hostility (E) were observed on the relationships between mood volatility and external aggression behaviors, including indirect aggression (left), verbal aggression (middle), and physical aggression (right). Red lines indicate negative predictions; blue lines indicate positive predictions, with bold lines for significant paths, and thin black lines for non-significant paths. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

correlation was observed in the dSMC (Spearman's ρ= 0.120; $p_{permutation}$ = 0.02, Fig. 2**A** & 2**C**).

We conducted an ROI-based IS-RSA using two resting-state brain activity indicators—ALFF and ReHo—to investigate the spontaneous brain neuronal activity in the *rs-fMRI* subset (n = 505). For ALFF, spontaneous neuronal activity patterns in the vSMC correlated with HPT (Spearman's ρ = 0.062; $p_{permutation} < 0.001$), while the dSMC did not show a significant effect (Spearman's ρ = 0.004; $p_{permutation} = 0.540$). When examining ReHo, significant similarity effects were found in both the vSMC (Spearman's ρ = 0.063; $p_{permutation} < 0.001$) and dSMC (Spearman's ρ = 0.036; $p_{permutation} < 0.001$). These findings suggest functional segregation during spontaneous brain activity, highlighting the different roles of local activity intensity (ALFF) and local functional properties (ReHo) in the sensorimotor cortices.

Brain neural patterns and BIS mediate the association between mood volatility and aggression with parallel pathway

We investigated how the observed double dissociation pattern mediates the association between mood volatility and aggression together with BIS. We found that neural activation patterns in the dSMC during the processing of the amount of future rewards, in conjunction with

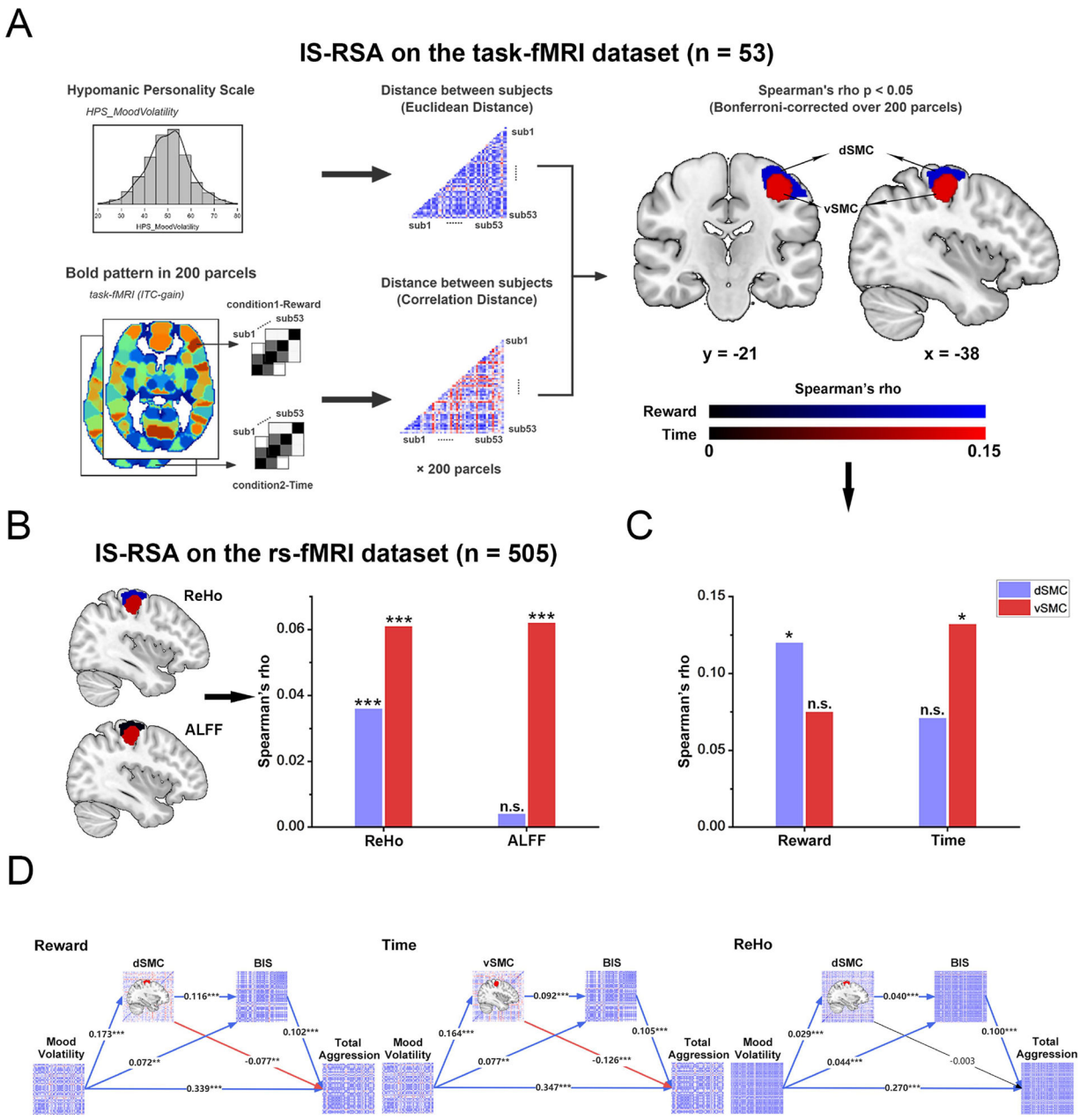

Fig. 2. Dissociation effects between ventral and dorsal SMC and their mediations on the relationships between mood volatility and aggression.
(A) The specific flows of IS-RSA analysis are given showing that individuals with similar mood volatility traits exhibit similar neural activity patterns in brain parcels when processing reward magnitude (blue: dSMC) or time delay length (red: vSMC) in the t-fMRI subset (n = 53). Spatial similarity in neural activity between participants was calculated via correlation distance whereas the behavioral similarity in mood volatility was calculated by Euclidean distance, with representation similarity measured through Spearman correlation. For the dSMC and vSMC, similar double-dissociated patterns were observed in both the rs-fMRI (n = 505) (B) and t-fMRI subsets (C) through IS-RSA. The neural patterns of the SMC during the rest and task conditions and the BIS-mediated associations between emotional volatility and aggression in the serial pathway are shown in (D). These pathways included intersubject variations in mood volatility → activation patterns in the dSMC (e.g., when processing reward) → BIS → total aggression (left), mood volatility → activation patterns in the vSMC (e.g., when processing time length) → BIS → total aggression (middle), and mood volatility → ReHo patterns in the dSMC → BIS → total aggression (right). Red lines indicate negative predictions, while blue lines indicate positive predictions, with bold lines for significant paths, and thin black lines for non-significant paths. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

inter-subject variations in BIS, mediated the association between mood volatility and total aggression. Specifically, there was a significant serial mediation effect: mood volatility → dSMC → BIS → total aggression (indirect effect = 0.002, 95% CI [0.001, 0.004], Fig. 2**D**, left). Similarly, the vSMC's activation patterns, when processing time information of future rewards, mediated the pathway from mood volatility to total aggression: mood volatility → vSMC → BIS → total aggression (indirect effect = 0.002, 95% CI [0.001, 0.003], Fig. 2**D**, middle).

These serial mediation effects were also evident in models examining individual variability in hostility (Reward: indirect effect = 0.002, 95% CI [0.001, 0.004]; Time: indirect effect = 0.001, 95% CI [0.001, 0.003], **Fig. S2A**) and anger (Reward: indirect effect = 0.002, 95% CI [0.001, 0.003]; Time: indirect effect = 0.001, 95% CI [0.0004, 0.003], **Fig. S2B**). This indicates that specific neural activation patterns in the sensorimotor cortices play critical roles in mediating the effects of mood volatility on various forms of aggression, with BIS acting as a key intermediary in this process.

Additionally, a similar phenomenon of functional segregation was

observed in spontaneous brain activity patterns within the SMC. The serial mediation effect of mood volatility → dSMC (ReHo) → BIS → total aggression was significant (indirect effect = 0.0001, 95% CI [0.0001, 0.00014], Fig. 2**D**, right), while the serial mediation effect involving the vSMC (ReHo) was not significant. Interestingly, individual variability in the ALFF of spontaneous brain activity within the SMC did not predict the pathway model, suggesting that ReHo, rather than ALFF, is more relevant for understanding the mediation pathways linking mood volatility, SMC activity, BIS, and aggression.

Specific functional connectivity patterns of sensorimotor circuit linking HPT to aggression

We further investigated how the functional connectivity patterns between the left SMC and other regions contribute to mood volatility, emphasizing the functional integration of large-scale brain networks. Our findings showed that the whole-brain functional connectivity patterns of both the dSMC and vSMC associated with mood volatility exhibited similar distribution characteristics (Fig. 3**A & B**). Notably, the shared network connections were primarily distributed in the DMN (26.0%), FPN (20.1%), and DAN (16.4%) (**Fig. S3B**).

We examined the non-overlapping functional connectivity of dSMC and vSMC and their corresponding connections to large-scale brain networks to explore the specific mechanisms of functional integration for the two seed regions in representing individual differences in mood volatility. Our analysis revealed distinct distribution patterns specific to mood volatility within the whole-brain functional connectivity map (Fig. 3**C**), highlighting differences selectively in the sensorimotor network (SMN) (dSMC: 2.7%; vSMC: 68.7%) and FPN (dSMC: 17.7%; vSMC: 0.4%) brain networks (Fig. 3**D**).

We subsequently tested whether the mood-volatility-specific functional integration patterns mediate the pathway model. The results

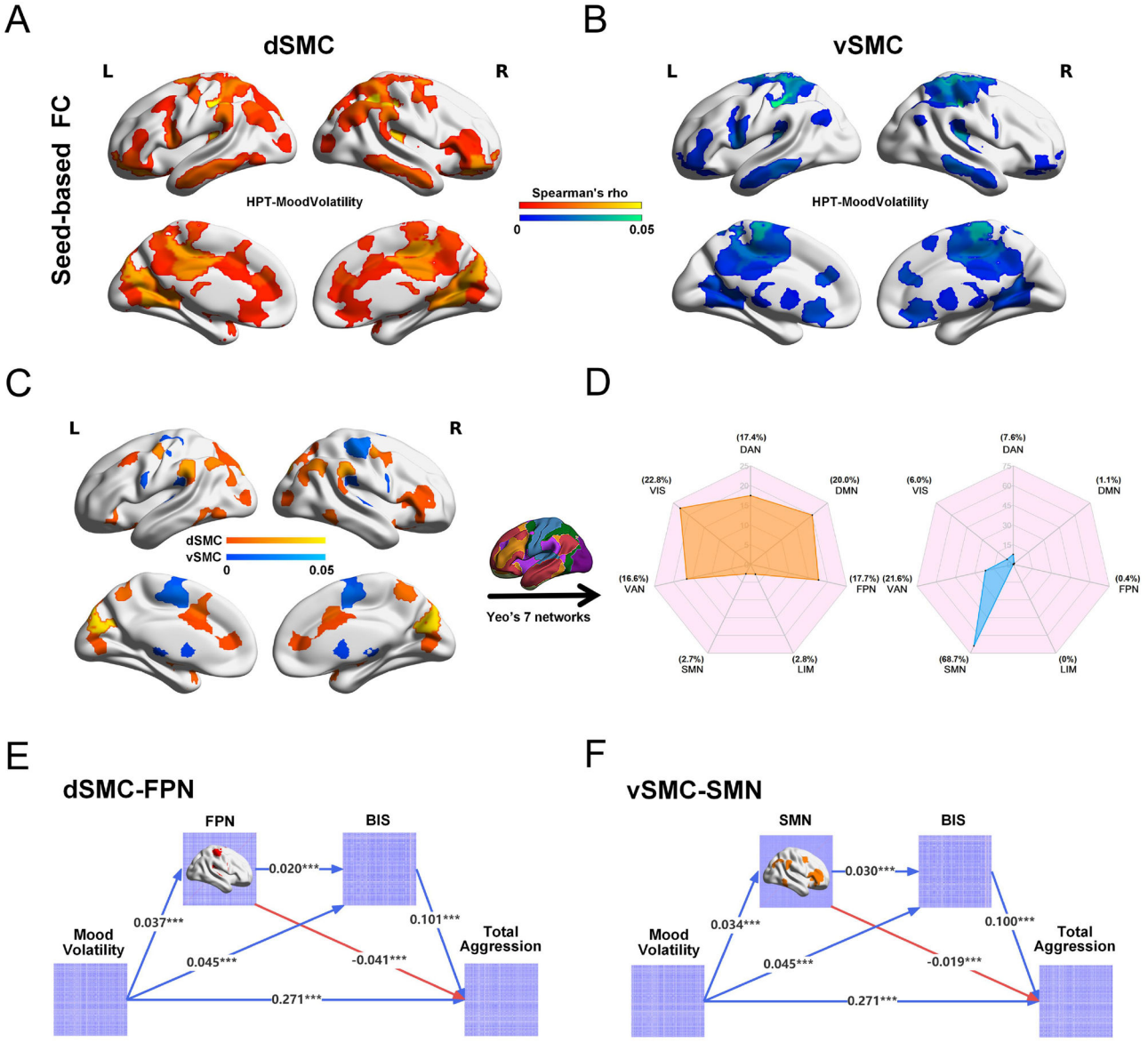

Fig. 3. Seed-based functional connectivity patterns predicting mood volatility and the potential chain mediation effects in the rs-fMRI subset (n = 505). Functional connectivity patterns of the dSMC (A) and vSMC (B) seeds were associated with inter-subject variations in mood volatility. The color bar represents Spearman correlation coefficients (orange: dSMC; blue: vSMC). Whole-brain IS-RSA revealed distinct patterns of the dSMC and vSMC seeds with the specific regions (C) and their distributions in the brain network level (D) based on Yeo's 7-network template. The radar plots depict proportions of significant voxels within each of the 7 networks. Functional connectivity patterns of dSMC-FPN (E) and vSMC-SMN (F) mediated the associations between mood volatility and aggression with BIS in a parallel way.

showed significant serial mediation effects involving the dSMC and the FPN as well as vSMC and SMN. Specifically, there were significant mediation effects for mood volatility → dSMC-FPN connectivity → BIS → total aggression (indirect effect = 0.0001, 95% CI [0.00008, 0.00013], Fig. 3**E**, left) and mood volatility → vSMC-SMN connectivity → BIS → total aggression (indirect effect = 0.00007, 95% CI [0.00005, 0.00011], Fig. 3**E**, right). Similar mediation effects were also observed for hostility (**Fig. S3C**) and anger (**Fig. S3D**). These findings suggest that inter-regional functional integration between dSMC and FPN, and intra-regional functional integration within SMN, may exert different roles in the pathway models linking HPT to aggressive behaviors.

Functional attribution of vSMC and dSMC via neurosynth

We used Neurosynth (https://www.neurosynth.org/) to decode the functions of the vSMC and dSMC to HPT and their association with aggression. The center coordinates of vSMC (x = -38, y = −22, z = 54) and dSMC (x = -40, y = −22, z = 68) were inputted into Neurosynth for location-based analysis.

For dSMC, the top 15 cognitive or social function-related terms ranked by posterior probability, included (Fig. 4**A**): “response selection” (0.79), “sensory motor” (0.78), “cognitive emotional” (0.74), “control processes” (0.71), “execution” (0.71), “exploration” (0.70), “effort” (0.69), “inhibitory control” (0.68), “recognition memory” (0.68), “confidence” (0.66), “encoding retrieval” (0.65), “motor control” (0.64), “anger” (0.63), “autobiographical” (0.62), and “negative affect” (0.61). Similarly, for vSMC, the top 15 terms were (Fig. 4B): “motor task” (0.85), “coordination” (0.78), “execution” (0.76), “detection task” (0.70), “exploration” (0.70), “illusion” (0.69), “confidence” (0.68), “planning” (0.66), “sequence” (0.65), “reaction time” (0.65), “cognitive tasks” (0.64), “executive functions” (0.63), “estimation” (0.63) “monitoring” (0.60), and “decision making” (0.59).

Based on these meta-analytic findings, the functions of dSMC extend beyond sensorimotor processes to include domains related to executive control, self-referential processing, emotional regulation, and memory. In contrast, although vSMC shares some overlap with dSMC in areas like sensorimotor processing, task engagement, and executive functions, it diverges significantly in terms of function. For instance, vSMC is not involved in control processes and is primarily associated with time-related processes such as planning, sequence processing, reaction time, and monitoring.

Discussion

We used psychometric network analysis and IS-RSA to investigate the neuropsychological mechanisms linking HPT with aggression in a large non-clinical sample. Focusing on BIS and the SMC, we identified the pathways through which mood volatility is linked to externalized aggressive behaviors (e.g., physical and verbal aggression) and highlighted the roles of BIS and internalized emotional aggression (e.g., anger and hostility). At the neural level, we identified significant brain activity patterns—including brain activation, ReHo, ALFF, and functional connectivity indices—within the dSMC and vSMC. These patterns were essential in understanding mood volatility’s relationship with aggression, mediated through a parallel pathway involving BIS. This study provides a foundational systematic examination of the distinct roles of the vSMC and dSMC to HPT, offering novel insights into the neuropsychological pathways to aggression in individuals with a propensity for hypomania.

Psychological pathways linking mood volatility and externalized aggression

Previous research has linked overt aggression with BD (Ballester et al., 2012; Látalová, 2009), and similar associations have been observed between HPT and aggression in non-clinical populations, where individuals with prominent HPT display increased aggression and mood volatility (King et al., 2019; Zhu et al., 2023). Our study replicates these findings, benefiting from a large sample (n = 716), and identifies two psychological pathways that may drive this association, focusing on BIS and internalized emotional aggression (e.g., anger and hostility). Notably, BIS mediated the impact of mood volatility on internalized emotional aggression, while both BIS and internalized aggression together mediated the link between mood volatility and externalized aggression through a parallel pathway.

The negative association between BIS activation and internalized aggression suggests that BIS may function as a protective mechanism to inhibit experiences of anger and hostility. According to the Reinforcement Sensitivity Theory, appetitive and aversive systems drive adaptive behavior, offering a framework to understand personality (Bijttebier et al., 2009; Corr, 2004). Individuals with more active BIS are more likely to experience anxiety in response to potentially harmful stimuli, thereby reducing tendencies toward both internalized and externalized aggression. This perspective is supported by neuroimaging research

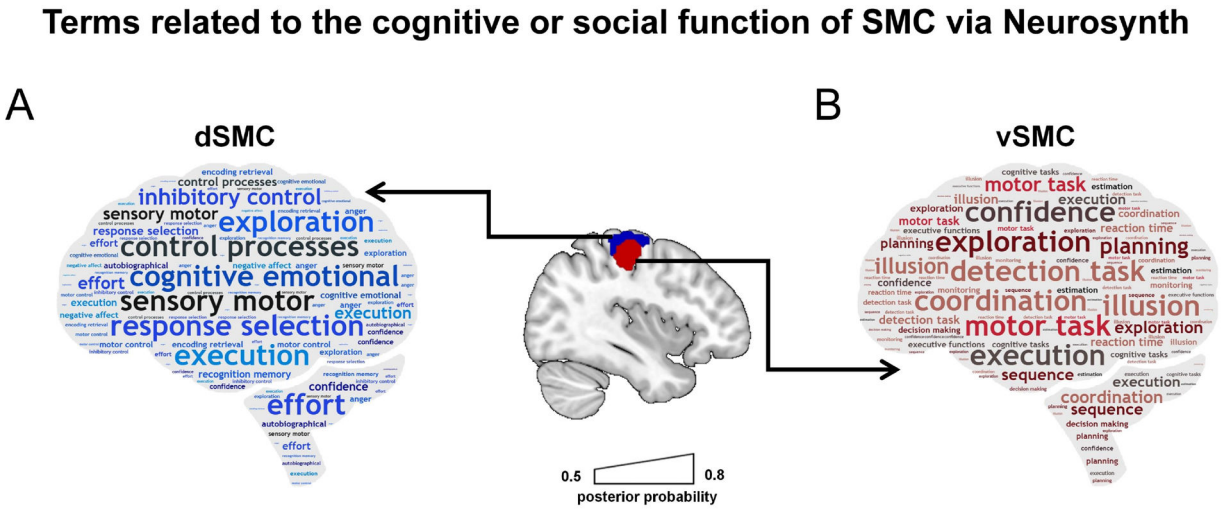

Fig. 4. Location-based decoding findings for the dSMC and vSMC via Neurosynth.
Terms associated with the cognitive or social functions of dSMC (A) and vSMC (B) were presented. Terms are ranked by the posterior probability—the conditional probability of a term being used in a study conditional on activation being present at this voxel. For instance, a posterior probability of 0.8 indicates that 80% of studies reporting activation at this voxel also use this term in their abstracts.

showing impaired response inhibition and heightened impulsivity in individuals with high trait aggression, alongside reduced activation in the pre-supplementary motor areas and motor cortex—areas associated with response inhibition (Pawliczek et al., 2013). We hypothesize that BIS activation serves as a regulatory mechanism, particularly in those experiencing mood volatility, where increased awareness of negative consequences tempers aggressive impulses and mitigates negative emotional states. This insight has important implications for developing therapeutic interventions aimed at enhancing BIS sensitivity in individuals prone to aggression.

In contrast, internalized emotional aggression, marked by self-directed negative emotions and unresolved anger, was positively correlated with externalized aggression (e.g., physical and verbal aggression). This finding aligns with literature documenting the link between internalized anger and outward aggression (Buss & Perry, 1992; Maalouf et al., 2022). According to the *Frustration-Aggression Hypothesis*, frustration can incite aggressive tendencies even when not directed toward a particular individual (Berkowitz, 1965, 1989). Suppressed anger or hostility may accumulate to manifest as outward expressions of aggression, particularly among men (Kim et al., 2022). For individuals with high levels of internalized emotional aggression, challenges in emotional regulation may create a spillover effect, where internal tensions drive external aggressive behaviors.

Our parallel mediation model revealed that BIS and internalized emotional aggression together mediate the association between mood volatility and aggression, providing nuanced insight into how mood volatility translates into aggressive actions. Although BIS activation appears to inhibit aggression (Hofman & Schutter, 2012; Smits & Kuppens, 2005), internalized aggression intensifies it (Maalouf et al., 2022), and the interaction between these two pathways may be crucial in predicting aggressive outcomes. This dual-pathway model deepens our understanding of the roles of inhibitory control and emotional regulation in the formation of aggression. Furthermore, this model underscores the value of targeted interventions that enhance inhibitory control (e.g., cognitive-behavioral strategies to increase BIS responsiveness) and address internal emotional conflicts (e.g., therapies such as emotion regulation training or mindfulness-based approaches).

Double dissociation in sensorimotor contributions to mood volatility

We provide evidence for a double dissociation between the dSMC and vSMC in their roles supporting HPT, particularly mood volatility. Specifically, we observed that functional activation in the dSMC during reward magnitude processing in a delayed reward task correlated with individual variability in mood volatility. Conversely, vSMC activation during the processing of temporal aspects of delayed rewards also showed a notable association with mood volatility. This double dissociation offers a unique perspective on how distinct sensorimotor regions contribute to mood regulation linked with HPT.

The dSMC's role in processing reward magnitude, associated with mood volatility, suggests that this region may be integral in evaluating future rewards and their emotional relevance. Individuals with heightened mood volatility may show greater fluctuations in reward-related neural responses, potentially contributing to inconsistent emotional regulation and impulsive tendencies when facing delayed rewards (Zhu et al., 2023). Prior research has highlighted disrupted reward processing in individuals with hypomanic or manic symptoms, such as reduced reward learning in probabilistic tasks (Pizzagalli et al., 2008) and a tendency to prefer immediate rewards (Mason et al., 2012). In contrast, the vSMC's role in processing temporal information about delayed rewards suggests a distinct neural mechanism through which time perception, episodic thinking, and emotional stability interconnect. This may reflect a sensitivity to delayed outcomes, where individuals with mood volatility struggle with anticipating rewards over time, contributing to impulsivity and affective instability. This insight may clarify why hypomania is often related to impulsive decision-making in response to immediate options (Mason et al., 2012).

The double dissociation between dSMC and vSMC in supporting different aspects of reward processing and episodic thinking provides a neural basis for understanding mood volatility associated with HPT in clinical and non-clinical populations. Traits like elevated mood, heightened reward sensitivity, and difficulties with episodic thinking and emotional regulation are often early indicators of mood disorders such as bipolar disorder (Johnson et al., 2012). The specific roles of dSMC and vSMC in processing reward magnitude and temporal information suggest that distinct areas within the SMC may support separate psychological processes, thereby contributing to the erratic emotional and behavioral patterns observed in HPT.

We observed a similar double dissociation pattern in the SMC across various resting-state functional measures, including ALFF, ReHo, and functional connectivity. ALFF patterns in the vSMC were linked with individual differences in mood volatility, while the dSMC did not show a similar association. ReHo exhibited a comparable pattern, with stronger associations in the vSMC than the dSMC. In terms of functional connectivity, a notable pattern emerged: vSMC's connectivity with the SMN was specifically associated with mood volatility, while dSMC's connectivity with the FPN showed a stronger link to mood volatility than vSMC. These findings support the double dissociation within the SMC from the perspective of spontaneous neural activity and connectivity.

Although previous studies have documented specific ReHo and ALFF changes in the SMC in clinical populations with mania (Cui et al., 2016; Xiao et al., 2013), this study provides an early exploration into these neural characteristics underlying HPT in a non-clinical population. ReHo reflects local neural synchronization, while ALFF measures spontaneous neural fluctuations at rest (Zang et al., 2007). Both indices effectively capture the functional characteristics of the human brain and are often considered biomarkers for various psychiatric conditions, including BD (Nan et al., 2024), autism (Paakki et al., 2010), and ADHD (Cortese et al., 2021). Combined with task-based brain activation patterns, the SMC's functional patterns—not merely the activation strength—may play a critical role in the development and manifestation of HPT. Further functional decoding using meta-analytic tools, such as Neurosynth, revealed that the dSMC is involved in executive control, self-referential processing, and emotional regulation, while the vSMC is associated with planning and monitoring. These findings suggest that HPT may reshape specific psychological processes, including reward and emotion processing, top-down cognitive control, episodic projection, and their interactions.

Beyond ReHo and ALFF, intrinsic brain connectivity networks have been utilized to explore the underlying mechanisms of human behavior and psychiatric disorders, including schizophrenia (Cheng et al., 2024; Mo et al., 2024) and suicidality (Zhang et al., 2024). In the present study, we identified distinct functional connectivity patterns linked to HPT in the dSMC and vSMC, particularly with the FPN and SMN, respectively. This distinction emphasizes functional specialization within the sensorimotor system: the dorsal region may be governed by top-down cognitive control from the FPN, whereas the ventral region may integrate high-order sensory and motor information. Previous research has implicated the FPN in cognitive control and the regulation of reward, emotion, and goal-directed behavior (Marek & Dosenbach, 2018; Zanto & Gazzaley, 2013), which are all relevant to the elevated reward sensitivity, mood instability, and goal-oriented behaviors seen in individuals with HPT (Barnett et al., 2011; Johnson et al., 2005; Zhu et al., 2023). Moreover, the morphological and functional connectivity within the SMC and its links to the FPN and motor networks have been associated with HPT (Zhu et al., 2023). Our findings suggest that dysregulation within these networks may contribute to altered affective and behavioral characteristics of HPT.

This study raises important questions about the role of sensorimotor circuits in the broader neurobiology of mood disorders. Dysfunctional reward processing, episodic thinking, cognitive control, and sensorimotor integration, as observed in dSMC and vSMC activity and their

connectivity with the FPN and SMN, may underlie the emotional and behavioral fluctuations characteristic of bipolar disorder, where individuals alternate between periods of high mood and energy (hypomania or mania) and low mood (depression). Understanding the link between sensorimotor activation and mood volatility could lead to potential biomarkers for identifying individuals at risk for mood disorders and offer new targets for therapeutic interventions stabilizing mood through neuromodulatory techniques, such as transcranial magnetic stimulation.

Parallel neuropsychological pathways linking mood volatility and aggression

In this study, we found that BIS activity, along with functional patterns in the dSMC and vSMC, mediated the association between mood volatility and aggression. Specifically, functional patterns include brain activation and spontaneous neural activity within the dSMC and vSMC, as well as their connectivity with other neural networks. We observed that reward-related activation in the dSMC and time-related activation in the vSMC—alongside their respective connectivity patterns (dSMC with the FPN and vSMC with the SMN)—play key roles in mediating the relationship between mood volatility and aggression via parallel pathways involving BIS.

The reward-related activation in the dSMC, linked with the FPN, suggests a mechanism through which heightened reward sensitivity, often seen in hypomanic states, could intensify mood swings and lead to impulsive, aggressive behaviors. Prior studies have shown that both the SMC and FPN are active in individuals with manic or hypomanic tendencies (Northoff et al., 2021; Zhu et al., 2023). Conversely, the time-related activation in the vSMC and its connectivity with the SMN suggest an alternative pathway: difficulties in episodic thinking and motor responses may contribute to increased aggression via sensorimotor integration. These parallel pathways represent distinct mechanisms by which mood volatility can influence aggression: the dSMC-FPN pathway reflects primarily top-down, reward-driven processes, while the vSMC-SMN pathway may be more related to sensorimotor reactivity and episodic thinking. Additionally, BIS, known for its role in regulating aversive motivation and curbing impulsive behavior (Henderson et al., 2015), appears to interact with both the dSMC and vSMC, modulating the impact of mood volatility on aggression. This study provides the first evidence of neural substrates that support psychological pathways contributing to the link between mood volatility and aggression. The dSMC-FPN connectivity, associated with reward processing, may directly influence BIS function, thereby inhibiting approach behaviors during periods of intense mood volatility. In contrast, the vSMC-SMN pathway may be involved in curbing reactive aggression arising from temporal misalignment in sensorimotor processing. This interaction suggests that BIS dysfunction could lead to ineffective regulation of aggression in response to mood instability, resulting in disinhibited behaviors.

Limitations

Although we identified psychological pathways explaining the link between HPT and aggression, it is important to note that these associations are correlational rather than causal. Future experimental and longitudinal studies will be necessary to draw greater causal inferences regarding these pathways. Second, our sample consisted primarily of university students, which limited the generalizability to the broader population, including clinical populations. Replicating these findings in more more diverse cohorts would enhance the external validity of our conclusions. Third, although we accounted for gender differences in our analysis, the psychological pathways linking mood volatility to externalized aggression may differ across genders (Cui et al., 2023). Future research should aim to independently confirm and elucidate the specific contributions of gender to these associations. If significant gender differences are found, gender-specific interventions should be considered. Fourth, our study did not utilize formal mental health screening tools, such as commonly used depression, anxiety, or psychiatric disorder scales. Incorporating standardized mental health assessments in future studies would better ensure the exclusion of undiagnosed mental health issues, especially in the context of subclinical symptoms or preclinical manifestations. Fifth, although our task-based fMRI analyses were based on a relatively small sample, the reliability and generalization of the findings were supported by rigorous statistical controls and the novel IS-RSA approach. Nonetheless, future studies should validate these findings in larger sample to increased confidence in their generalizability. Lastly, additional replication and validation attempts using other neuroimaging datasets and methods can strengthen our conclusions.

Conclusion

This study systematically investigated the neuropsychological pathways linking HPT and aggression in a large non-clinical sample. Our findings reveal complex interactions between psychological factors (e.g., BIS and internalized emotional aggression) and brain functions, particularly within the SMC and its dorsal and ventral regions. These findings highlight the neuropsychological pathways underlying the associations observed and the intricate interplay between psychological processes and brain functions in shaping dispositional personality traits, such as hypomania.

Data Availability & Transparency Statement

All quantitative data and analysis scripts are available in OSF (https://osf.io/wub5s/).

CRediT authorship contribution statement

Wei Ge: Conceptualization, Methodology, Investigation, Formal analysis, Visualization, Writing – original draft, Writing – review & editing. **Yuanyuan Gao:** Investigation. **Xiang Li:** Investigation. **Jinlian Wang:** Investigation. **Hohjin Im:** Writing – review & editing. **Wenwei Zhu:** Investigation. **Guang Zhao:** Investigation. **Ying Hu:** Investigation. **Pinchun Wang:** Investigation. **Xia Wu:** Investigation. **Qiong Yao:** Investigation. **Xin Niu:** Investigation, Writing – review & editing. **Xiongying Chen:** Investigation. **Qiang Wang:** Conceptualization, Methodology, Writing – original draft, Writing – review & editing, Supervision.

Declaration of competing interest

The authors declare no conflicts of interest.

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Supplementary materials

Supplementary material associated with this article can be found, in

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