Personality Types in Depression and Their Brain Functional Connectivity Basis

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Abstract

This study employed a functional random forest approach, integrating the clustering process with depression diagnosis to identify personality types (combinations of neuroticism and extraversion) within both depression and control groups, and further investigated differences in resting-state functional connectivity across different personality types. Cluster analysis results revealed that the depression group predominantly consisted of individuals with high neuroticism and low extraversion tendencies, but also included individuals with low neuroticism and high extraversion tendencies. The control group samples were predominantly composed of individuals with low neuroticism and high extraversion. Resting-state functional connectivity results showed that, without considering personality subtypes, there were no significant differences between the depression and control groups in functional connectivity of the amygdala/hippocampus/insula-limbic network/default mode network/control network. After incorporating the subtypes derived from cluster analysis into the statistics, multiple personality types exhibited significant differences in functional connectivity strength of the left amygdala/insula-limbic network (primarily in the orbitofrontal cortex region). The depression personality types identified from an individual perspective in this study better reflect real-world conditions and individual cognitive patterns, possess potential clinical application value, and their differences in functional connectivity provide a neural-level reference for understanding depression heterogeneity.

Full Text

Personality Subtypes of Depressive Disorders and Their Functional Connectivity Basis

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Abstract

This study employed a functional random forest approach to integrate clustering with depression diagnosis, identifying personality types (combinations of neuroticism and extraversion) within both depressed and control groups, and further investigated differences in resting-state functional connectivity across these personality types. Clustering analysis revealed that depression was predominantly characterized by individuals with high neuroticism and low extraversion, though individuals with low neuroticism and high extraversion were also present. The control sample was primarily composed of individuals with low neuroticism and high extraversion. Resting-state functional connectivity results showed no significant differences between depressed and control groups in amygdala/hippocampus/insula-limbic/default/control network connectivity when personality subtypes were not considered. However, after incorporating subtypes derived from cluster analysis, significant differences emerged across multiple personality types in the functional connectivity strength of the left amygdala/insula-limbic network (primarily in the orbitofrontal cortex region). The personality types identified from a person-centered perspective in this study align more closely with real-world conditions and individual cognitive patterns, offering potential clinical applicability, while the observed differences in functional connectivity provide a neural-level reference for understanding depression heterogeneity.

Keywords: neuroticism, extraversion, resting-state functional connectivity, depressive disorders, person-centered

Introduction

Heterogeneity in mental health research has garnered significant attention in recent years, with researchers noting that it limits progress in mental health and cognitive neuroscience and calling for a "heterogeneity revolution." Concurrently, person-centered approaches to personality research—emphasizing sample heterogeneity from a typological perspective—have gained increasing attention. Researchers leveraging personality typology have found that different personality types exhibit distinct relationships with mental health problems and treatment outcomes. Meanwhile, heterogeneity among depressed patients has long been a non-negligible issue, as most studies have ignored actual conditions and assumed sample homogeneity. Although numerous empirical studies have focused on isolated personality traits associated with depression, few have considered individual differences from a personality classification perspective.

Contemporary personality typology research originated with Jack Block, and subsequent work by Asendorpf, Robins, and Caspi replicated three personality types (RUO types): resilients, undercontrollers, and overcontrollers. The RUO

types generally correspond to the Five-Factor Model as follows: resilients show low neuroticism and high scores on other dimensions; overcontrollers exhibit high neuroticism and low extraversion; and undercontrollers display low agreeableness and openness. However, the RUO classification has faced extensive statistical challenges, as results obtained using different methods and samples often fail to replicate or identify more than three types, and even studies achieving RUO classification show considerable variation, suggesting limited consensus and replicability.

Empirical studies based on the Five-Factor Model have identified two or five personality types in depressed patients. For instance, Wardenaar and colleagues used latent profile analysis with 146 major depression patients to identify two types: a vulnerable type with high neuroticism, low extraversion, low conscientiousness, and high agreeableness, and a resilient type with moderate neuroticism, moderate extraversion, high conscientiousness, and high agreeableness. Another longitudinal study of depressed patients with acute coronary syndrome using non-hierarchical K-means clustering also found resilient and vulnerable types. Additionally, a study on comorbid depression-anxiety patients defined five personality types through latent class analysis, which could be summarized as two resilient subtypes and three overcontroller subtypes. Overall, research on personality types in depression lacks consistency, though most studies have identified resilient and vulnerable types.

Previous research has primarily employed unsupervised clustering to identify personality subtypes, which is appropriate for exploring possible configurations of all personality traits. However, for investigating personality types associated with specific problems or outcomes (e.g., depression severity, diagnosis, creativity), unsupervised clustering is insufficient. Moreover, from cognitive function to clinical disorders, not all conditions relate to the same personality traits; rather, they may correspond to different combinations of trait inputs. For example, when seeking highly creative individuals, openness should be included as an input feature given its strong association with creativity, whereas agreeableness may be unnecessary. Neuroticism and extraversion have demonstrated moderate to strong predictive validity for depressive symptoms, with conscientiousness showing weak effects and agreeableness and openness demonstrating low replicability. Therefore, this study selected neuroticism and extraversion—the traits most relevant to depression—for subsequent cluster analysis.

We employed the recently developed functional random forest algorithm applied in clinical psychiatry. Unlike previous depression typology studies using unsupervised clustering methods (e.g., K-means, latent class analysis) with predetermined numbers of categories, this method integrates supervised (random forest machine learning) and unsupervised (community detection clustering) approaches, making it more suitable for our research aims.

Research indicates significant differences in brain function between depressed patients and healthy controls, particularly in subcortical regions such as the amygdala, hippocampus, and insula, as well as cortical networks including the

limbic, default mode, and control networks. The amygdala is central to emotion perception and recognition, the hippocampus is involved in episodic memory retrieval, and the insula is associated with attention monitoring, emotion perception, reward systems, and decision-making. These regions are critical for perceiving, transmitting, and integrating emotions, and they work with the limbic, default mode, and control networks to regulate complex emotional and physiological responses, showing abnormal activation or connectivity patterns in depression. Furthermore, these brain functions relate to personality traits; for example, higher emotion susceptibility (a neuroticism subdimension) in depressed patients during negative emotion cognitive reappraisal tasks correlates with weaker connectivity between the dorsolateral prefrontal cortex and amygdala. However, few researchers have explored the neural basis of personality types in depression, leaving the neurobiological mechanisms unclear. Such research could elucidate depression heterogeneity at the neural level.

This study aims to identify different personality types in depression and validate differences across types from a brain functional connectivity perspective, providing evidence for the validity of personality classification and a reference for future heterogeneity research. As described, we selected neuroticism and extraversion—the traits most relevant and replicable for depression—as input features for functional random forest clustering. We then examined whether personality types differed in resting-state functional connectivity of key depression-related subcortical regions and cortical networks (amygdala/hippocampus/insula-limbic/default/control networks). Although depression typology research lacks consistent conclusions, we hypothesized that depression would include two types: a low-risk type with low neuroticism and high extraversion (similar to the resilient type) and a high-risk type with high neuroticism and low extraversion (similar to the vulnerable type). We further hypothesized that personality types would differ in key resting-state functional connectivity measures. Compared to previous studies examining statistical interactions of personality traits that may not exist in real-world contexts, our person-centered approach identifies depression personality types that better align with reality and individual cognitive patterns, offering greater potential clinical utility. Exploring the brain functional connectivity basis of different types not only tests classification validity but also enhances understanding of depression heterogeneity at the neural level, informing future research on depression mechanisms.

2.1 Participants

Depressed patients were recruited from the Psychiatry Department of Chongqing Medical University Affiliated Hospital. Inclusion criteria were: (1) meeting DSM-IV (Axis I disorders) diagnostic criteria for depression; (2) no serious physical illness; (3) no severe neurological disease; (4) no acute or chronic infection, trauma, inflammation, fever, or allergy history in the past two weeks; (5) no alcohol or drug abuse history. Depression severity was assessed

using the 17-item Hamilton Depression Scale (HAMD), with scores below 7 indicating no depressive symptoms. A total of 159 patients were selected (mean age = 38.9 years, SD = 13.3 years; 98 females). All patients had HAMD scores ≥ 7.

Control participants were recruited from universities and surrounding communities. Inclusion criteria were: (1) no depressive episodes or history; (2) no other psychiatric history; (3) no first-degree relatives with psychiatric disorders; (4) no severe physical or neurological disease history; (5) no acute or chronic infection, trauma, inflammation, fever, or allergy history in the past two weeks; (6) no alcohol or drug abuse history. A total of 156 controls were selected (mean age = 41.7 years, SD = 15.9 years; 102 females). All controls had HAMD scores < 7. Given that high scores on the lie scale of the Eysenck Personality Questionnaire indicate response distortion, all participants scored ≤ 18 on this dimension. All participants were informed about the study's purpose, procedures, and potential risks, and provided written informed consent. No significant differences existed between groups in age, gender, or education duration. Detailed demographic information is presented in Table 1 .

Table 1 Basic demographic information

Group	Mean Age	SD	Range	Statistical Value
Depression	38.9	13.3	18-71	$t(313) = 1.68$, $p = 0.094$
Control	41.7	15.9	19-70	$^2(1) = 0.478$, $p = 0.490$
				$t(313) = 1.23$, $p = 0.22$
				$t(313) = 44.08$, $p < 0.001$

Note: HAMD = Hamilton Depression Scale.

2.2.1 Psychometric Assessment

Personality traits were assessed using the Chinese version of the Adult Eysenck Personality Questionnaire. Originally developed for clinical psychiatric assessment and widely used in hospital psychiatry and psychology departments, the EPQ comprises four dimensions: neuroticism, extraversion, psychoticism, and lie. High neuroticism scores reflect a tendency to experience negative emotions, emotional lability, overreaction, and slow recovery from emotional experiences. High extraversion scores reflect being outgoing, talkative, impulsive, uninhibited, socially engaged, and experiencing high positive affect. Psychoticism relates to aggression, antisocial behavior, and impulsivity, while the lie scale reflects response distortion tendency. This questionnaire covers our traits of interest and enjoys widespread use across countries and regions.

2.2.2 Resting-State Data Acquisition and Preprocessing

Resting-state fMRI data were acquired on a 3.0T Siemens Trio MRI scanner. During scanning, participants were instructed to lie still with eyes closed, avoid thinking or recalling specific events, and remain awake. Whole-brain gradient-echo planar imaging was used with the following parameters: echo time = 30 ms; repetition time = 2000 ms; flip angle = 90°; matrix size = 64 × 64; field of view = 192 × 192 mm; slices = 32; thickness = 3 mm; gap = 1 mm; voxel size = 3.4 × 3.4 × 4 mm^3. Scan duration was 8 minutes 4 seconds.

Preprocessing was conducted using DPARSF (Yan & Zang, 2010) (http://restfmri.net). After quality control inspection and removal of poor-quality data, the following steps were performed: removal of the first 10 time points (due to initial signal instability), slice timing correction, head motion correction, spatial normalization to MNI template with resampling to 3 × 3 × 3 mm^3, spatial smoothing (8 mm kernel), regression of white matter and cerebrospinal fluid signals plus Friston' s 24 motion parameters, band-pass filtering (0.01–0.1 Hz), and additional head motion reduction using DPARSF' s scrubbing function. This pipeline is consistent with published studies.

2.3.1 Clustering Analysis

The recently developed functional random forest algorithm for clinical psychiatry was used to identify personality subtypes in depressed patients. This hybrid method integrates supervised (top-down hypothesis-driven) and unsupervised (bottom-up data-driven) approaches, linking clinical diagnosis with heterogeneity and overcoming the limitation of unsupervised clustering producing categories unrelated to the research question.

First, neuroticism and extraversion (input features) were fitted to depression diagnosis using a random forest model with random seed 1234, 500 decision trees, and tenfold cross-validation for performance assessment. In each decision tree, internal nodes represent predictors (input features), connections represent decision rules, and terminal nodes represent outcomes (depression diagnosis). The random forest analysis used the CORElearn package in RStudio, implementing Breiman' s algorithm combining bagging and CART decision trees. The process involved: (1) bootstrap sampling with replacement of n training samples, (2) building CART models for each sample, (3) repeating 500 times to create the forest, and (4) majority voting for final predictions. Detailed formulas are available in Breiman (2001). The similarity between any two participants was defined as the proportion of trees in which they were assigned to the same terminal node, represented in a proximity matrix (Figure 1 [Figure 1: see original paper]) where rows and columns represent participants and cells indicate co-assignment probabilities.

Second, the ExplainPrediction package in RStudio was used to interpret the model, generating evaluation metrics including accuracy, specificity, and sensitivity from confusion matrices. Community detection clustering analysis was

then applied to the proximity matrices for depressed and control groups separately. Community detection, a graph-theoretic method for iterative subgroup identification, was implemented using the Generalized Louvain Method (Lucas et al., 2020), which optimizes modularity Q (Blondel et al., 2008) through iterative refinement to achieve maximal modularity. Given the algorithm's stochastic nature involving the Newman–Girvan null model, 100 runs were performed per group, yielding 100 Q values. The averaged Q served as a quality index for community separation strength. The GenLouvain toolbox was used with gamma and omega parameters set to 1, following previous research.

Figure 1 Clustering process

Note: Random forest: Internal nodes in a single decision tree represent independent variables (input features), connections represent decision rules, and terminal nodes represent outcomes (predicted variables). Proximity matrix: Rows and columns represent participants; each cell indicates the probability of paired participants being assigned to the same terminal node across all trees.

2.3.1 Resting-State Functional Connectivity Analysis

To examine differences in functional connectivity strength between the amygdala, hippocampus, and insula with the limbic, default mode, and control networks across classifications, and to further validate classification validity, we conducted functional connectivity analysis. First, regions of interest (ROIs) were defined using the 400-node Schaefer-Yeo brain functional template corresponding to 17 functional networks, which offers high specificity and network homogeneity. Although this template excludes subcortical regions, it provides detailed network parcellation, including two limbic networks (LimbicA_{TempPole}, LimbicB_{OFC}), three control networks (ContA_{Cingm}, ContB_{PFCmp}, ContC_{Cingp}), and three default networks (DefaultA_{PFCm}, DefaultB_{PFCv}, DefaultC_{PHC}). All nodes from these eight networks were selected as ROIs (detailed node information in Appendix Table S3). Second, bilateral amygdala, hippocampus, and insula were defined as six subcortical ROIs using the AAL atlas. Pearson correlations were computed between each subcortical ROI and all network nodes, then aggregated to calculate functional connectivity strength between each subcortical ROI and the eight networks. Finally, one-way ANOVAs were performed on connectivity strength across groups derived from cluster analysis, with gender, age, and education as covariates. FDR correction was applied for multiple comparisons across six subcortical regions, yielding a threshold of 0.0083 (0.05/6). Post-hoc tests were conducted for connectivity surviving correction.

Additionally, independent samples t-tests were performed between depressed and control groups on connectivity between the six subcortical ROIs and eight networks to examine differences without considering subtypes. Correlations between neuroticism/extraversion and functional connectivity were also examined,

with FDR correction at 0.0083.

3.1 Clustering Results

Tenfold cross-validation showed the random forest model achieved average accuracy of 77.50%, specificity of 74.96%, and sensitivity of 80.04%, indicating good model fit and adequate discrimination between groups. Community detection applied to the proximity matrix revealed four personality types in depressed patients (average Q = 0.19). Type 1 (n = 41, 25.79%) showed low neuroticism and moderately high extraversion; Types 2 and 4 (n = 44, 27.67% and n = 32, 20.13%) showed high neuroticism and low extraversion; Type 3 (n = 42, 26.42%) showed high neuroticism and moderately high extraversion (Figure 2 [Figure 2: see original paper] and Figure S1). Types were relatively balanced, with high-neuroticism/low-extraversion types being most prevalent.

Control participants yielded five personality types (average Q = 0.31). Type 1 (n = 47, 30.13%) and Type 3 (n = 44, 28.21%) showed low neuroticism with high or moderately high extraversion, consistent with the resilient profile. Type 2 (n = 39, 25.00%) showed moderately low neuroticism and extraversion, similar to an average type. Type 4 (n = 15, 9.62%) showed moderate neuroticism and high extraversion, while Type 5 (n = 11, 7.05%) showed high neuroticism and moderate extraversion (Figure 2 and Figure S1). Types 4 and 5 were less common, while Types 1–3 were more prevalent and balanced. Overall, depression was dominated by high-neuroticism/low-extraversion individuals, but also included low-neuroticism/high-extraversion individuals, whereas controls were primarily low-neuroticism/high-extraversion.

Independent samples t-tests confirmed depressed patients had significantly higher neuroticism (Mean difference = 7.77, $t(313) = 13.98$, $p < 0.001$, Cohen' s d = 1.575) and lower extraversion (Mean difference = -4.09, $t(313) = -7.94$, $p < 0.001$, Cohen' s d = -0.895) than controls. One-way ANOVAs across the nine groups (with covariates) revealed significant group differences in neuroticism ($F(8, 303) = 131.62$, $p < 0.001$, $^2 = 0.771$) and extraversion ($F(8, 303) = 51.79$, $p < 0.001$, $^2 = 0.575$). Detailed post-hoc results are in Appendix Table S1.

Figure 2 Z-scores of neuroticism and extraversion for personality types in depression and control groups

Note: Depression Type 1: low neuroticism, moderately high extraversion; Types 2 and 4: high neuroticism, low extraversion; Type 3: high neuroticism, moderately high extraversion. Control Type 1 and 3: low neuroticism, high or moderately high extraversion; Type 2: moderately low neuroticism and extraversion; Type 4: moderate neuroticism, high extraversion; Type 5: high neuroticism, moderate extraversion. N = neuroticism; E = extraversion; CON = control group; DD = depressive disorder; DD1 = Depression Type 1; CON1 = Control Type 1.

3.2 Resting-State Functional Connectivity Results

After preprocessing, usable data were available for 125 depressed participants (Type 1: n = 32; Type 2: n = 32; Type 3: n = 33; Type 4: n = 28) and 122 controls (Type 1: n = 34; Type 2: n = 28; Type 3: n = 37; Type 4: n = 12; Type 5: n = 11). Control Types 4 and 5 were excluded from subsequent analyses due to small sample sizes.

One-way ANOVAs across the seven remaining types (with covariates) revealed significant group differences after FDR correction (threshold = 0.05/6) in left amygdala-limbic network connectivity (LimbicB_{OFC}, F(6, 214) = 4.273, p = 0.0004) and left insula-limbic network connectivity (LimbicB_{OFC}, F(6, 214) = 4.177, p = 0.0005). No significant differences were found for connectivity involving the hippocampus or default/control networks.

Post-hoc tests (Holm-corrected) for left amygdala-limbic network (LimbicB_{OFC}) showed significantly weaker connectivity in Depression Type 1 (t = -3.47, df = 214, p = 0.013, Cohen' s d = -0.977) and Type 2 (t = -3.34, df = 214, p = 0.018, Cohen' s d = -0.851) compared to Control Type 3. Control Type 2 showed weaker connectivity than Control Type 1 (t = -3.38, df = 214, p = 0.016, Cohen' s d = -0.893) and Control Type 3 (t = -3.49, df = 214, p = 0.013, Cohen' s d = -1.180) (Table 2 and Figure 3 [Figure 3: see original paper]).

Post-hoc tests for left insula-limbic network (LimbicB_{OFC}) revealed significantly weaker connectivity in Depression Type 2 compared to Control Type 3 (t = -4.06, df = 214, p = 0.001, Cohen' s d = -1.034). Depression Type 3 showed marginally stronger connectivity than Type 2 (t = 3.04, df = 214, p = 0.053, Cohen' s d = 0.757), and Control Type 2 differed marginally from Type 3 (t = -2.94, df = 214, p = 0.070, Cohen' s d = -0.995) (Table 2 and Figure 4 [Figure 4: see original paper]). All significant connectivity differences showed large effect sizes (|d| > 0.8).

Independent samples t-tests between depressed and control groups (without subtype consideration) revealed no significant connectivity differences after FDR correction. Correlation analyses showed extraversion was significantly correlated with left/right insula-limbic (LimbicB_{OFC}) and left/right insula-default network (DefaultA_{PFCm}, DefaultB_{PFCv}) connectivity (details in Appendix Table S2).

Table 2 Post-hoc test results for functional connectivity strength of left amygdala/insula-limbic network

Comparison	Left Amygdala-Limbic Network	Left Insula-Limbic Network
	p_{holm}	Cohen' s d
DD1-CON2		
DD1-CON1		
DD1-CON3		

Comparison	Left Amygdala-Limbic Network	Left Insula-Limbic Network
DD2-CON2		
DD2-CON1		
DD2-CON3		
DD3-CON2		
DD3-CON1		
DD3-CON3		
DD4-CON2		
DD4-CON1		
DD4-CON3		

Note: CON = control group; DD = depressive disorder; DD1 = Depression Type 1; CON1 = Control Type 1.

Figure 3 Group differences in functional connectivity strength between left amygdala and limbic network

Note: CON = control group; DD = depressive disorder; DD1 = Depression Type 1; CON1 = Control Type 1.

Figure 4 Group differences in functional connectivity strength between left insula and limbic network

Discussion

This study adopted a person-centered perspective using functional random forest to link clustering with depression diagnosis, identifying personality types in both depressed and control groups and exploring their resting-state functional connectivity basis. Clustering results showed that depression was dominated by high-neuroticism/low-extraversion individuals but also included low-neuroticism/high-extraversion individuals, while controls were primarily low-neuroticism/high-extraversion. Resting-state functional connectivity revealed no significant differences between depressed and control groups when personality subtypes were ignored, but significant differences emerged across multiple personality types in left amygdala/insula-limbic network (LimbicB_{OFC}) connectivity strength.

Personality traits are closely linked to depression vulnerability, particularly neuroticism. Higher neuroticism is associated with poorer emotion regulation, weaker psychological resilience, increased negative thinking and life stress, and greater depression severity. Extraversion also relates to emotion regulation and resilience, influencing its association with depression. Numerous studies have confirmed that high neuroticism and low extraversion represent "risk factors" for depression, with evidence for their interaction effects. Our identified depression personality types, dominated by high neuroticism/low extraversion with

controls showing the opposite pattern, reflect this high-risk profile. The low-neuroticism/high-extraversion depression type aligns with Kim et al.' s resilient type, suggesting better treatment outcomes. Control group types generally showed at least one trait at moderate/low risk, reflecting lower depression vulnerability in healthy populations.

The limbic network' s primary nodes were located in the orbitofrontal cortex (OFC), anatomically consistent with the ventromedial prefrontal cortex. The amygdala is central to emotion perception and physiological arousal, while OFC is core to emotion processing (integration, evaluation, reward, decision-making) and top-down regulation of amygdala activity. Amygdala-OFC connectivity is implicated in emotion processing, fear extinction, and inhibiting amygdala overactivation to negative stimuli. Neuroticism positively correlates with amygdala activation but negatively with OFC activation during emotional tasks, while extraversion positively correlates with amygdala activation during positive emotion processing and with both amygdala and OFC activation during reward tasks. Low neuroticism/high extraversion may thus relate to stronger amygdala-OFC connectivity (higher activation synchrony) and better regulatory control, consistent with our findings. For instance, Control Type 2 (moderate neuroticism/extraversion) showed weaker left amygdala-limbic network (LimbicB_{OFC}) connectivity than Control Types 1 and 3 (low neuroticism, high extraversion). Additionally, more severe depressive symptoms correlate with weaker OFC activation and amygdala-OFC connectivity at rest, supporting our finding that even low-neuroticism/high-extraversion depressed patients (Type 1) showed weaker connectivity than matched controls (Type 3). Depression Type 2 also showed weaker connectivity than Control Type 3, reflecting either opposite neuroticism/extraversion profiles or sample differences (depression status).

The insula is involved in attention monitoring, emotion perception, reward systems, and decision-making. Higher neuroticism correlates with stronger insula activation during emotional tasks, and depressed patients show greater insula activation than controls during negative stimuli. OFC activation during emotion processing negatively correlates with neuroticism and depression severity. Insula-OFC functional coordination may negatively correlate with neuroticism and positively correlate with extraversion. Our results support this: Depression Type 2 (high neuroticism/low extraversion) showed weaker insula-OFC connectivity than Control Type 3 (low neuroticism/high extraversion) and marginally weaker than Depression Type 3 (high neuroticism/high extraversion); Control Type 2 (moderate neuroticism/extraversion) was marginally weaker than Control Type 3. These differences likely reflect varying neuroticism/extraversion combinations or sample differences.

Our findings of connectivity differences only after incorporating personality subtypes support classification validity and enhance understanding of depression heterogeneity at the neural level, informing future research on depression mechanisms and targeted interventions. However, three limitations should be noted. First, our clustering was primarily data-driven; future research should address

more substantively meaningful scientific questions to enhance innovation. Second, our imaging analysis was limited to seed-based functional connectivity; future studies could employ whole-brain analyses combining multiple structural and functional metrics to deepen understanding of neural bases. Third, we did not consider other symptoms (e.g., anxiety) or comorbidities (e.g., anxiety disorders) that may influence personality classification and brain function; future research should examine these factors.

In conclusion, this study linked clustering with depression diagnosis to identify personality types and explored their resting-state functional connectivity differences, advancing understanding of depression heterogeneity. Depression was dominated by high-neuroticism/low-extraversion types but also included low-neuroticism/high-extraversion types, while controls were primarily low-neuroticism/high-extraversion. Multiple personality types showed significant differences in left amygdala/insula-limbic network connectivity: high-neuroticism/low-extraversion types showed weaker connectivity, and even low-neuroticism/high-extraversion depressed patients showed weaker connectivity than matched controls.

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Figure S1 Three-dimensional scatter plot of neuroticism, extraversion, and Hamilton scores for depression and control groups

Note: CON = control group; DD = depressive disorder; DD1 = Depression Type 1; CON1 = Control Type 1.

Table S1 Group differences in neuroticism and extraversion across personality types

Comparison	Neuroticism	Extraversion
	p_{holm}	Cohen' s d
DD1-DD2	<.001	
DD1-DD3	<.001	
DD1-DD4	<.001	
DD2-DD3	<.001	
DD2-DD4	<.001	
DD3-DD4	<.001	
DD1-CON1	<.001	
DD1-CON2	<.001	
DD1-CON3	<.001	
DD1-CON4	<.001	
DD1-CON5	<.001	
DD2-CON1	<.001	
DD2-CON2	<.001	
DD2-CON3	<.001	
DD2-CON4	<.001	
DD2-CON5	<.001	
DD3-CON1	<.001	
DD3-CON2	<.001	
DD3-CON3	<.001	
DD3-CON4	<.001	
DD3-CON5	<.001	
DD4-CON1	<.001	
DD4-CON2	<.001	
DD4-CON3	<.001	
DD4-CON4	<.001	
DD4-CON5	<.001	

Note: CON = control group; DD = depressive disorder; DD1 = Depression Type 1; CON1 = Control Type 1.

Table S2 Correlation matrix between extraversion and functional connectivity

Region-Network	Pearson r	95% CI	p-value
Left insula-LimbicB			<.001
Left insula-DMNA			<.001
Left insula-DMNB			<.001
Right insula-LimbicB			<.001
Right insula-DMNA			<.001
Right insula-DMNB			<.001

Note: LimbicB = LimbicB_{OFC}; DMNA = DefaultA_{PFCm}; DMNB = DefaultB_{PFCv}.

Table S3 Node information for eight networks

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Abbreviations: - AntTemp: anterior temporal - Cingm: mid-cingulate - Cingp: posterior cingulate - IPL: inferior parietal lobule - IPS: intraparietal sulcus - OFC: orbital frontal cortex - pCun: precuneus - pCunPCC: precuneus posterior cingulate cortex - PFCd: dorsal prefrontal cortex - PFCl: lateral prefrontal cortex - PFClv: lateral ventral prefrontal cortex - PFCm: medial prefrontal cortex - PFCmp: medial posterior prefrontal cortex - PFCv: ventral prefrontal cortex - PHC: parahippocampal cortex - Rsp: retrosplenial - Temp: temporal - TempPole: temporal pole