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CORTICO-STRIATAL NETWORKS AND COGNITIVE DEFICIT STRUCTURE IN ENDOGENOUS DISORDERS WITH CATATONIC SYMPTOMATOLOGY: A FACTOR-ANALYTIC STUDY OF NEUROPSYCHOLOGICAL PROFILES

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RESUME

Background. *Contemporary neuroimaging studies reveal that catatonia is associated with specific dysfunction patterns in cortico-striatal-thalamic networks, with particular involvement of fronto-striatal circuits regulating motor control and executive functions. Factor-analytic approaches to cognitive assessment can identify distinct neuropsychological profiles reflecting underlying neural network alterations in catatonic disorders.*
Objective. *To characterize the structure of neurocognitive deficits in endogenous disorders with catatonic symptomatology through factor analysis, identifying distinct cognitive profiles associated with cortico-striatal and temporo-parietal network dysfunction, and to evaluate the modifying effect of affective comorbidity on these patterns.*
Material and Methods. *The study included 139 patients: main group (n = 69) with endogenous disorders including catatonic symptomatology, divided into subgroups without (n = 35) and with pronounced depressive symptomatology (n = 34), and control group (n = 70) with similar subdivision. Catatonia was assessed using Bush-Francis Scale (BFCRS). Neurocognitive evaluation included BACS battery, MoCA and FAB scales. Factor analysis with varimax rotation was applied to identify cognitive deficit structure. Statistical analysis included MANOVA with Bonferroni correction.*
Results. *Factor analysis revealed distinct cognitive structures. In catatonia without affective symptomatology, three factors emerged: (1) a fronto-striatal factor (47.2 % of variance), comprising executive functions, psychomotor speed, and inhibitory control; (2) a temporo-parietal factor (26.3 % of variance), including visuospatial functions and constructive praxis; and (3) a hippocampal factor (19.8 % of variance), associated with episodic memory and delayed recall. In the presence of affective comorbidity, two factors were identified: (1) a fronto-limbic factor, accounting for 53.7% of the variance and encompassing attention, working memory, and emotional regulation; and (2) a fronto-striatal factor, accounting for 30.1 % of the variance and encompassing psychomotor speed and executive functions. MANOVA revealed significant group differences across all cognitive domains ($d = 0.52–1.31$, $p < 0.001$). Two-factor analysis showed main effect of catatonia ($F = 31.2$, $p < 0.001$, $\eta^2 = 0.19$), moderate effect of affectivity ($F = 12.4$, $p < 0.01$, $\eta^2 = 0.08$) and their interaction ($F = 7.8$, $p < 0.01$, $\eta^2 = 0.05$).*
Conclusion. *Catatonic disorders demonstrate distinct cognitive profiles on factor analysis, reflecting specific cortico-striatal and temporo-parietal network dysfunction. Affective comorbidity transforms the cognitive structure toward fronto-limbic involvement, indicating the need for differentiated rehabilitation targeting specific neural networks.*
Keywords: *catatonia, cortico-striatal networks, cognitive factor analysis, neuropsychological profiles, fronto-limbic dysfunction*

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КОРТИКО-СТРИАРНАЯ СИСТЕМА И СТРУКТУРА КОГНИТИВНОГО ДЕФИЦИТА ПРИ ЭНДОГЕННЫХ РАССТРОЙСТВАХ С КАТАТОНИЧЕСКОЙ СИМПТОМАТИКОЙ: ФАКТОРНО-АНАЛИТИЧЕСКОЕ ИССЛЕДОВАНИЕ НЕЙРОПСИХОЛОГИЧЕСКИХ ПРОФИЛЕЙ

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РЕЗЮМЕ

Обоснование. *Современные нейровизуализационные исследования показывают, что кататония связана со специфическими формами дисфункции кортико-стриатально-таламических систем, с особым вовлечением фронто-стриатальных структур, регулирующих двигательный контроль и исполнительные функции.*

Цель. *Охарактеризовать структуру нейрокогнитивного дефицита при эндогенных расстройствах с кататонической симптоматикой с помощью факторного анализа, выявив различные когнитивные профили, связанные с дисфункцией кортико-стриарной и височно-теменной систем, и оценить модифицирующее влияние сопутствующей аффективной (преимущественно депрессивной) патологии.*

Материалы и методы. *Исследованы 139 пациентов: основная группа (n = 69) с эндогенными расстройствами, включая кататоническую симптоматику, разделенная на подгруппы без (n = 35) и с выраженной депрессивной симптоматикой (n = 34), и контрольная группа (n = 70). Кататонию оценивали с помощью шкалы Буша–Фрэнсиса (BFCRS). Нейрокогнитивная оценка включала шкалы BACS, MoCA и FAB. Применен факторный анализ с чередованием переменных.*

Результаты. *Факторный анализ выявил различные когнитивные структуры. При кататонии без аффективной симптоматики выявлялись три фактора: фронто-стриатный (47,2 % дисперсии), темпоро-париетальный (26,3 %) и гиппокампальный (19,8 %). При сопутствующей аффективной патологии – два фактора: фронто-лимбический (53,7 %) и фронто-стриатный (30,1 %). MANOVA выявил значимые групповые различия во всех когнитивных областях (d = 0,52–1,31, p < 0,001).*

Заключение. *Кататонические расстройства демонстрируют различные факторно-аналитические когнитивные профили, отражающие специфическую дисфункцию кортико-стриарных и темпоро-париетальных систем. Сопутствующая аффективная патология трансформирует структуру когнитивных нарушений в сторону фронто-лимбической дисфункции, что указывает на необходимость дифференцированной реабилитации.*

Ключевые слова: *кататония, кортико-стриарная система, когнитивный факторный анализ, нейропсихологические профили, фронто-лимбическая дисфункция*

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BACKGROUND

Catatonia represents a complex neuropsychiatric syndrome characterized by psychomotor, behavioral, and affective disturbances that reflect specific patterns of neural network dysfunction [1]. Recent neuroimaging studies have consistently demonstrated that catatonia is associated with alterations in cortico-striatal-thalamic loop systems, with particular involvement of connections between the thalamus and primary motor cortex [2, 3]. The salience network, which includes cortico-striatal-thalamic circuits, plays a central role in cognitive control by integrating sensory input and recruiting appropriate functional networks to modulate behavior [4].

Contemporary neurobiological models emphasize that catatonic manifestations arise from dysfunction in specific neural circuits rather than representing a unitary disorder [5, 6]. Neuropsychological investigations have identified that catatonia involves alterations in fronto-parietal cortical function, affecting attention, executive, visual-spatial, and working memory domains [7, 8]. However, the structure of cognitive deficits in catatonia remains insufficiently characterized, particularly regarding how different neural network dysfunctions contribute to distinct cognitive profiles [9, 10].

Factor-analytic approaches to neuropsychological assessment offer a powerful method for identifying latent cognitive structures that may reflect underlying neural network alterations [7]. By examining patterns of covariation among cognitive measures, factor analysis can reveal distinct cognitive profiles associated with specific brain circuits [8]. This approach is particularly relevant for catatonia, where heterogeneous clinical presentations may reflect different patterns of network dysfunction [9, 11].

Our theoretical framework is based on Luria's theory of functional brain systems, which conceptualizes cognitive functions as complex functional systems comprising multiple brain regions working in concert. This approach, combined with contemporary network neuroscience models, allows us to understand catatonic cognitive dysfunction as disruption of hierarchically organized functional systems. Specifically, we propose that catatonia represents a multilevel dysfunction affecting: (1) subcortical motor regulation (basal ganglia-thalamic loops), (2) cortical executive control (prefrontal systems), and (3) integrative functions (parietal association areas). This theoretical synthesis enriches our understanding by bridging classical neuropsychology with modern network approaches.

The role of affective symptomatology in modifying cognitive profiles represents another critical consideration [12]. Recent precision functional mapping studies have shown that fronto-striatal salience networks occupy expanded cortical territory in depression, suggesting that affective disorders may fundamentally alter the neural substrates underlying cognitive function [8]. In catatonic disorders, the interaction between motor, executive, and affective networks may produce distinct cognitive profiles that require specific therapeutic approaches [13, 14].

Understanding the factor structure of cognitive deficits in catatonia has important implications for both theoretical models of the disorder and clinical practice [14]. By identifying distinct cognitive profiles associated with specific neural networks, clinicians can develop targeted rehabilitation strategies that address the underlying neurobiological dysfunction rather than treating symptoms in isolation [15, 12].

From a neuropsychological perspective, Luria's theory of functional brain systems provides a foundational framework for understanding catatonic cognitive dysfunction. The third functional block of the brain, comprising the frontal lobes, addresses the formation of intention, programming, regulation, and control of behavior. This conceptualization is particularly relevant for catatonia, where disruption of motor programming and executive control represents core features of the syndrome [14].

Classical neuropsychological assessments, such as the Frontal Assessment Battery incorporating Luria's motor sequences, have demonstrated specific patterns of impairment in catatonic patients. The three-step Luria test reveals regulatory dysfunction of facilitating and inhibiting responses in frontal lobe pathology, with abnormal performance occurring in up to 69.8 % of patients with frontotemporal features compared to controls [13].

Combined neuropsychological and neuroimaging studies have further elucidated these patterns. Northoff G. et al. demonstrated right lower prefronto-parietal cortical dysfunction in akinetic catatonia, with poorer performance in visuospatial abilities associated with right parietal function. These findings bridge classical neuropsychological observations with modern network neuroscience, suggesting that catatonic cognitive dysfunction reflects systematic disruption of hierarchically organized functional systems [4].

STUDY OBJECTIVE

To characterize the factor structure of neurocognitive deficits in endogenous disorders with catatonic symptomatology, identifying distinct cognitive profiles reflecting cortico-striatal, temporo-parietal, and fronto-limbic network dysfunction, and to evaluate how affective comorbidity modifies these cognitive structures compared to controls without catatonic symptomatology.

MATERIAL AND METHODS

Study Participants

The study included 139 patients who underwent inpatient treatment in general psychiatric departments No. 12 and No. 11 of Irkutsk regional neuropsychiatric dispensary and No. 2 and No. 6 of Irkutsk Regional Clinical Psychiatric Hospital No. 1 from April 2023 to December 2024. All requirements of the Helsinki Declaration were observed.

The study was approved by Ethics Committee of Irkutsk State Medical University, protocol No. 2 dated April 17, 2023.

The main group consisted of 69 patients with various endogenous disorders including catatonic symptomatology, divided into two subgroups:

Group 1 – without pronounced affective symptomatology (*n* = 35):

- F20.0 Paranoid schizophrenia *n* = 27 (77.1 %)
- F20.1 Hebephrenic schizophrenia *n* = 2 (5.7 %)
- F20.2 Catatonic schizophrenia *n* = 6 (17.1 %)
- Gender: males 16 (45.7 %); females 19 (54.3 %)
- Mean age: 30.8 ± 8.9 years

Group 2 – with pronounced affective (predominantly depressive) symptomatology (*n* = 34):

- F23.2 Acute polymorphic schizophreniform disorder *n* = 15 (44.1 %)
- F25 Schizoaffective disorder *n* = 13 (38.2 %)
- F32.3 Severe depressive episode with psychotic symptoms *n* = 3 (8.8 %)
- F33.3 Recurrent depressive disorder, current severe episode with psychotic symptoms *n* = 3 (8.8 %)
- Gender: males 9 (26.5 %); females 25 (73.5 %)
- Mean age: 32.1 ± 8.4 years

The control group (*n* = 70) included patients with endogenous disorders without catatonic symptomatology, similarly divided into subgroups with and without pronounced affective symptomatology.

Inclusion and Exclusion Criteria

Inclusion Criteria:

- Age 18–55 years
- Established diagnosis according to ICD-10
- Stabilization of acute psychotic symptomatology (PANSS reduction > 20 % in 48 hours)
- Ability to participate in neuropsychological testing

Exclusion Criteria:

- Organic CNS lesions
- Substance abuse (based on history and toxicological screening)
- Severe somatic diseases
- Mental retardation (IQ < 70 based on history)
- Research Methods

Catatonia Assessment: The Bush-Francis Catatonia Rating Scale (BFCRS) was used for diagnosis and severity assessment [16]. All patients in the main group had confirmed catatonic symptomatology with mean BFCRS score of 8.4 ± 4.2 (range 3–18 points).

Neurocognitive Assessment: Assessment was conducted 48–72 hours after stabilization using:

1. BACS (Brief Assessment of Cognition in Schizophrenia) – 6 subtests covering verbal memory, working memory, motor functions, verbal fluency, attention/processing speed, executive functions [17];
2. MoCA (Montreal Cognitive Assessment) – visual-spatial functions, naming, attention, speech, abstraction, delayed recall, orientation [18];
3. FAB (Frontal Assessment Battery) – conceptualization, mental flexibility, programming, interference sensitivity, inhibition, behavioral autonomy [19].

Functional Assessment: Personal and Social Performance Scale (PSP) evaluated functioning across four domains: socially useful activity, personal/social relationships, self-care, disturbing/aggressive behavior [20].

Statistical Analysis

Statistical processing used SPSS 28.0. Factor analysis with principal components method and varimax rotation was performed to identify cognitive deficit structure [21]. Kaiser–Meyer–Olkin (KMO) adequacy and Bartlett's sphericity test confirmed suitability for factor analysis [22]. Multivariate analysis of variance (MANOVA) compared groups, with Bonferroni correction for multiple comparisons ($\alpha = 0.05/9 = 0.006$). Effect sizes were assessed using partial η^2 [23].

RESULTS

Factor Structure of Cognitive Deficits

Factor analysis with varimax rotation revealed distinct cognitive structures across groups (KMO = 0.82, Bartlett's test $p < 0.001$), providing clear evidence for different patterns of neural network dysfunction [24].

Main Group without Affective Symptomatology

Three distinct factors emerged, explaining 93.3 % of total variance:

Factor 1: Fronto-striatal network (eigenvalue = 3.8, 47.2 % variance)

- Executive functions (loading = 0.84)
- Psychomotor speed (loading = 0.81)
- Inhibitory control (loading = 0.78)
- Motor programming (loading = 0.76)

This factor reflects dysfunction in cortico-striatal circuits responsible for motor control and executive regulation, consistent with the cardinal features of catatonic psychomotor disturbance [25].

Factor 2: Temporo-parietal network (eigenvalue = 2.1, 26.3 % variance)

- Visual-spatial functions (loading = 0.79)
- Constructive praxis (loading = 0.74)
- Spatial working memory (loading = 0.71)
- Perceptual organization (loading = 0.68)

This factor indicates involvement of temporo-parietal areas in visual-spatial processing and constructive abilities, suggesting broader cortical dysfunction beyond motor systems [26].

Factor 3: Hippocampal-medial temporal network (eigenvalue = 1.6, 19.8 % variance)

- Episodic memory (loading = 0.77)
- Delayed recall (loading = 0.73)
- Verbal learning (loading = 0.69)
- Memory consolidation (loading = 0.65)

This factor reflects dysfunction in medial temporal lobe structures critical for memory formation and consolidation [27].

From a neuropsychological perspective, the observed factor structure aligns with Luria's three functional units of the brain. The fronto-striatal factor corresponds to the third unit (programming and control),

the temporo-parietal factor to the second unit (receiving and processing information), with additional involvement of occipito-parietal regions for spatial synthesis. The hippocampal factor represents the memory consolidation system, operating across all three units.

The syndrome analysis reveals that catatonia disrupts the dynamic localization of higher mental functions, with primary deficits in the efferent (motor output) and regulatory systems, and secondary effects on afferent (sensory input) and mnestic processes.

Main Group with Affective Symptomatology

Two primary factors emerged, explaining 83.8 % of total variance:

Factor 1: Fronto-limbic network (eigenvalue = 4.3, 53.7 % variance)

- Attention regulation (loading = 0.85)
- Working memory (loading = 0.82)
- Emotional control (loading = 0.79)
- Cognitive flexibility (loading = 0.76)
- Processing speed (loading = 0.71)

This expanded factor reflects integration of cognitive control with emotional regulation, suggesting involvement of fronto-limbic circuits when affective symptoms are prominent [28, 29].

Factor 2: Fronto-striatal motor network (eigenvalue = 2.4, 30.1 % variance)

- Psychomotor speed (loading = 0.83)
- Executive functions (loading = 0.80)
- Motor inhibition (loading = 0.75)
- Sequential processing (loading = 0.70)

This factor maintains the core fronto-striatal dysfunction pattern but with reduced variance contribution compared to the non-affective group [30].

Control Group

Two factors emerged, explaining 85.0 % of total variance:

Factor 1: General cognitive factor (eigenvalue = 4.1, 51.2 % variance)

- Uniform loading across all cognitive domains (0.65–0.75)
- Represents non-specific cognitive decline

Factor 2: Executive factor (eigenvalue = 2.7, 33.8 % variance)

- Selective impairment of frontal functions
- Working memory and attention predominance

Comparative Analysis of Cognitive Profiles

Multivariate analysis (MANOVA) confirmed significant differences between groups (Wilks' $\lambda = 0.61$, $F = 5.34$, $p < 0.001$, $\eta^2 = 0.39$) [23].

As shown in Table 1, significant group differences were observed across all cognitive domains, with the most pronounced deficits in the catatonic groups.

Neural Network-Specific Impairment Patterns

Fronto-striatal network dysfunction

Both catatonic subgroups showed severe impairments in this network, but with different patterns:

- Without affectivity: primary psychomotor speed deficits (d = 1.24), moderate executive dysfunction (d = 0.96)
- With affectivity: Integrated attention-executive deficits (d = 1.51) and preserved but modified psychomotor pattern (d = 1.41)

Temporo-parietal network dysfunction

- Group 1: pronounced visual-spatial function deficits (d = 1.18), constructive praxis impairments
- Group 2: less severe but present visual-spatial deficits (d = 0.89), compensated by attention mechanisms

Fronto-limbic network involvement

- Group 2 only: maximal attention regulation deficits (d = 1.51), working memory impairments (d = 1.33)
- Integration of cognitive control with emotional dysregulation

Interaction effects and network connectivity

Two-factor ANOVA (2×2: catatonia × affectivity) revealed significant interactions [21]:

- Main effect of catatonia: $F = 31.2$, $p < 0.001$, $\eta^2 = 0.19$

TABLE 1

COMPARATIVE RESULTS OF NEUROCOGNITIVE TESTING BY SUBGROUP

Cognitive Domain	Group 1 (*n*=35)	Group 2 (*n*=34)	Control 1 (*n*=35)	Control 2 (*n*=35)	F	*p*	η^2	Post-hoc
BACS verbal memory	34.2±8.4	30.8±8.9	42.1±9.2	40.3±10.4	18.3	<0.001	0.29	Gr1,2<C1,2
BACS motor functions	42.1±11.8	35.2±12.9	56.4±13.1	53.2±15.1	22.6	<0.001	0.33	Gr1,2<C1,2
BACS working memory	4.4±1.3	3.8±1.5	5.8±1.7	5.4±1.9	15.7	<0.001	0.26	Gr1,2<C1,2
BACS executive functions	13.9±4.2	11.6±4.8	18.7±5.1	17.1±5.7	19.4	<0.001	0.30	Gr1,2<C1,2
MoCA total score	19.8±4.6	17.5±5.1	24.2±4.2	23.0±4.8	21.8	<0.001	0.32	Gr1,2<C1,2
MoCA visual-spatial	2.3±1.1	1.9±1.3	3.5±1.0	3.3±1.2	24.1	<0.001	0.35	Gr1,2<C1,2
MoCA attention	3.5±1.4	2.8±1.6	4.8±1.2	4.4±1.4	17.9	<0.001	0.28	Gr2<Gr1<C1,2
FAB total score	9.4±3.6	7.7±3.9	13.8±3.7	12.4±4.1	25.3	<0.001	0.36	Gr1,2<C1,2
FAB interference	0.9±0.6	0.7±0.8	2.2±0.9	2.0±1.0	28.7	<0.001	0.39	Gr1,2<C1,2
FAB inhibition	1.1±0.7	0.8±0.9	2.4±0.8	2.2±0.9	26.4	<0.001	0.37	Gr1,2<C1,2

- Main effect of affectivity: F = 12.4, $p < 0.01$, $\eta^2 = 0.08$
- Interaction effect: F = 7.8, $p < 0.01$, $\eta^2 = 0.05$

Specific Network Interactions:

- Fronto-striatal × fronto-limbic: enhanced deficit in combined catatonia + affectivity (23 % increase)
- Attention networks: maximum dysfunction in catatonia + depression group
- Motor networks: preserved dysfunction pattern across affective states

Correlation Analysis with Neural Network Specificity

Catatonia severity (BFCRS) correlations by network [11]:

Group 1 (Fronto-striatal predominance):

- Psychomotor speed: r = -0.72, $p < 0.001$
- Visual-spatial functions: r = -0.65, $p < 0.001$
- Executive functions: r = -0.58, $p < 0.001$

Group 2 (Fronto-limbic predominance):

- Attention regulation: r = -0.74, $p < 0.001$
- Working memory: r = -0.69, $p < 0.001$
- Emotional control: r = -0.61, $p < 0.001$

Functional Outcomes and Network Dysfunction

Social functioning (PSP) varied significantly between groups [31]:

- Group 1: 46.2 ± 17.8 (fronto-striatal predictors: $\beta = 0.71$, $p < 0.001$)
- Group 2: 39.1 ± 18.9 (fronto-limbic predictors: $\beta = 0.58$, $p < 0.001$)
- Control groups: 60.4 ± 15.7 and 56.8 ± 18.1

Network-specific predictors of functioning:

- Fronto-striatal network: psychomotor speed and visual-spatial functions explain 62 % of variance;
- Fronto-limbic network: attention, working memory, and emotional regulation explain 67 % of variance are shown in the Table 2

DISCUSSION

Neural Network Architecture in Catatonic Cognitive Dysfunction

The factor-analytic findings are convincing that there are distinct patterns of neural network dysfunction in catatonic disorders [1]. The emergence of three separate factors in non-affective catatonia—fronto-striatal, temporo-parietal, and hippocampal—supports contemporary models of catatonia as a network disorder rather than a focal brain dysfunction [32].

Fronto-striatal network dysfunction. The prominence of the fronto-striatal factor (47.2% variance) in non-affective catatonia aligns with established neurobiological models of catatonia [1]. Neuroimaging studies have consistently shown altered connectivity between the thalamus and primary motor cortex in catatonia, which corresponds to the motor control and executive dysfunction captured by this factor. The high loadings for psychomotor speed, executive functions, and inhibitory control reflect the cardinal features of catatonic motor disturbance and cognitive rigidity.

Temporo-parietal network involvement. The identification of a separate temporo-parietal factor challenges traditional views of catatonia as primarily a motor disorder [33]. This finding is consistent with neuropsychological investigations demonstrating fronto-parietal cortical alterations in catatonia, extending dysfunction beyond motor circuits to include spatial processing and constructive abilities. This broader involvement of the cortical system may explain the complex perceptual and integrative deficits observed in catatonic patients.

Hippocampal-medial Temporal Contributions. The emergence of a distinct hippocampal factor suggests that memory consolidation deficits in catatonia reflect specific medial temporal lobe dysfunction rather than secondary effects of attention or executive impairments [27]. This finding has important implications for understanding the comprehensive cognitive profile of catatonic disorders.

Affective Modulation of Network Architecture

The transformation from three factors to two factors with affective comorbidity represents a fundamental reorganization of cognitive architecture [34]. The emergence of a dominant fronto-limbic factor (53.7 % variance) indicates that affective symptomatology does not simply add to existing deficits but reorganizes the entire pattern of network dysfunction.

Fronto-limbic integration. Recent precision functional mapping studies showing expanded fronto-striatal

TABLE 2

NEUROANATOMICAL CORRELATES OF COGNITIVE FACTORS

Factor	Cognitive Functions	Primary Brain Regions	Secondary Regions	Functional Systems (Luria)
Fronto-striatal	Executive functions, Psychomotor speed, Inhibitory control	Dorsolateral PFC, Caudate, Putamen	Supplementary motor area, Anterior cingulate	Third functional unit (programming/control)
Temporo-parietal	Visual-spatial functions, Constructive praxis	Superior parietal lobule, Inferior parietal lobule	Occipito-parietal junction, Posterior temporal	Second unit (processing/storage)
Hippocampal	Episodic memory, Delayed recall	Hippocampus, Entorhinal cortex	Parahippocampal gyrus, Fornix	Memory consolidation system
Fronto-limbic	Attention regulation, Emotional control	Orbitofrontal cortex, Amygdala	Anterior insula, Subgenual cingulate	Emotion-cognition integration

salience networks in depression provide a neurobiological basis for the observed fronto-limbic integration [8]. The high loadings for attention regulation, working memory, and emotional control suggest that affective symptoms create a unified dysfunction pattern that integrates cognitive control with emotional regulation are shown in the Table 3.

Network Efficiency and Compensation. The reduced number of factors with affective comorbidity may reflect either network breakdown or compensatory reorganization [13]. The higher variance explained by fewer factors suggests that emotional dysregulation creates a more unified but severe pattern of dysfunction, potentially reducing the brain's ability to maintain specialized network functions.

Clinical Implications of Network-Based Cognitive Profiles

Diagnostic precision. The distinct factor structures provide objective markers for different subtypes of catatonic cognitive dysfunction. Clinicians can use neuropsychological profiles to identify whether patients show predominantly fronto-striatal, temporo-parietal, or fronto-limbic patterns, informing both diagnosis and treatment planning.

Targeted rehabilitation. The network-specific findings suggest that rehabilitation should target particular neural circuits rather than treating cognitive deficits in isolation:

- Fronto-striatal dysfunction: psychomotor activation, executive training, inhibitory control exercises;
- Temporo-parietal dysfunction: visual-spatial rehabilitation, constructive skill training;
- Fronto-limbic dysfunction: Integrated attention-emotion regulation programs

Prognostic Implications. The different functional outcome predictors across groups (psychomotor speed vs. attention regulation) suggest that recovery patterns may vary systematically based on the underlying network dysfunction pattern [35].

Theoretical Implications for Catatonia Models

Network disorder hypothesis. The factor-analytic findings strongly support conceptualizing catatonia as a network disorder with distinct phenotypes reflecting different patterns of circuit dysfunction. This challenges unitary models of catatonia and suggests that the syndrome represents convergent behavioral endpoints arising from diverse network alterations.

Dimensional vs. categorical classification. The continuous nature of factor scores and their correlations with symptom severity support dimensional models of catatonic cognitive dysfunction. Rather than discrete categories, cognitive profiles appear to represent points along continuous dimensions of network dysfunction.

Interaction models. The significant interaction between catatonia and affectivity in network organization suggests that psychiatric comorbidity fundamentally alters brain network architecture rather than simply adding additional symptoms. This has important implications for understanding treatment resistance and complex presentations.

Our findings provide critical insights for neuropsychological theory and practice by demonstrating how catatonic cognitive dysfunction aligns with established theoretical frameworks:

Integration with Luria's functional systems theory. The three-factor structure in non-affective catatonia directly corresponds to Luria's three functional units of the brain. The fronto-striatal factor (47.2 % variance) maps onto the third functional unit (programming and control), the temporo-parietal factor (26.3 %) corresponds to the second unit (information processing and storage), while the hippocampal factor (19.8 %) represents the memory consolidation system operating across all units. This alignment validates Luria's model in a clinical population with specific network dysfunction, extending its applicability beyond focal lesions to distributed network disorders.

Contribution to cognitive neuropsychology models. Our factor-analytic approach reveals that catatonic cognitive dysfunction follows predictable patterns based on network architecture rather than random impairment. The transformation from three to two factors with affective comorbidity demonstrates that cognitive architecture is dynamically reorganized rather than simply degraded. This supports dynamic localization theories suggesting that mental functions are not rigidly localized but emerge from flexible network configurations.

Clinical neuropsychological assessment implications. The distinct factor profiles enable the development of targeted neuropsychological assessment protocols:

TABLE 3

NEUROPSYCHOLOGICAL MARKERS AND THEIR NETWORK CORRELATES

Neuropsychological Domain	Test/Measure	Factor Loading	Network Correlation	Clinical Cutoff	Sensitivity	Specificity
Psychomotor Speed	Symbol Coding	0.81	Fronto-striatal	<35 scaled	78%	82%
Executive Control	Stroop Interference	0.78	Fronto-striatal	>70 sec	75%	79%
Visuospatial Function	Block Design	0.79	Temporo-parietal	<7 scaled	72%	77%
Working Memory	Digit Span Backward	0.82	Fronto-limbic	<4 span	80%	75%
Attention Regulation	CPT d-prime	0.85	Fronto-limbic	<2.0	83%	78%

- For suspected fronto-striatal dysfunction: Prioritize tests of psychomotor speed (Symbol Coding), executive control (Stroop, Trail Making B), and motor programming (Luria sequences);
- For temporo-parietal involvement: Emphasize visuospatial tasks (Block Design, Rey Figure Copy), constructive praxis, and spatial working memory;
- For fronto-limbic patterns: Focus on attention regulation (CPT), emotional Stroop, and integrated cognitive-affective tasks.

Theoretical advancement in understanding catatonia. Our findings challenge the traditional view of catatonia as primarily a motor disorder by revealing systematic cognitive architecture disruption. The factor structure suggests catatonia represents a "disconnection syndrome" where cognitive modules become functionally isolated rather than destroyed. This aligns with Geschwind's disconnection theory and modern network neuroscience, positioning catatonia as a model disorder for understanding brain-behavior relationships.

Neuropsychological Rehabilitation Strategies

Based on our findings and Luria's restoration principles, we propose differentiated rehabilitation protocols:

For Fronto-striatal Dysfunction:

- Graduated motor program training using Luria's method of external mediation
- Rhythmic organization exercises (metronome-paced movements)
- Goal-Management Training for executive dysfunction
- Implementation intentions technique for action initiation

For Temporo-parietal Dysfunction:

- Visual-spatial restoration using graduated complexity tasks
- Constructional ability training through block design exercises
- Body schema restoration techniques
- Spatial navigation training in virtual environments

For Fronto-limbic Dysfunction:

- Cognitive-Behavioral Therapy adapted for attention deficits
- Mindfulness-based cognitive training
- Emotional regulation through biofeedback
- Working memory training with emotional valence manipulation

These approaches represent novel integration of classical neuropsychological methods with network-based understanding, offering precision rehabilitation tailored to individual cognitive profiles [35].

Limitations and future directions

Methodological Considerations. The cross-sectional design limits causal inferences about network dysfunction development. Longitudinal studies tracking factor structure changes over time would provide insights into whether these represent stable trait markers or dynamic state-dependent patterns.

Sample heterogeneity. Despite subdivision by affective symptoms, diagnostic heterogeneity within groups may influence factor structure. Future studies should examine factor stability across specific diagnostic categories and medication states.

Network validation. While factor analysis provides evidence for cognitive network organization, direct validation through neuroimaging studies examining structure-function relationships would strengthen the neural network interpretations.

Clinical translation. Prospective studies examining whether factor-guided rehabilitation strategies improve outcomes compared to standard approaches would validate the clinical utility of this network-based approach.

CONCLUSION

This factor-analytic study provides novel evidence for distinct patterns of cognitive network dysfunction in catatonic disorders. The identification of separate fronto-striatal, temporo-parietal, and hippocampal factors in non-affective catatonia, and their reorganization into fronto-limbic and motor patterns with affective comorbidity, demonstrates that catatonia represents a network disorder with multiple phenotypic expressions.

The findings have immediate clinical implications for precision diagnosis and targeted rehabilitation. Rather than treating catatonic cognitive dysfunction as a unitary impairment, clinicians should assess and target specific network dysfunctions based on individual cognitive profiles. The strong correlations between factor scores and functional outcomes validate the clinical relevance of these network-based distinctions.

Future research should focus on validating these cognitive network patterns through neuroimaging studies, developing network-targeted interventions, and examining the stability and modifiability of these patterns over time. The factor-analytic approach provides a framework for understanding the neurobiological heterogeneity of catatonic disorders and developing personalized treatment strategies based on individual patterns of network dysfunction.

This network-based understanding of catatonic cognitive dysfunction represents a significant advance in our ability to characterize, predict, and treat the complex cognitive impairments associated with this challenging neuropsychiatric syndrome.

The factor-specific predictors of functioning (psychomotor speed and visuospatial functions explaining 62 % of variance in non-affective; attention and emotion regulation explaining 67 % in affective catatonia) provide clinicians with concrete targets for assessment and intervention. These findings transform catatonia from a phenomenological syndrome to a neuropsychologically-defined disorder with measurable cognitive endophenotypes.

This study advances neuropsychological understanding by demonstrating that catatonic cognitive dysfunction reflects systematic disruption of Luria's functional brain systems, manifested as distinct factor-analytic profiles.

The practical significance lies in enabling precision rehabilitation through network-targeted interventions. Our findings support a paradigm shift from treating catatonia as a unitary syndrome to addressing specific patterns of functional system dysfunction, offering hope for improved outcomes through personalized neuropsychological rehabilitation.

Conflicts of interest

The authors declare that there are no obvious and potential conflicts of interest in connection with the publication of this article.

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