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Testing the Structural Model of the Relationship Between Metacognitive Deficits, Social Cognition, and Executive Functions With Dysfunction in Patients With Schizophrenia Considering the Mediating Role of Intrinsic Motivation

Elham. Zandasta[1], Kobra. Haji Alizadeh[1*], Seyed Hamzeh. Seddigh[2]

[1] Department of Psychology, BA.C.,Islamic Azad University, Bandar Abbas, Iran
[2] Associate Professor, Department of Psychiatry, Faculty of Medicine, Hormozgan University of Medical Sciences, Iran

*** Corresponding author email address**: Hajializadehk@iau.ac.ir

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A B S T R A C T

Purpose: This study aimed to investigate the structural model of the relationship between metacognitive deficits, social cognition, and executive functions with cognitive dysfunction in patients with schizophrenia, considering the mediating role of intrinsic motivation.

Methods and Materials: The research employed a descriptive-analytical and applied design. The statistical population consisted of 789 patients with schizophrenia in Bandar Abbas, from which 400 participants were selected using Cochran's formula and simple random sampling. Measurement tools included a researcher-developed questionnaire and three standardized instruments: Nejati's Cognitive Abilities Questionnaire, the Barkley Deficits in Executive Functioning Scale (BDEFS), and the McAuley Intrinsic Motivation Inventory. Content validity was confirmed by psychology experts, and construct validity was verified via confirmatory factor analysis (CFA). Reliability was assessed using Cronbach's alpha, composite reliability (CR), and average variance extracted (AVE). Data were analyzed using Pearson correlation, regression analysis, and structural equation modeling (SEM), with bootstrapping applied to examine the mediating effect of intrinsic motivation.

Findings: Results demonstrated significant positive correlations between all core variables: metacognitive deficits, social cognition, executive functions, intrinsic motivation, and cognitive dysfunction. Regression analysis showed that metacognitive deficits ($\beta = 0.273$, $p < .001$), social cognition ($\beta = 0.363$, $p < .001$), and executive functions ($\beta = 0.256$, $p < .001$) significantly predicted dysfunction, while intrinsic motivation did not have a direct significant effect ($\beta = 0.095$, $p = .084$). However, intrinsic motivation significantly mediated the relationship between metacognitive deficits and dysfunction (indirect effect = 0.175, 95% CI [0.093, 0.245], $p = .004$).

Conclusion: The findings confirm that metacognitive deficits, social cognition, and executive functions are significant predictors of dysfunction in patients with schizophrenia, with intrinsic motivation playing a meaningful mediating role in the pathway from metacognitive deficits to dysfunction. These results highlight the importance of cognitive and motivational factors in therapeutic interventions for schizophrenia.

Keywords: *Schizophrenia; Metacognitive Deficits; Social Cognition; Executive Functions; Dysfunction; Structural Equation Modeling (SEM)*

1. Introduction

Schizophrenia is a chronic and debilitating mental disorder characterized by a complex interplay of cognitive, emotional, and behavioral impairments that interfere with individuals' ability to function effectively in daily life. While the clinical picture of schizophrenia has traditionally emphasized hallucinations, delusions, and negative symptoms, increasing attention in recent decades has shifted toward understanding the cognitive mechanisms that underpin dysfunction in this population. Among these mechanisms, metacognitive deficits, social cognition impairments, and executive dysfunctions have emerged as key predictors of functional disability in individuals with schizophrenia (Kazemi, 2024; Motut et al., 2023; Parola et al., 2020). Metacognition, or the ability to reflect upon, monitor, and regulate one's own cognitive and emotional processes, plays a critical role in one's capacity to understand the mental states of oneself and others, and in turn influences executive performance and social interaction quality (Rivas et al., 2022; Wells & Nordahl, 2023).

Recent theoretical and empirical frameworks have posited that cognitive and functional impairments in schizophrenia are not merely the result of deficits in isolated domains but are better understood as interrelated dysfunctions within broader cognitive systems (Afar & Bilgiç, 2021; Mahmoud Alilou et al., 2020). For instance, metacognitive deficits often co-occur with impairments in social cognition, such as theory of mind and empathy, which further compromise patients' ability to interpret and respond to social cues appropriately (Ashaieri, 2018; Güner & Erbay, 2021). These interrelations suggest that social dysfunction in schizophrenia may be a downstream consequence of impaired self-awareness and mental state attribution. Similarly, executive functions—encompassing domains such as planning, inhibitory control, and working memory—are essential for implementing goal-directed behaviors, and their disruption has been consistently linked with both symptom severity and everyday functioning (Goodarzi et al., 2019; Motavallibashi Naeini, 2018).

In recent years, research has further emphasized the mediating roles of motivational factors, particularly intrinsic motivation, in shaping the impact of cognitive deficits on functional outcomes. Intrinsic motivation—the inherent drive to engage in activities for their own sake—has been shown to moderate the relationship between cognitive processes and behavioral engagement, especially in populations with severe mental illness (Luther et al., 2016; Rezazadeh et al., 2023). In individuals with schizophrenia, low intrinsic motivation has been associated with negative symptoms such as avolition and anhedonia, which exacerbate social withdrawal and cognitive inertia. Conversely, improvements in intrinsic motivation have been linked to better therapeutic engagement and functional recovery. This line of inquiry has inspired a growing number of studies examining how motivation mediates the influence of metacognitive, social, and executive dysfunctions on real-world performance (Benjamin et al., 2021; Diop et al., 2022).

The role of metacognitive functioning is particularly salient in understanding how individuals with schizophrenia perceive their internal states, assess their cognitive errors, and develop adaptive strategies for coping with illness-related challenges. Deficits in metacognitive awareness have been consistently reported across the schizophrenia spectrum and are strongly linked with impaired insight and poor psychosocial outcomes (Luther et al., 2016; Zekri et al., 2024). These deficits not only affect symptom appraisal but also compromise the ability to set meaningful goals, self-monitor progress, and engage in self-directed behavior—all of which are essential for adaptive functioning. Studies such as those by Kul et al. (2024) have shown that even in related disorders such as OCD, metacognition has a significant effect on social functioning, further reinforcing its relevance in psychiatric populations (Kul et al., 2024).

Social cognition, encompassing theory of mind, emotion recognition, and empathy, has also been recognized as a distinct and essential domain of cognitive functioning in schizophrenia (Ashaieri, 2018; Motut et al., 2023). Impairments in this area are particularly damaging because

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they directly hinder interpersonal interactions and contribute to social isolation—a common and debilitating feature of schizophrenia. Research indicates that social cognitive deficits are more predictive of community functioning than general neurocognition, suggesting that targeted interventions to enhance social cognition may yield significant functional benefits (Parola et al., 2020; Tabassum et al., 2023). Furthermore, there is emerging evidence that these impairments are intricately linked to metacognitive processes, forming a dynamic cognitive-affective loop that influences real-world behavior (Diop et al., 2022; Motut et al., 2023).

Executive dysfunctions in schizophrenia are well-documented, with impairments in working memory, cognitive flexibility, and inhibitory control representing core features of the disorder (Jokar Kamalabadi et al., 2021; Mahmoud Alilou et al., 2020). These deficits often manifest early in the illness course and have been shown to persist even during periods of symptomatic remission. They significantly interfere with treatment adherence, vocational functioning, and independent living. Bahr et al. (2019), for example, demonstrated that medication side effects, such as those associated with antipsychotic use, can further exacerbate cognitive impairments by altering neurobiological substrates critical for executive control (Bahr & et al., 2019). Consequently, any comprehensive model of schizophrenia-related dysfunction must account for the contributory role of executive function in day-to-day performance.

In addition to these cognitive domains, physiological studies have underscored the importance of neurobiological markers in understanding the complexity of schizophrenia. Measures such as heart rate variability (HRV) have been explored as potential indices of autonomic dysfunction and illness severity (Benjamin et al., 2021; Suda & et al., 2022). Afar and Bilgiç (2021) showed that auditory stimulation elicited significant changes in HRV among patients with schizophrenia, suggesting an interaction between sensory processing, emotional regulation, and cardiovascular function (Afar & Bilgiç, 2021). These findings align with broader neuropsychiatric models positing that disruptions in brain-body connectivity may underlie both cognitive and affective dysregulation in schizophrenia (Williams, 2019).

Given the multidimensional nature of schizophrenia, it is essential to adopt integrative frameworks that examine the interrelationships between cognitive, motivational, and functional variables. For instance, Asdolahzadeh et al. (2021) proposed a structural model linking metacognition, self-efficacy, and behavioral tendencies in adolescents, which may be applicable in understanding motivational processes in schizophrenia as well (Asdolahzadeh et al., 2021). Similarly, Rivas et al. (2022) emphasize the need to incorporate metacognitive strategies into educational and therapeutic interventions to improve self-regulation and critical thinking skills—both of which are compromised in patients with schizophrenia (Rivas et al., 2022). These approaches are further supported by research highlighting the efficacy of training programs designed to enhance metacognitive awareness and social-emotional reasoning, which may have downstream benefits for functional recovery (Kazemi, 2024; Zekri et al., 2024).

In light of the above evidence, the present study aims to contribute to the growing body of literature by proposing and testing a structural model that explores the direct and indirect relationships between metacognitive deficits, social cognition, executive functions, and dysfunction in patients with schizophrenia.

2. Methods and Materials

2.1. Study Design and Participants

The present study is descriptive-analytical in nature and falls within the category of applied research. Theoretical models were employed to design the conceptual model, which examines the relationship between metacognitive deficits, social cognition, executive functions, and social functioning in patients with schizophrenia, considering the mediating role of intrinsic motivation. The statistical population consists of individuals diagnosed with schizophrenia. A simple random sampling method was used, and a sample of 50 participants was selected for the initial pilot testing of the instruments. Data collection was conducted using both field and library methods.

2.2. Measures

The instruments included one researcher-developed questionnaire and three standardized questionnaires:

1. **Researcher-Developed Questionnaire:** This instrument contains 21 items across four core components (metacognition, intrinsic motivation, executive functions, and social cognition). Items are rated on a four-point Likert scale. For a subset of questions (items 19 to 21), scoring is reversed.
2. **Nejati's Cognitive Abilities Questionnaire (2013):** This tool consists of 30 items across seven

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components, evaluated using a five-point Likert scale. The overall reliability of the questionnaire has been reported with a Cronbach's alpha of 0.834.

3. **Barkley Deficits in Executive Functioning Scale (BDEFS):** This scale includes 89 items distributed across five subscales and uses a Likert scale for scoring. Some items are reverse-scored. The tool's validity and reliability have been confirmed in previous studies.

2.3. Data Analysis

In the present research, content validity was approved by experts in psychology, and construct validity was assessed through confirmatory factor analysis (CFA) using AMOS software. Fit indices including CFI = 0.93, RMSEA = 0.07, and GFI = 0.91 indicate an acceptable fit of the conceptual model to the data. The internal consistency of the instruments was evaluated using Cronbach's alpha; the overall value for the questionnaire was 0.90, with subscale values ranging between 0.79 and 0.87, indicating good reliability. Ultimately, the data were analyzed using confirmatory factor analysis and hypothesis testing.

3. Findings and Results

The sample consisted of 400 patients diagnosed with schizophrenia, of whom 63.7% (n = 255) were female and 36.3% (n = 145) were male. Participants' ages ranged from 19 to 61 years, with a mean age of 36.45 years (SD = 7.7). Most participants (43.5%) were between 30 and 40 years old, followed by those aged 41–51 years (32%), 19–29 years (24%), and only 0.5% aged over 51. Regarding marital status, 47.8% of participants were single, 46.3% married, 3.3% divorced, and 2.8% widowed. Concerning hospitalization history, 43% had been hospitalized more than three times, 30.3% one to three times, and 26.8% only once. In terms of comorbidity, 59% reported having at least one comorbid condition, whereas 41% did not. Additionally, 58.3% of participants reported a family history of psychiatric disorders, while 41.8% reported no such history. The mean duration since diagnosis was 11.16 years (SD = 5.47), ranging from 1 to 27 years.

To better understand the research population and gain a clearer view of the study variables, descriptive statistics were examined prior to conducting statistical analyses. Before testing the study hypotheses, descriptive statistics for the variables used in the research were reviewed. The mean, as a measure of central tendency, represents the central point of the distribution and reflects a value which, if replaced with all observations in the dataset, would not alter the overall sum of the data. Furthermore, the maximum and minimum represent the highest and lowest values of the variables in the sample, respectively. The results of the descriptive statistics are presented in the following table.

Table 1

Mean and Standard Deviation of Variables and Subscales of the Model

Factor	Mean	SD	Skewness	Kurtosis	Min	Max
Social Functioning	51.42	20.28	-0.344	-1.70	19	78
Memory	19.64	7.73	-0.373	-1.47	6	30
Inhibitory Control & Selective Attention	19.81	7.94	-0.406	-1.47	6	30
Decision-Making	16.48	6.60	-0.384	-1.43	5	25
Planning	9.97	4.11	-0.362	-1.30	3	15
Sustained Attention	9.82	4.25	-0.318	-1.43	3	15
Social Cognition	9.87	4.22	-0.356	-1.39	3	15
Cognitive Flexibility	13.05	5.26	-0.339	-1.37	4	20
Social Cognition (Total)	98.66	36.90	-0.450	-1.68	40	144
Interest / Enjoyment	29.96	11.71	-0.305	-1.50	7	48
Perceived Competence	25.56	10.41	-0.295	-1.48	6	42
Effort / Importance	21.43	8.61	-0.303	-1.43	5	35
Tension / Pressure	21.54	8.97	-0.348	-1.47	5	34
Perceived Choice	30.47	12.01	-0.303	-1.54	8	49
Value / Usefulness	30.41	11.75	-0.321	-1.49	8	48
Communication	30.48	11.95	-0.331	-1.47	8	49
Intrinsic Motivation (Total)	189.88	71.25	-0.367	-1.78	81	274
Subscale 1	55.54	19.98	-0.299	-1.79	25	80
Subscale 2	63.64	22.69	-0.310	-1.79	29	91
Subscale 3	50.19	18.48	-0.319	-1.77	20	72
Subscale 4	31.86	11.51	-0.293	-1.70	14	48

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Subscale 5	34.49	12.41	-0.299	-1.71	15	51
Executive Functions (Total)	235.74	83.71	-0.322	-1.87	120	326
Metacognitive Domain	16.36	5.97	-0.437	-1.45	6	24
Intrinsic Motivation Domain	18.99	6.84	-0.390	-1.55	7	28
Executive Function Domain	13.46	4.85	-0.393	-1.41	5	20
Social Cognition Domain	8.27	3.06	-0.433	-1.29	3	12
Metacognitive Deficits	57.09	19.71	-0.450	-1.67	24	81

Based on the data in the table above, the mean and standard deviation of social functioning were 51.42 ± 20.28, social cognition 98.66 ± 36.90, intrinsic motivation 189.88 ± 71.25, and executive functions 235.74 ± 83.71. For the researcher-developed questionnaire, the mean and standard deviation were 57.09 ± 19.71. Additionally, the subdomains reported the following mean and SD values: metacognitive domain = 16.32 ± 5.97, intrinsic motivation domain = 18.99 ± 6.84, executive function domain = 13.43 ± 4.85, and social cognition domain = 8.26 ± 3.06. The table includes descriptive statistics such as mean, standard deviation, minimum, and maximum values for each research variable and subscale. To assess normality of data distribution, skewness and kurtosis indices were used, given the large sample size. All skewness and kurtosis values fall within the acceptable range of -2 to +2, indicating a normal distribution.

To conduct appropriate statistical procedures and make logical inferences regarding the study hypotheses, it is essential to select the proper statistical method. Determining the distribution of data is a primary step. Since the distribution of data was parametric (normal), Pearson correlation analysis was applied to assess the relationships between the core variables.

Table 2

Correlation Between Model Factors and Subscales

Variable	*Dysfunction*	*Social Cognition*	*Executive Functions*	*Metacognitive Deficits*	*Intrinsic Motivation*
Dysfunction	*1*				
Social Cognition	*.650***	*1*			
Executive Functions	*.651***	*.595***	*1*		
Metacognitive Deficits	*.635***	*.584***	*.637***	*1*	
Intrinsic Motivation	*.605***	*.596***	*.689***	*.646***	*1*

*p<0.05, **p<0.01

The results of the Pearson correlation presented in the table above demonstrate statistically significant relationships among all the main variables. As shown, all correlation coefficients fall between -1 and +1, and each variable is significantly correlated with the others at the 0.01 level, indicating robust and meaningful associations between dysfunction, social cognition, executive functions, metacognitive deficits, and intrinsic motivation.

Before conducting factor analysis, it is essential to ensure that the available data are suitable for such analysis. To this end, the Kaiser–Meyer–Olkin (KMO) index and Bartlett's test of sphericity were used. Based on the obtained significance level, it was determined that the data were appropriate for factor analysis. The KMO index assesses whether the sample size is adequate for factor analysis. This index ranges from 0 to 1, and values closer to 1 (with a minimum acceptable value of 0.60) indicate that the data are suitable for factor analysis. Conversely, values lower than 0.60 suggest that factor analysis is not appropriate for the data. Given the obtained KMO value of 0.728, the sample size is considered adequate for factor analysis.

Bartlett's test of sphericity tests the null hypothesis that the correlation matrix of the observed variables is an identity matrix. A significant result ($p < .05$) indicates that the variables are correlated, and thus, factor analysis is appropriate. The chi-square test used in Bartlett's test confirms this association.

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Table 3

Bartlett's Test Results

Test	Value
Chi-Square (χ^2)	71830.42
Degrees of Freedom	19900
Significance Level	.000

According to the table, the significance level is .000, which indicates that the null hypothesis is rejected. Therefore, there are significant correlations among the variables. Model fit refers to how well a model corresponds to the observed data. This section assesses the goodness-of-fit of the hypothesized model to ensure its compatibility with the research data and to eventually derive answers to the research questions. The conceptual model's structural fit was evaluated and is discussed in detail below.

Table 4

Structural Equation Modeling Fit Indices

Fit Index	Acceptable Range	Obtained Value	Evaluation
χ^2 / df	< 5	1.857	Acceptable
RMSEA	< 0.08	0.046	Acceptable
RMR	> 0	20.514	Acceptable
GFI	> 0.90	0.912	Acceptable
AGFI	> 0.85	0.893	Acceptable
NFI	> 0.90	0.967	Acceptable
CFI	> 0.90	0.985	Acceptable
IFI	> 0.90	0.985	Acceptable

Figure 1

Model with T-Values

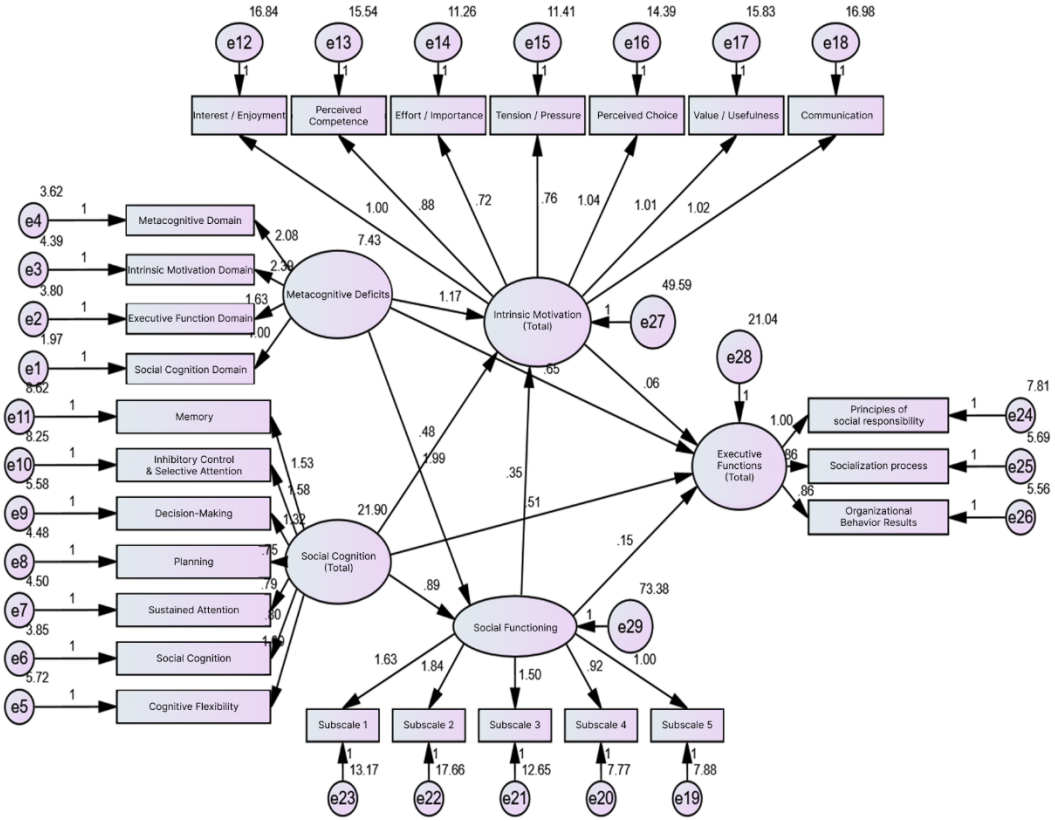

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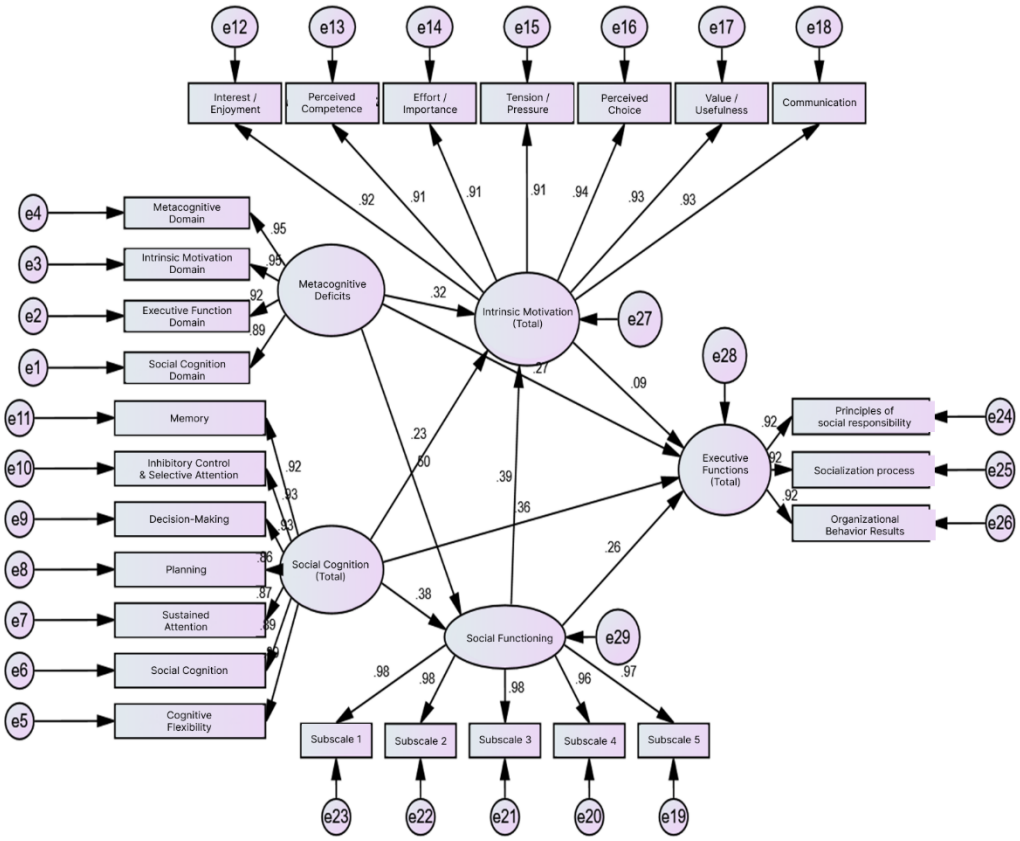
Figure 2

Model with Path Coefficients



The table includes the goodness-of-fit indices for the structural equation model. Various indices were applied to evaluate the model's compatibility with the data. The chi-square to degrees of freedom ratio (χ^2/df) reflects the overall fit and should be below 5 to be considered acceptable. RMSEA (Root Mean Square Error of Approximation) reflects how well the model approximates the population covariance matrix; a value below 0.08 suggests good fit. RMR (Root Mean Square Residual) indicates the residuals between predicted and observed values and should be above zero. GFI (Goodness-of-Fit Index) evaluates the proportion of variance accounted for by the estimated population covariance. AGFI (Adjusted Goodness-of-Fit Index) adjusts for degrees of freedom. NFI (Normed Fit Index), CFI (Comparative Fit Index), and IFI (Incremental Fit Index) compare the proposed model to a null model and should all exceed 0.90 for the model to be considered a good fit. As shown, all indices fall within acceptable ranges, confirming that the model demonstrates an appropriate and desirable fit with the data.

Table 5

Results of Cronbach's Alpha, Composite Reliability, and Convergent Validity

Variables	Cronbach's Alpha (α > 0.7)	Composite Reliability (CR > 0.7)	Average Variance Extracted (AVE > 0.5)
Dysfunction	0.952	0.954	0.873
Social Cognition	0.972	0.967	0.808
Executive Functions	0.992	0.991	0.957
Metacognitive Deficits	0.965	0.960	0.859
Intrinsic Motivation	0.980	0.979	0.872

Since all values of Cronbach's alpha, composite reliability (internal consistency), and AVE fall within the accepted thresholds, the model demonstrates satisfactory reliability and convergent validity.

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Table 6

Regression Coefficients for Research Hypotheses

Hypothesis	Path Description	Coefficient	Standard Error	Critical Ratio	*p*-value
Hypothesis 1	Dysfunction → Metacognitive Deficits	0.273	0.120	5.451	.000
Hypothesis 2	Dysfunction → Social Cognition	0.363	0.064	7.884	.000
Hypothesis 3	Dysfunction → Executive Functions	0.256	0.032	4.767	.000
Hypothesis 4	Dysfunction → Intrinsic Motivation	0.095	0.036	1.730	.084
Hypothesis 5	Intrinsic Motivation → Metacognitive Deficits	0.321	0.167	7.012	.000
Hypothesis 6	Intrinsic Motivation → Social Cognition	0.228	0.090	5.392	.000
Hypothesis 7	Intrinsic Motivation → Executive Functions	0.389	0.044	8.006	.000

The regression coefficients in the table indicate positive effects between paired variables. Generally, a critical ratio greater than 1.96 implies statistical significance at the 5% level.

The following table presents the indirect effects of metacognitive deficits on dysfunction through intrinsic motivation, assessed using the bootstrapping method.

Table 7

Mediation Effect Analysis

Path	Indirect Effect	Lower Bound	Upper Bound	*p*-value
Metacognitive Deficits → Intrinsic Motivation → Dysfunction	0.175	0.093	0.245	.004

The results of the indirect path analysis indicate that intrinsic motivation significantly mediates the relationship between metacognitive deficits and cognitive dysfunction in patients with schizophrenia. The computed indirect effect is 0.175 (CI: 0.093 to 0.245), with a significance level of .004, which is below the 0.05 threshold, confirming statistical significance. Therefore, these findings suggest that metacognitive deficits indirectly affect cognitive dysfunction through their influence on intrinsic motivation, underscoring the importance of intrinsic motivation as a mediator in this relationship.

Hypothesis 1: There is a relationship between metacognitive deficits and dysfunction in patients with schizophrenia. The regression analysis shows that the coefficient for the effect of metacognitive deficits on dysfunction is 0.273, indicating a significant positive relationship. The critical ratio is 5.451, which exceeds the 1.96 threshold, confirming significance at the 5% level. This suggests that increased metacognitive deficits are associated with increased cognitive dysfunction, highlighting metacognitive deficits as a key predictor of cognitive impairments in schizophrenia.

Hypothesis 2: There is a relationship between social cognition and dysfunction in patients with schizophrenia. The regression coefficient for this relationship is 0.363, with a critical ratio of 7.884, significantly higher than the 1.96 threshold. This confirms a statistically significant positive relationship between social cognition and dysfunction, indicating that higher levels of social cognition are significantly associated with increased cognitive dysfunction. These findings point to the role of social cognition as a potential predictor of functional cognitive impairments in this population.

Hypothesis 3: There is a relationship between executive functions and dysfunction in patients with schizophrenia. The regression coefficient is 0.256, and the critical ratio is 4.767, which is also well above the 1.96 threshold, confirming significance. This implies that impairments in executive functions are significantly associated with increased cognitive dysfunction, further suggesting executive functions as a key variable in explaining and predicting dysfunction in schizophrenia.

Hypothesis 4: There is a relationship between intrinsic motivation and dysfunction in patients with schizophrenia. The regression coefficient is 0.095, with a critical ratio of 1.730—below the 1.96 threshold—indicating that this relationship is not statistically significant. Thus, intrinsic motivation alone does not appear to be a strong predictor of cognitive dysfunction in this population, and its influence may be moderated or overshadowed by other variables.

Hypothesis 5: There is a relationship between metacognitive deficits and intrinsic motivation in patients

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with schizophrenia. The regression coefficient is 0.321, and the critical ratio is 7.012, exceeding the 1.96 threshold and confirming significance. This demonstrates that higher levels of metacognitive deficits are significantly associated with increased intrinsic motivation. These findings emphasize the potential impact of metacognitive deficits on motivational processes in schizophrenia and the relevance of metacognitive factors in explaining variations in intrinsic motivation.

4. Discussion and Conclusion

The aim of the present study was to examine the structural relationships among metacognitive deficits, social cognition, and executive functions with cognitive dysfunction in patients with schizophrenia, with intrinsic motivation considered as a mediating variable. The results confirmed significant positive relationships among all key variables, including metacognitive deficits, social cognition, executive functions, intrinsic motivation, and dysfunction. Specifically, metacognitive deficits, social cognition, and executive functions significantly predicted cognitive dysfunction, while intrinsic motivation did not have a direct significant effect on dysfunction. However, the mediation analysis indicated that intrinsic motivation significantly mediated the relationship between metacognitive deficits and dysfunction. These findings provide empirical support for the proposed structural model and align with previous research in this domain.

The significant and positive relationship between metacognitive deficits and dysfunction in patients with schizophrenia is consistent with prior findings emphasizing the role of metacognitive impairments in poor insight, diminished self-regulation, and reduced functional capacity (Luther et al., 2016; Wells & Nordahl, 2023). Metacognition enables individuals to interpret, evaluate, and adaptively respond to internal cognitive and emotional experiences. Its disruption in schizophrenia impairs one's ability to monitor thoughts, recognize errors, and modify behaviors accordingly. This aligns with the conclusions of Motut et al. (2023), who highlighted that metacognitive deficits have a cascading effect on social cognition and functional outcomes (Motut et al., 2023). The significant indirect effect of metacognitive deficits on dysfunction via intrinsic motivation also echoes the findings of Rezazadeh et al. (2023), who demonstrated that cognitive regulation and motivational dynamics are closely intertwined in models of metacognitive development (Rezazadeh et al., 2023).

The current study also found a significant direct relationship between social cognition and dysfunction, reinforcing the pivotal role of social cognitive processes in shaping real-world functioning. This is in line with Parola et al. (2020), who emphasized that theory of mind, pragmatic reasoning, and emotion perception are critical for maintaining interpersonal relationships and community integration in schizophrenia (Parola et al., 2020). Impairments in social cognition disrupt one's ability to navigate social exchanges, often leading to isolation and miscommunication. The association between social cognition and intrinsic motivation further supports the notion that social understanding contributes to self-initiated engagement with the environment, potentially enhancing one's willingness to participate in socially demanding contexts (Diop et al., 2022). Furthermore, findings by Ashaieri (2018) and Güner and Erbay (2021) substantiate that poor social cognition not only relates to misinterpretation of others' intentions but also correlates with emotional dysregulation and susceptibility to paranoia in schizophrenia (Ashaieri, 2018; Güner & Erbay, 2021).

Another important outcome was the strong association between executive function and dysfunction. Executive functions, including working memory, inhibitory control, cognitive flexibility, and planning, are fundamental to adaptive behavior. Their impairments have long been considered core features of schizophrenia (Mahmoud Alilou et al., 2020). This study supports previous work by Motavallibashi Naeini (2018), who showed that deficits in executive functions are predictive of behavioral disruptions and maladaptive responses in adolescence—a pattern that likely persists and intensifies in clinical schizophrenia populations (Motavallibashi Naeini, 2018). The link between executive function and intrinsic motivation observed here also highlights how cognitive control supports goal-directed behavior. Without sufficient executive capacity, individuals may struggle to initiate and sustain internally motivated activity. This aligns with earlier studies suggesting that motivational decline in schizophrenia may be partially attributable to impaired cognitive infrastructure (Luther et al., 2016; Wells & Nordahl, 2023).

Interestingly, while intrinsic motivation did not directly predict dysfunction, it played a significant mediating role between metacognitive deficits and dysfunction. This suggests that metacognitive awareness may influence functioning not only through cognitive and behavioral pathways but also by shaping motivational states. This is congruent with models proposed by Rivas et al. (2022), who

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argue that metacognitive training enhances learners' self-efficacy and goal orientation by improving their perception of self-agency and mental competence (Rivas et al., 2022). Additionally, Zekri et al. (2024) demonstrated that therapeutic interventions combining metacognitive therapy with value-based approaches such as logotherapy and hope therapy resulted in significant improvements in dysfunctional belief systems, supporting the idea that motivational processes can be shaped through metacognitive modulation (Zekri et al., 2024). Our findings imply that intrinsic motivation may serve as a conduit through which self-reflective capacity translates into meaningful behavior change in schizophrenia.

Further supporting the multidimensional perspective of dysfunction in schizophrenia, the study aligns with Afar and Bilgiç (2021), who highlighted physiological underpinnings of dysfunction through heart rate variability analyses, linking poor autonomic regulation with disrupted attentional and motivational states (Afar & Bilgiç, 2021). Similarly, Benjamin et al. (2021) reported that HRV correlates with disease severity in psychosis, suggesting that biological markers may reflect not only clinical symptomatology but also cognitive and motivational dysfunction (Benjamin et al., 2021). This implies that intrinsic motivation may have a psychophysiological basis, interacting with neurocognitive and emotional systems to influence daily functioning.

The implications of this study extend to therapeutic and rehabilitative practices. Enhancing metacognitive abilities may indirectly improve patient functioning through strengthening motivation. This supports the inclusion of metacognitive therapy (MCT) and motivational enhancement strategies in treatment programs for schizophrenia (Asdolahzadeh et al., 2021; Kazemi, 2024). Targeting metacognition could improve insight and emotional awareness, which in turn would support the development of intrinsic goals and personal agency. Moreover, social cognition training and executive function remediation may further enhance the likelihood that improvements in metacognitive awareness translate into real-world gains. As emphasized by Tabassum et al. (2023), understanding the interplay between cognitive structures and emotional constructs such as guilt and self-blame is critical in creating effective, individualized treatment strategies (Tabassum et al., 2023).

Ultimately, this study reinforces the idea that dysfunction in schizophrenia is not solely a product of symptom severity but is shaped by a complex web of cognitive, emotional, and motivational factors. The integration of metacognition, executive function, social cognition, and intrinsic motivation into a single model provides a comprehensive understanding of the psychological architecture underlying functional impairment. The findings suggest a pathway through which therapeutic interventions can be optimized to target both cognitive skills and motivational processes, potentially leading to improved outcomes in patients with schizophrenia.

Despite its strengths, this study is not without limitations. First, the cross-sectional design limits the ability to infer causality among the variables. Longitudinal data would be necessary to determine the temporal ordering and potential bidirectionality of the relationships observed. Second, all data were collected using self-report and standardized questionnaires, which may be subject to biases such as social desirability and inaccurate introspection—particularly in a clinical population known for impaired insight. Third, the study focused on patients from a specific geographic location, which may limit the generalizability of the findings to other sociocultural or clinical contexts. Additionally, physiological or neuroimaging data were not included, which might have provided deeper insight into the biological correlates of the psychological constructs examined.

Future studies should adopt longitudinal or experimental designs to explore causal mechanisms between metacognitive processes, intrinsic motivation, and functional outcomes in schizophrenia. Incorporating neurobiological or psychophysiological markers such as HRV, EEG, or fMRI data could enrich understanding of how brain-body interactions influence motivation and cognition. Comparative studies between different subgroups of schizophrenia (e.g., paranoid vs. disorganized) may reveal differential patterns of cognitive and motivational disruption. Furthermore, future research could examine the effectiveness of integrated interventions that simultaneously target metacognitive skills and motivational enhancement, possibly using ecological momentary assessment (EMA) methods to track changes in real-time.

Clinicians and mental health professionals should consider incorporating metacognitive assessment and training into the standard care of individuals with schizophrenia. Psychoeducation programs that foster insight, self-monitoring, and reflective thinking may enhance intrinsic motivation and, in turn, functional capacity. Combining cognitive remediation with motivational interviewing techniques may also yield synergistic effects. Rehabilitation efforts should not only focus on symptom reduction but also aim to empower

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patients to set personal goals, develop self-efficacy, and engage meaningfully with their social environment. Lastly, community-based support systems that reinforce autonomy and offer structured opportunities for social and cognitive engagement can play a crucial role in sustaining therapeutic gains.

Authors' Contributions

All authors significantly contributed to this study.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

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Declaration of Interest

The authors report no conflict of interest.

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Ethical Considerations

In this study, to observe ethical considerations, participants were informed about the goals and importance of the research before the start of the study and participated in the research with informed consent.

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