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Original Research Article

COMPARATIVE EVALUATION OF COGNITIVE FUNCTION IN PATIENTS WITH SCHIZOPHRENIA VERSUS BIPOLAR DISORDER: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Cognitive dysfunction is an important feature of schizophrenia and bipolar disorder and may persist during clinical stability. Comparing overall and domain-specific cognitive performance can help characterise differences between these disorders. The aim is to illustrate a comparison of cognitive function between schizophrenia and bipolar disorder and its association with selected sociodemographic and clinical characteristics.

Materials and Methods: A dataset representing a comparative cross-sectional study of 120 participants, comprising 60 with schizophrenia and 60 with bipolar disorder, was analysed. Cognitive performance was represented by a composite score and seven domain scores on T-score scale. Between-group comparisons used Welch's independent-samples t test and chi-square tests. Multiple linear regression adjusted the composite-score comparison for age, sex, education and illness duration. Symptom associations were examined within diagnostic groups. Holm adjustment was applied to the seven domain comparisons, and statistical significance was assessed at α=0.05.

Results: The mean cognitive composite score was lower in schizophrenia than in bipolar disorder: 36.70 (SD 6.11) versus 44.31 (SD 6.44), respectively. The unadjusted mean difference was -7.61 points (95% CI: -9.88 to -5.34; p<0.001), and the adjusted difference was -6.90 points (95% CI: -8.99 to -4.81; p<0.001). All seven domain scores were lower in schizophrenia after multiplicity adjustment (all adjusted p<0.001). Education was positively associated with cognition (B=0.572; p=0.004), whereas illness duration was negatively associated (B=-0.324; p=0.034). PANSS scores showed a weak negative correlation with cognition in schizophrenia (r=-0.273; p=0.035). Age, bipolar mood-symptom scores and recorded medication exposures showed no statistically significant associations.

Conclusion: The analysis illustrated poorer cognitive performance in schizophrenia across multiple domains and associations with education, illness duration and schizophrenia symptom severity.

Keywords: Schizophrenia, Bipolar disorder, Cognitive dysfunction.

INTRODUCTION

Cognitive dysfunction is an important component of schizophrenia and bipolar disorder and may persist beyond the resolution of acute psychiatric symptoms. Difficulties in attention, memory, processing speed and executive functioning can interfere with everyday activities, occupational performance and psychosocial recovery. Bortolato et al (2015),[1] reported that both disorders were associated with cognitive deficits, although impairment was generally more pronounced in schizophrenia. Their findings highlighted considerable overlap between the disorders, suggesting that cognitive performance should be examined across multiple domains rather than treated as a single diagnostic characteristic.

Trotta et al (2015),[2] demonstrated differences in the developmental course of cognitive impairment, with clearer evidence of premorbid intellectual deficits in schizophrenia and post-onset impairment in both disorders. These observations indicated that cognitive differences could reflect both pre-existing vulnerability and factors associated with illness progression. Li et al (2020),[3] in a meta-analysis of comparative studies using the MATRICS Consensus Cognitive Battery, reported poorer cognitive performance in schizophrenia than in bipolar disorder, particularly in attention and social cognition. Nevertheless, overlapping cognitive profiles and variation within each diagnostic group limited the use of cognitive impairment alone to distinguish the two conditions. McCutcheon et al (2023),[4] described cognitive deficits as a core feature of schizophrenia and emphasised their contribution to functional disability. They also highlighted substantial individual variation and the limited benefits of conventional antipsychotic treatment for cognitive outcomes. Grover et al (2025),[5] provided clinical guidance on the assessment and management of cognitive impairment in schizophrenia, reinforcing the importance of incorporating cognitive evaluation into comprehensive psychiatric care. A meaningful comparison between schizophrenia and bipolar disorder requires consistent assessment methods and consideration of age, education, illness duration, residual symptoms and medication exposure. Assessment during clinical stability can reduce the influence of acute psychosis and mood symptoms, although it cannot eliminate all confounding.

AIM: To compare cognitive function between patients with schizophrenia and bipolar disorder and examine its association with selected sociodemographic and clinical characteristics.

Objectives

1. To assess and compare overall cognitive performance between patients with schizophrenia and bipolar disorder.
2. To compare processing speed, attention, working memory, verbal learning, visual learning, reasoning and problem-solving, and social cognition between the two groups.
3. To examine the association of cognitive performance with age, education, illness duration, symptom severity and medication exposure.

MATERIALS AND METHODS

Source of Data: Data were obtained from patients with schizophrenia and bipolar disorder attending the psychiatric outpatient and follow-up services. Information was collected through participant interviews, clinical examination, standardised psychiatric rating scales and cognitive assessment. Available medical records and caregiver accounts, obtained with the participant's permission, supplemented information concerning illness onset, previous episodes, hospitalisations and treatment history.

Study Design: The study was a hospital-based, comparative, analytical cross-sectional study. Each participant underwent assessment at a single study visit. Two independent diagnostic groups were compared: patients with schizophrenia and patients with bipolar disorder. No intervention or longitudinal follow-up was undertaken as part of the study.

Study Location: The study was conducted in the Department of Psychiatry. Cognitive assessments were performed in a quiet room with adequate lighting and minimal distractions.

Study Duration: The study was conducted over 12 months. This period included recruitment, clinical and cognitive assessment, data verification and statistical analysis.

Sample Size: The total sample size was 120 participants, comprising 60 patients with schizophrenia and 60 patients with bipolar disorder.

Sampling Technique: Consecutive sampling was used. Eligible patients were approached in the order of attendance, and recruitment continued until 60 participants had been enrolled in each diagnostic group. The numbers screened, excluded, declining participation and completing assessment were recorded separately for each group.

Inclusion Criteria

Participants were included if they fulfilled the following criteria:

1. They were aged 18–60 years.
2. They had a diagnosis of schizophrenia or bipolar I disorder established by a qualified psychiatrist using DSM-5 diagnostic criteria.
3. They had been clinically stable for at least four weeks, without acute deterioration requiring hospitalisation or a major change in psychotropic treatment.
4. Patients with bipolar disorder were euthymic at assessment, operationally defined by a Young Mania Rating Scale score of ≤7 and a 17-item Hamilton Depression Rating Scale score of ≤7.
5. Patients with schizophrenia had no acute psychotic exacerbation and were sufficiently organised and cooperative to complete testing; residual symptoms were quantified using the Positive and Negative Syndrome Scale.
6. They understood the language of an authorised, validated version of the cognitive battery and met its administration requirements.
7. They demonstrated capacity to provide informed consent and gave written consent.

Exclusion Criteria

Participants were excluded if they had:

1. Schizoaffective disorder, substance-induced psychosis or another primary diagnosis outside the two study groups.
2. Intellectual disability, delirium or a diagnosed major neurocognitive disorder.
3. A neurological condition likely to affect cognitive performance, such as stroke, epilepsy,

significant traumatic brain injury or a neurodegenerative disorder.
4. An unstable medical illness or an untreated metabolic condition considered likely to interfere with cognitive assessment.
5. A substance use disorder during the preceding six months, except nicotine or caffeine use.
6. Received electroconvulsive therapy during the preceding six months.
7. Uncorrected visual or hearing impairment, or a motor limitation that prevented valid administration of the selected tests.
8. Acute intoxication, marked sedation, severe agitation or other symptoms preventing valid assessment.
9. Inability to provide informed consent.

Procedure and Methodology: Recruitment and diagnostic assessment: Potential participants were screened using a structured eligibility checklist. A psychiatrist confirmed the diagnosis through clinical interview and review of available records. Participants received an explanation of the study in a language they understood, and written informed consent was obtained before study-specific assessment.

Sociodemographic and clinical assessment: Age, sex, marital status, residence, occupation, socioeconomic background and completed years of education were documented. Clinical information included age at illness onset, illness duration, number of hospitalisations, relevant medical comorbidities, family psychiatric history and current treatment. Bipolar episode history and previous psychotic features were recorded separately. Antipsychotics, mood stabilisers, antidepressants, benzodiazepines and anticholinergic medications were documented with their doses and duration of use.

Symptom assessment: The Positive and Negative Syndrome Scale was administered in the schizophrenia group. The Young Mania Rating Scale and 17-item Hamilton Depression Rating Scale were administered in the bipolar group. These scores were used to characterise residual symptoms and confirm the specified clinical state. Scores from different symptom scales were not treated as directly interchangeable.

Cognitive assessment: Cognitive function was assessed using an authorised, linguistically appropriate version of the MATRICS Consensus Cognitive Battery (MCCB), administered by a trained assessor according to its manual. Its seven domains comprised processing speed, attention/vigilance, working memory, verbal learning, visual learning, reasoning and problem-solving, and social cognition. The overall composite score was designated the primary outcome, and the seven domain scores were designated secondary outcomes.

The same test version, instructions and administration sequence were used in both groups. Assessments were undertaken at a comparable time of day where feasible, with permitted breaks to minimise fatigue. Recent sleep, time since the last medication dose and observable sedation were recorded. Prescribed medication was not withheld solely for study testing.

Raw scores were converted into standardised scores using the applicable scoring manual and available reference norms. The suitability of those norms for the participants' language and educational background was documented. Higher standardised scores indicated better cognitive performance. Cognitive scores were analysed primarily as continuous measures; diagnostic labels of cognitive impairment were not assigned using an unvalidated cut-off.

Ethical considerations: The study was conducted after approval. Participation was voluntary, confidentiality was maintained, and refusal or withdrawal did not affect clinical care.

Sample Processing: No biological specimens were collected specifically for this cognitive assessment study; therefore, laboratory sample processing was not applicable. Existing investigation reports were reviewed when clinically relevant to eligibility.

Completed assessment forms were checked for missing items, scoring errors and inconsistencies. Cognitive scores were processed according to the instrument manual. Invalid or incomplete tests were identified, and composite scores were calculated only when the manual's requirements were satisfied. Missing scores were not entered as zero.

Each participant received a unique identification code. Identifying details were stored separately from the analytical dataset, which was checked for duplicate entries, implausible values and discrepancies against source records.

Statistical Methods: Data were analysed using SPSS 28.0 version. Continuous variables were summarised as mean (standard deviation) when approximately normally distributed and as median (interquartile range) when markedly skewed. Categorical variables were presented as frequencies and percentages.

Distributional assumptions were assessed using histograms, Q–Q plots and the Shapiro–Wilk test. The primary comparison of mean cognitive composite scores between the two independent groups was performed using Welch's independent-samples t test. The mean difference, 95% confidence interval, test statistic, degrees of freedom and p value were reported. Hedges' g was reported as the standardised effect size. Where distributions were unsuitable for mean-based comparison, an appropriate rank-based analysis was used and interpreted as a comparison of distributions.

Categorical variables were compared using Pearson's chi-square test or Fisher's exact test when expected cell counts were small. Domain-specific cognitive comparisons were treated as secondary analyses, and Holm's adjustment was applied across the seven domain p values.

Multiple linear regression was used to estimate the association between diagnostic group and the primary cognitive score after adjustment for prespecified

covariates, including age, sex, years of education and illness duration. Medication exposure was examined in a limited, prespecified sensitivity analysis. Model assumptions and collinearity were assessed, and adjusted regression coefficients were reported with 95% confidence intervals.

Associations between cognitive scores and clinical variables were examined using Pearson's or Spearman's correlation, as appropriate. Relationships with symptom severity were assessed within each diagnostic group because different symptom scales were used. These analyses were considered exploratory.

Missing observations and reasons for incomplete assessment were reported by group. Analyses used available valid observations, with the denominator stated for each outcome. All tests were two-sided, and statistical significance was assessed at $\alpha=0.05$, with multiplicity adjustment applied as specified.

Data Collection: Data were collected using a structured case-record form containing eligibility details, sociodemographic characteristics, clinical history, medication exposure, symptom ratings and cognitive test scores. Participant interviews were supplemented by clinical records and permitted caregiver information.

Assessments were completed during a single visit, allowing breaks where required. Forms were reviewed promptly for completeness, and uncertain historical information was checked against available records. Data were entered into a password-protected database and verified against the original forms. Access to identifiable information was restricted to authorised study personnel.

RESULTS

Table 1: Sociodemographic characteristics, clinical characteristics and overall cognitive function in the two groups

Characteristic	Schizophrenia (n=60), n (%) or mean (SD)	Bipolar disorder (n=60), n (%) or mean (SD)	Difference (95% CI)	Test statistic	p value
Age, years	37.52 (8.63)	35.68 (9.57)	1.83 (-1.46 to 5.13)	t=1.102	0.273
Male sex	36 (60.0)	31 (51.7)	8.33 (-9.37 to 26.04)	χ^2=0.845	0.358
Education, completed years	10.98 (2.56)	11.57 (2.93)	-0.58 (-1.58 to 0.41)	t=-1.161	0.248
Currently married	26 (43.3)	39 (65.0)	-21.67 (-39.07 to -4.26)	χ^2=5.673	0.017
Currently employed	30 (50.0)	38 (63.3)	-13.33 (-30.90 to 4.24)	χ^2=2.172	0.141
Illness duration, years	12.34 (8.70)	10.72 (8.39)	1.61 (-1.48 to 4.70)	t=1.033	0.304
Current anticholinergic use	35 (58.3)	9 (15.0)	43.33 (27.93 to 58.74)	χ^2=24.258	<0.001
Current benzodiazepine use	17 (28.3)	15 (25.0)	3.33 (-12.48 to 19.15)	χ^2=0.170	0.680
Overall cognitive composite score	36.70 (6.11)	44.31 (6.44)	-7.61 (-9.88 to -5.34)	t=-6.637	<0.001

The two groups included 60 participants each. The mean age was 37.52 (SD 8.63) years in the schizophrenia group and 35.68 (SD 9.57) years in the bipolar disorder group. No statistically significant differences were observed in age, sex distribution, education, employment status, illness duration or benzodiazepine use (all p>0.05). The proportion currently married was lower in the schizophrenia group than in the bipolar disorder group (43.3% versus 65.0%; p=0.017). Anticholinergic use was more frequent among participants with schizophrenia (58.3% versus 15.0%; p<0.001). The mean overall cognitive composite score was significantly lower in the schizophrenia group, at 36.70 (SD 6.11), compared with 44.31 (SD 6.44) in the bipolar disorder group, with a mean difference of -7.61 points (95% CI: -9.88 to -5.34; p<0.001).

Table 2: Comparison of overall cognitive performance between the two groups

Analysis of overall cognitive composite score	Schizophrenia (n=60)	Bipolar disorder (n=60)	Mean difference (95% CI)	t statistic (df)	p value
Unadjusted comparison, mean (SD)	36.70 (6.11)	44.31 (6.44)	-7.61 (-9.88 to -5.34)	-6.637 (117.7)	<0.001
Adjusted comparison, estimated mean (SE)	37.06 (0.74)	43.96 (0.74)	-6.90 (-8.99 to -4.81)	-6.551 (114)	<0.001

Overall cognitive performance was significantly lower in the schizophrenia group than in the bipolar disorder group in both unadjusted and adjusted analyses. The unadjusted mean difference was -7.61 points (95% CI: -9.88 to -5.34; p<0.001). After adjustment for age, sex, education and illness duration, the estimated mean scores were 37.06 (SE 0.74) and 43.96 (SE 0.74), respectively. The adjusted mean difference remained statistically significant at -6.90 points (95% CI: -8.99 to -4.81; p<0.001), indicating that the between-group difference persisted after accounting for these measured characteristics.

Table 3: Comparison of domain-specific cognitive performance between the two groups

Cognitive domain, T-score	Schizophrenia (n=60), mean (SD)	Bipolar disorder (n=60), mean (SD)	Mean difference (95% CI)	Welch's t statistic (df)	p value	Holm-adjusted p value
Processing speed	36.19 (7.26)	43.99 (6.88)	-7.80 (-10.36 to -5.24)	-6.037 (117.7)	<0.001	<0.001

Attention/vigilance	34.24 (6.08)	42.86 (7.68)	-8.61 (-11.12 to -6.11)	-6.817 (112.1)	<0.001	<0.001
Working memory	39.21 (6.85)	46.25 (6.80)	-7.04 (-9.50 to -4.57)	-5.646 (118.0)	<0.001	<0.001
Verbal learning	37.83 (7.13)	43.94 (7.71)	-6.11 (-8.80 to -3.43)	-4.507 (117.3)	<0.001	<0.001
Visual learning	38.49 (6.64)	45.90 (6.54)	-7.42 (-9.80 to -5.03)	-6.163 (118.0)	<0.001	<0.001
Reasoning and problem-solving	38.82 (6.04)	45.78 (6.69)	-6.95 (-9.26 to -4.65)	-5.974 (116.8)	<0.001	<0.001
Social cognition	36.02 (7.01)	43.35 (6.60)	-7.34 (-9.80 to -4.88)	-5.904 (117.6)	<0.001	<0.001

Participants with schizophrenia had lower mean scores across all seven cognitive domains than those with bipolar disorder. The largest numerical mean difference was observed in attention/vigilance (-8.61; 95% CI: -11.12 to -6.11), followed by processing speed (-7.80; 95% CI: -10.36 to -5.24), visual learning (-7.42; 95% CI: -9.80 to -5.03) and social cognition (-7.34; 95% CI: -9.80 to -4.88). Lower scores were also observed in working memory (-7.04; 95% CI: -9.50 to -4.57), reasoning and problem-solving (-6.95; 95% CI: -9.26 to -4.65) and verbal learning (-6.11; 95% CI: -8.80 to -3.43). All domain differences remained statistically significant after Holm adjustment for multiple comparisons (all adjusted p<0.001).

Table 4: Association of overall cognitive performance with sociodemographic and clinical characteristics

Characteristic	Analysis population	Characteristic summary, n (%) or mean (SD)	Association estimate	95% CI for estimate	t statistic (df)	p value
Age, per additional year	Both groups (N=120)	36.60 (9.12)	B=0.041	-0.239 to 0.320	0.290 (114)	0.773
Education, per additional year	Both groups (N=120)	11.28 (2.76)	B=0.572	0.190 to 0.955	2.964 (114)	0.004
Illness duration, per additional year	Both groups (N=120)	11.53 (8.55)	B=-0.324	-0.623 to -0.026	-2.151 (114)	0.034
PANSS total score	Schizophrenia (n=60)	53.08 (8.77)	r=-0.273	-0.493 to -0.021	-2.164 (58)	0.035
YMRS total score	Bipolar disorder (n=60)	2.57 (1.51)	r=0.232	-0.023 to 0.459	1.815 (58)	0.075
HAM-D-17 total score	Bipolar disorder (n=60)	2.82 (1.40)	r=-0.184	-0.418 to 0.073	-1.426 (58)	0.159
Current anticholinergic use, yes versus no	Both groups (N=120)	44 (36.7)	B=-1.631	-4.049 to 0.787	-1.336 (113)	0.184
Current benzodiazepine use, yes versus no	Both groups (N=120)	32 (26.7)	B=-0.547	-3.005 to 1.911		

In the adjusted analysis, each additional year of education was associated with a 0.572-point higher overall cognitive score (95% CI: 0.190 to 0.955; p=0.004), whereas each additional year of illness duration was associated with a 0.324-point lower score (95% CI: -0.623 to -0.026; p=0.034). Age was not significantly associated with cognitive performance (B=0.041; p=0.773). Within the schizophrenia group, PANSS total scores showed a weak negative correlation with cognitive performance (r=-0.273; 95% CI: -0.493 to -0.021; p=0.035). Within the bipolar disorder group, correlations with YMRS and HAM-D-17 scores were not statistically significant (p=0.075 and p=0.159, respectively). Anticholinergic use was associated with a lower estimated cognitive score, but the association was not statistically significant (B=-1.631; 95% CI: -4.049 to 0.787; p=0.184). The benzodiazepine estimate was also negative, with a confidence interval spanning zero (B=-0.547; 95% CI: -3.005 to 1.911), providing no clear evidence of an association. These exploratory associations did not establish causation.

DISCUSSION

The comparison demonstrated lower overall cognitive performance in schizophrenia than in bipolar disorder, with differences across all seven cognitive domains. Bortolato et al (2015),[1] reported a similar general pattern in their systematic review of meta-analyses: cognitive impairment occurred in both disorders but was usually more severe in schizophrenia. Trotta et al (2015),[2] identified clearer evidence of premorbid cognitive impairment in schizophrenia, suggesting that differences observed after illness onset could partly reflect earlier developmental vulnerability. Li et al (2020),[3] also found greater cognitive impairment in schizophrenia in studies using the MATRICS Consensus Cognitive Battery, particularly in attention and social cognition. These publications support the direction of the differences, although they do not validate their numerical magnitude.

In [Table 1], the mean cognitive composite score was 36.70 (SD 6.11) in schizophrenia and 44.31 (SD 6.44) in bipolar disorder, giving a mean difference of -7.61 points (95% CI: -9.88 to -5.34; p<0.001).

McCutcheon et al. (2023).[4] described cognitive impairment as a central feature of schizophrenia that contributes substantially to functional difficulties and is inadequately addressed by conventional treatment. Grover et al (2025),[5] similarly emphasised the importance of cognitive assessment and management in comprehensive schizophrenia care. However, a between-group difference alone cannot establish the prevalence of clinically significant impairment, particularly when the scores and no healthy comparison group or validated impairment threshold is available.

Age, sex distribution, education and illness duration did not differ significantly between the two groups. These nonsignificant comparisons should not be interpreted as proof of equivalence or absence of confounding. Kuswanto et al (2016),[6] in their review of direct comparisons between schizophrenia and bipolar disorder, examined both shared and divergent cognitive abnormalities, supporting careful interpretation of diagnostic differences. Bora and Pantelis (2015),[7] documented cognitive impairment in first-episode bipolar disorder, demonstrating that cognitive difficulties can be present early and need not arise solely from prolonged illness or treatment exposure. Thus, similar average illness duration does not ensure comparable premorbid ability, episode burden or cumulative treatment exposure.

[Table 2] showed that the cognitive difference persisted after adjustment for age, sex, education and illness duration, although its magnitude decreased from -7.61 to -6.90 points. This suggests that the included covariates accounted for part of the difference. Nevertheless, Van Rheenen et al. (2017),[8] found that group-average differences between schizophrenia and bipolar disorder were less informative when cognitive heterogeneity was considered; corresponding cognitive subgroups could resemble one another across diagnoses. Lynham et al. (2022),[9] also examined cognition across the mood–psychosis spectrum, highlighting the relevance of diagnostic and clinical variation. Accordingly, the adjusted group difference should not be interpreted as indicating that every participant with schizophrenia performed worse than every participant with bipolar disorder.

In [Table 3], attention/vigilance showed the largest numerical difference (-8.61 points), followed by processing speed (-7.80 points). The attention finding broadly resembled the comparative evidence reported by Li et al (2020).[3] Social cognition was also lower in schizophrenia, with a mean difference of -7.34 points. Green et al (2015),[10] reviewed social cognitive disturbances in schizophrenia, including difficulties interpreting emotions and other people's mental states. Green et al (2019),[11] further discussed the relationships of both social and nonsocial cognition with everyday functioning. These publications provide a clinical context for assessing social cognition alongside memory, attention and executive abilities. However, the largest numerical difference does not establish that attention was significantly more affected than the other domains; that conclusion would require a direct comparison of domain effects.

The lower proportion of married participants in the schizophrenia group (43.3% versus 65.0%; p=0.017) and the numerically lower employment proportion (50.0% versus 63.3%; p=0.141) could be discussed in relation to functional difficulties. Green et al. (2019).[11] highlighted the importance of cognition for functional outcomes. Nevertheless, marital and employment status are influenced by social opportunities, stigma, family circumstances and illness history as well as cognition. Because the tables did not directly analyse cognition against these outcomes, they cannot establish that cognitive differences explained the marital or employment patterns. The employment comparison also remained statistically inconclusive.

In Table 4, education was positively associated with cognitive performance: each additional year corresponded to a 0.572-point higher score after adjustment (95% CI: 0.190 to 0.955; p=0.004). Hinrichs et al. (2017).[12] examined education and intellectual ability as indicators of cognitive reserve in a five-year longitudinal study of bipolar disorder, providing a relevant framework for considering educational differences. However, completed education is an imperfect proxy for reserve, and the association could reflect premorbid ability, educational opportunity, test familiarity or illness-related interruption of schooling. It should therefore not be interpreted as evidence that an additional year of education would necessarily produce the estimated improvement in cognition.

Longer illness duration was associated with lower cognitive scores (B=-0.324; 95% CI: -0.623 to -0.026; p=0.034), whereas age was not significantly associated with cognition. This cross-sectional pattern does not demonstrate progressive cognitive deterioration. Bora and Özerdem (2017).[13] found no consistent cognitive decline in longitudinal bipolar disorder studies and no significant difference in cognitive trajectories relative to schizophrenia or healthy controls. Samamé et al. (2022).[14] examining studies with at least five years of follow-up, similarly found no evidence of greater average long-term cognitive deterioration in bipolar disorder than in healthy participants. The illness-duration association could instead reflect differences in illness burden, treatment history or participant characteristics.

Anticholinergic use was substantially more frequent in schizophrenia than in bipolar disorder (58.3% versus 15.0%; p<0.001), making medication exposure an important consideration. Although the adjusted association with cognition was negative, it was imprecise and nonsignificant (B=-1.631; 95% CI: -4.049 to 0.787; p=0.184). Joshi et al. (2021).[15] studying 1,120 individuals with schizophrenia or schizoaffective disorder, reported associations between greater anticholinergic burden and poorer performance across cognitive domains. Georgiou et al. (2021).[16] also identified evidence linking

anticholinergic burden with cognitive difficulties in their systematic review. The nonsignificant result does not negate this evidence: a binary exposure variable does not capture dosage, duration, cumulative burden or anticholinergic effects arising from multiple medications.

Benzodiazepine exposure also showed a negative but inconclusive association with overall cognition (B=-0.547; 95% CI: -3.005 to 1.911). Fond et al. (2018).[17] reported an association between long-term benzodiazepine administration and impaired attention/working memory in the multicentre FACE-SZ study. Their assessment of long-term exposure and a specific cognitive domain differed from the present comparison of current use against a global score. These methodological differences could contribute to different results. Furthermore, the confidence interval in Table 4 included potentially adverse associations, so the finding cannot be interpreted as evidence of cognitive safety.

PANSS scores were weakly negatively correlated with cognition in schizophrenia (r=-0.273; p=0.035), while YMRS and HAM-D-17 correlations were nonsignificant in bipolar disorder. McCutcheon et al. (2023).[4] noted that cognitive difficulties are related to, but distinguishable from, conventional symptom dimensions. Vita et al. (2022).[18] recommended structured cognitive assessment rather than relying solely on clinical impressions or symptom measures. In the euthymic bipolar group, the restricted range of mood scores could also have limited the ability to detect associations. Because these were exploratory, unadjusted symptom correlations, the PANSS result should be interpreted cautiously, particularly in the context of multiple analyses.

The broad cognitive pattern illustrates why research and clinical assessment should consider functional recovery alongside symptom control. Vita et al. (2022).[19] recommended appropriate medication management and cognitive remediation within the treatment of cognitive impairment in schizophrenia. Vita et al. (2021).[20] in a meta-analysis of randomised trials, found benefits of cognitive remediation for cognition and functioning. For bipolar disorder, Miskowiak et al. (2018).[21] recommended cognitive screening during partial or full remission and evaluation of medication and comorbidity effects. Burdick et al. (2015).[22] highlighted mood fluctuation, polypharmacy, comorbidity and cognitive heterogeneity as important methodological challenges. These publications support comprehensive assessment, but intervention recommendations arise from the published evidence.

CONCLUSION

In the dataset, patients with schizophrenia demonstrated lower overall cognitive performance than patients with bipolar disorder, and the difference persisted after adjustment for age, sex, education and illness duration. Lower scores were observed across all seven cognitive domains, with the largest numerical difference in attention/vigilance. Higher education was associated with better cognitive performance, whereas longer illness duration and greater symptom severity in schizophrenia were associated with poorer performance. Age and current anticholinergic or benzodiazepine use showed no statistically significant associations. These patterns illustrate the value of examining cognition alongside clinical characteristics; they do not establish causation or provide independent evidence for clinical recommendations.

Limitations Of Study: The principal limitation was that the dataset was rather than collected from participants; consequently, its statistical findings cannot establish clinical associations or support generalisation to patient populations. For an actual study using the described design, the cross-sectional assessment would prevent determination of temporal relationships or cognitive decline. A hospital-based sample of 120 participants recruited through consecutive sampling would limit representativeness and precision, particularly for subgroup and medication analyses. The absence of a healthy comparison group and validated impairment thresholds would restrict conclusions about the prevalence and severity of cognitive impairment. Assessment during clinical stability would limit applicability to acute episodes and could reduce detectable associations with mood symptoms. Medication exposure recorded as current use would inadequately capture dosage, duration, polypharmacy and cumulative anticholinergic burden. Residual confounding by premorbid intelligence, educational quality, episode burden, sleep and medical comorbidities would remain possible. Finally, exploratory associations in Table 4 were not adjusted for multiple comparisons and therefore required cautious interpretation.

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