International Journal of Medicine and Medical Research

ISSN 2413-6077, e-ISSN 2414-9985
Journal homepage: https://ijmr.com.ua/
Volume 12, No. 1. 2026
Article's History: Received: 27.12.2025; Revised: 16.04.2026; Accepted: 26.05.2026; Published: 29.05.2026.

UDC 616.153.018.5:547.831.9:616.89
DOI: 10.63341/ijmmr/1.2026.06

Plasma levels of quinolinic acid and kynurenic acid in patients with major mental illnesses

Sheu Rahamon*
PhD, Lecturer
College of Medicine, University of Ibadan
200285, Queen Elizabeth II Rd., Agodi, Ibadan, Nigeria
https://orcid.org/0000-0003-0305-5026

Sofiyat Bello
Master, Postgraduate Student
College of Medicine, University of Ibadan
200285, Queen Elizabeth II Rd., Agodi, Ibadan, Nigeria
https://orcid.org/0009-0008-7780-8020

Abdulrahamon Sikirullah
Master, Postgraduate Student
College of Medicine, University of Ibadan
200285, Queen Elizabeth II Rd., Agodi, Ibadan, Nigeria
https://orcid.org/0009-0001-5579-8414

Oluremi Oladele
Bachelor, Lecturer, Consultant Psychiatrist
College of Medicine, University of Ibadan
200285, Queen Elizabeth II Rd., Agodi, Ibadan, Nigeria
https://orcid.org/0009-0001-0472-4714

Victor Lasebikan
PhD, Professor, Consultant Psychiatrist
College of Medicine, University of Ibadan
200285, Queen Elizabeth II Rd., Agodi, Ibadan, Nigeria
https://orcid.org/0000-0001-5834-9190

Abstract. Clarifying antipsychotic effects on kynurenine-pathway metabolites may improve understanding of their role in major mental illnesses. Therefore, this study aimed to assess plasma quinolinic and kynurenic acid levels in patients with major mental illnesses. 85 adults comprising 55 treatment-experienced patients with major mental illnesses; schizophrenia, bipolar and depression, and 30 controls were enrolled into this study. The plasma levels of quinolinic acid and kynurenic acid were determined using Enzyme Linked Immunosorbent Assay. Plasma quinolinic acid level was significantly lower in patients with major mental illnesses (14.52 (7.16-48.24) mg/mL) compared with the controls (157.79 (68.64-464.05) mg/mL). In contrast, plasma kynurenic acid level was higher in patients with major mental illnesses (243.96 (210.65-283.05) μmol/L) compared with the controls (215.36 (185.67-243.65) μmol/L). After stratification into groups, patients with schizophrenia (22.86 (7.62-53.14) mg/mL), bipolar (15.34 (8.37-48.21) mg/mL)

Suggest Citation:
Rahamon S, Bello S, Sikirullah A, Oladele O, Lasebikan V. Plasma levels of quinolinic acid and kynurenic acid in patients with major mental illnesses. Int J Med Med Res. 2026;12(1):6–13. DOI: 10.63341/ijmmr/1.2026.06

*Corresponding author (*sk.rahamon@ui.edu.ng*)

and depression (6.18 (2.66-43.05) mg/mL) had significantly lower quinolinic acid levels compared with the controls (157.79 (68.64-464.05) mg/mL). However, kynurenic acid levels were only significantly elevated in patients with schizophrenia (247.0 (208.34-290.22 μmol/L) and bipolar (243.20 (212.85-291.12) μmol/L) compared with the controls (215.36 (185.67-243.65) μmol/L). The plasma levels of quinolinic acid and kynurenic acid did not differ significantly among patients with schizophrenia, bipolar disorder, and depression. Treated patients with major mental illnesses exhibited a kynurenine metabolite profile indicative of reduced neurotoxicity and enhanced neuroprotection. These metabolites may serve as potential biomarkers for monitoring treatment effects

Keywords: antipsychotics; inflammation; tryptophan metabolism; mental health disorders; neuroprotection

Introduction

Major mental illnesses (MMI), including schizophrenia, bipolar disorder, and depression, are complex and heterogeneous conditions with substantial personal, social and economic burdens worldwide. Aetiology extends beyond neurotransmitter as it involves abnormalities in hypothalamic-pituitary-adrenal axis regulation, alongside epigenetic modifications, neuroinflammatory processes, and altered kynurenine pathway metabolism. Adequate understanding of the biological pathways that integrate these systems is clinically essential for improving diagnosis, monitoring treatment response, and advancing personalised therapy. Among these pathways, alterations in tryptophan metabolism and downstream neuroactive metabolites continue to gain growing attention as possible contributors to symptom progression and therapeutic outcomes.

Inflammation has been shown to play a significant role in the clinical progression of neuropsychiatric disorders. Elevated levels of proinflammatory cytokines, inflammatory mediators, acute phase proteins, cell sensors, and adhesion molecules were found in a study by K.S. Akinlade *et al.* [1] in the blood and cerebrospinal fluid of patients with MMI. The kynurenine pathway is a biological interface linking immune activation to neurochemical and behavioural disturbances in psychiatric disorders as the pathway generates multiple neuroactive metabolites. Approximately 95-99% of tryptophan is metabolized through the kynurenine pathway, while the remainder is used for protein synthesis or converted into serotonin and melatonin [2]. Several studies have examined alterations in kynurenine pathway metabolites in schizophrenia. M. Marković *et al.* [3] assessed plasma levels of tryptophan, kynurenine, and kynurenic acid (KA) during both the acute and remission phases of schizophrenia. Their findings showed that kynurenine and KA were consistently reduced in affected individuals, regardless of disease stage, antipsychotic therapy, episode number, or illness duration. In a related study, C. Kuuskmäe *et al.* [4] evaluated kynurenine pathway dynamics in schizophrenia spectrum disorders and similarly reported decreased KA and quinolinic acid (QA), noting that antipsychotic treatment diminished the magnitude of pathway alterations over time.

M. Li *et al.* [5] analysed serum and urine kynurenine metabolites in drug-naïve patients with first-episode schizophrenia and observed an elevated QA/KA ratio, indicating a shift toward neurotoxic dominance alongside reduced concentrations of neuroprotective metabolites. In patients with depression, Y. Pan *et al.* [6] examined the association between cognitive deficits and kynurenine-pathway metabolites in individuals with depression. They reported reduced plasma concentrations of kynurenine and QA, alongside a marked increase in KA, when compared with healthy controls. In a related study, R. Colle *et al.* [7] also found decreased circulating levels of kynurenine, KA, and picolinic acid in depressed patients, and further noted that KA levels rose significantly after treatment with antidepressant medications.

K. Hebbrecht *et al.* [8] investigated how alterations in the kynurenine pathway relate to cognitive performance across various mood states in individuals with bipolar disorder. Their findings showed that plasma KA concentrations were markedly reduced in patients compared with healthy controls, and lower KA levels correlated with poorer overall cognitive outcomes. More recently, E. Yavuz Ataşlar & K. Altınbaş [9] assessed the predictive value of circadian rhythm markers and tryptophan-kynurenine pathway indices in bipolar disorder. They found that both patients with bipolar disorder and their siblings exhibited an increased kynurenine/tryptophan ratio. In a study investigating if individual catabolites could serve as diagnostic biomarkers, M. Brum *et al.* [10] reported that plasma concentrations of tryptophan, kynurenine, KA, and QA were reduced in the overall patient cohort (schizophrenia, bipolar disorder and depression) compared with healthy controls, and that the pattern was largely driven by differences observed in individuals with bipolar disorder. They however, did not observe any significant difference among the specific diagnostic groups.

Findings across studies have been inconsistent, particularly regarding KA and QA concentrations, due to factors like study populations, ethnicity, diagnostic thresholds, medication exposure, and methodologies. Few studies have explored these metabolites in African populations, despite evidence that genetic, environmental, dietary, and treatment factors may affect immune-metabolic pathways. Given the differences in antipsychotic responses across populations, there is a need for population-specific investigations. Despite the clinical burden of mental illnesses in Nigeria, data on kynurenine-pathway metabolites during antipsychotic therapy remain limited. The purpose of this study was to assess alterations in specific metabolites of the kynurenine pathway in patients with major mental disorders receiving treatment, in order to address this knowledge gap.

Materials and Methods

This study was conducted in full compliance with the ethical principles of the Declaration of Helsinki [11]. The study was conducted at the University of Ibadan and approved by the University of Ibadan/University College Hospital (UI/UCH) Joint Ethics Committee (UI/EC/23/0456) signed on 05 August 2024. Written informed consent was obtained from all the study participants, and for individuals with impaired decision-making capacity, assent was secured from appropriate relatives or guardians. The study was conducted between August 2024 and January 2025. This study enrolled 85 participants, comprising 33 patients with schizophrenia, 12 with bipolar disorder, 10 with depression, and 30 apparently healthy controls. Considering the sex of the study participants, there were 15 males and 15 females among the controls, while the MMI group had 28 males and 27 females. Patients were recruited from the Psychiatry Department of the University College Hospital, Ibadan, and New World Specialist Hospital, Molete, Ibadan, Nigeria. Control participants were recruited from the Ibadan metropolis and were certified to be free of psychiatric disorders by a Consultant Psychiatrist.

Diagnoses of schizophrenia, bipolar disorder, and depression were established using the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria [12]. Exclusion criteria included current substance abuse or dependence, autoimmune disorders or steroid therapy, diabetes mellitus, and severe or unstable medical conditions. A short structured questionnaire was used to obtain information on demography and clinical history. The questions included age of the participant at the time of enrollment, sex (male or female), highest level of education attained, marital status (single, married, separated/divorced, widowed), employment status (employed, unemployed, student, retired), duration of illness since initial diagnosis (in years), family history of psychiatric disorders particularly first-degree relatives, current medications and duration of treatment, including antipsychotics, mood stabilizers, and antidepressants, history of substance abuse, and history of comorbid medical conditions, including autoimmune disorders, diabetes mellitus, or other chronic illnesses. Therefater, venous blood samples (5 mL) was collected from each study participant into heparin-containing sample bottles and plasma was obtained as appropriate. The plasma samples were thereafter, stored at -20°C until analysed. Plasma levels of KA and QA were determined using sandwich ELISA following the manufacturer's instruction (Melsin Medical Co., China).

Data were analysed using the Statistical Package for Social Sciences version 27.0 and Graphpad version 9.0 software package. Normal distribution of the data was assessed using Shapiro-Wilk test and Kolmogorov-Smirnov test. Differences in the mean values of age was determined using the Student's t-test, while the Kruskal-Wallis H and Man Whitney *U* tests were used to determine the differences in the median values of each variable between the groups. Chi-square was used to determine association between sex and various cohorts. Thereafter, a rank-based one-way analysis of covariance was conducted to compare the different groups on the dependent variable, adjusting for age as a covariate. The dependent variable was rank-transformed due to violations of normality assumptions. The correlation between the variables was assessed using Spearman's Rho correlation. P-values below 0.05 (2-tailed) were regarded as statistically significant. The results are presented as mean ± standard deviation, median (interquartile range), and in box plots, where applicable.

It is important to note that the statistical power of this study was limited by the small and unequal sample sizes in some diagnostic subgroups. This reduces the ability to detect small to moderate effect sizes and may affect the stability of variance estimates. Also, the absence of baseline QA and KA levels and medication history prevent causal inference regarding the effects of antipsychotic therapy on selected metabolites of the kynurenine pathway. Therefore, findings from this study should be interpreted as preliminary and require validation in larger, well-powered longitudinal studies involving drug-naive cohorts.

Results and Discussion

The mean ages of the patients with MMI and controls were 36.38 ± 11.65 years and 29.40 ± 7.41 years, respectively. There was significant difference between the mean ages of MMI compared with the controls (p = 0.001). There was no significant association between the sex and the study cohorts (p = 0.936). As shown in Table 1, plasma QA level was significantly lower in patients with MMI compared with the controls. In contrast, the plasma KA level was significantly higher in patients with MMI compared with the controls. Similarly, the median KA/QA ratio was significantly higher in MMI group compared with the controls.

Table 1. Age, sex and plasma levels of QA and KA in patients with MMI and control

Parameters	MMI (n = 55)	Controls (n = 30)	p-value
Age (years)	36.38 ± 11.65	29.40 ± 7.41	0.001*
Sex Male Female	28 (50.9%) 27 (49.1%)	15 (50.0%) 15 (50.0%)	0.936†
QA (mg/mL)	14.52 (7.16-48.24)	157.79 (68.64-464.05)	0.000*
KA (μmol/L)	243.96 (210.65-283.05)	215.36 (185.67-243.65)	0.004*

Continued Table 3

Parameters	MMI (n = 55)	Controls (n = 30)	p-value
KA/QA ratio	17.51 (5.46-44.21)	1.38 (0.25-2.47)	0.000*

Note: *significant at $p < 0.05$; †p-value from chi-square
Source: compiled by the authors

Since age was significantly different between the two groups, a rank-based analysis of covariance was performed to assess group differences in plasma QA, KA, and the KA/QA ratio in MMI group compared with the controls while adjusting for age. For QA, KA and KA/QA ratio, the overall models were statistically significant ($p = 0.000$, $p = 0.010$ and $p = 0.000$, respectively). As shown in Table 2, QA ranks differed significantly in patients with MMI and controls, after age adjustment. A similar pattern was observed for KA and KA/QA ratio. However, age showed insignificant independent associations with QA, KA and KA/QA ratio. Across all analyses, Bonferroni-adjusted pairwise comparisons revealed significant differences in QA, KA and KA/QA ratio between the two groups.

Table 2. Age-adjusted differences in the parameters in the study participants

Outcome variable	Source of variation	F (df = 1.82)	p-value	Partial η^2
QA rank	Diagnostic group	69.375	0.000*	0.458
	Age (covariate)	1.469	0.229	0.018
KA rank	Diagnostic group	7.255	0.009*	0.081
	Age (covariate)	0.428	0.515	0.005
KA/QA ratio rank	Diagnostic group	71.542	0.000*	0.466
	Age (covariate)	2.273	0.135	0.027

Note: *significant at $p < 0.05$. Rank-based Analysis of Covariance was used because QA and KA were non-normally distributed. Diagnostic groups included MMI and controls
Source: compiled by the authors

As shown in Figure 1, the plasma levels of QA were significantly lower in patients with schizophrenia, bipolar disorder and depression compared to controls (p-values; <0.0001, <0.0001, and <0.0001, respectively). In contrast, plasma KA levels were significantly higher in patients with schizophrenia, and bipolar compared to controls (p-values; 0.0030 and 0.0296, respectively) but the level was similar in patients with depression and the controls. Similarly, the ratio of KA to QA was significantly higher in patients with schizophrenia, bipolar and depression compared to controls (p-values; <0.0001, <0.0001, and <0.0001, respectively).

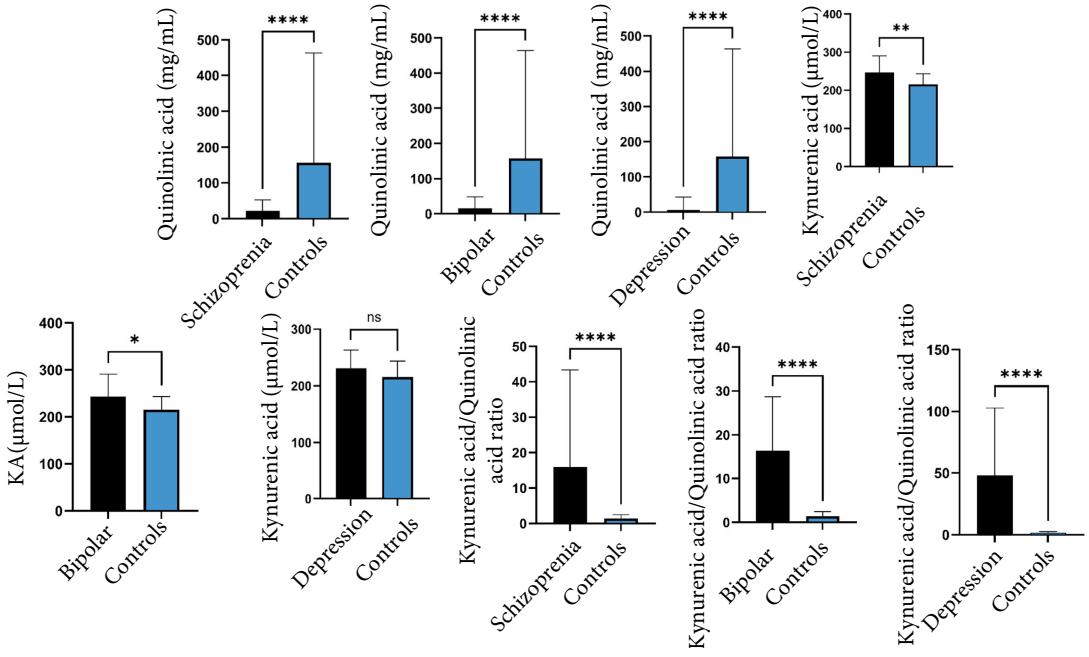

Figure 1. Plasma levels of QA, KA and KA/QA ratio in patients
Source: compiled by the authors

Table 3 presented the plasma concentrations of KA and QA in patients with schizophrenia, bipolar disorder, and depression. While significant age differences were observed between the three groups, the plasma levels of QA, KA, and the KA/QA ratio were comparable across all groups.

Since age was significantly different among the patients groups, a rank-based analysis of covariance was performed to assess group differences in plasma QA, KA, and the KA/QA ratio while adjusting for age. As shown in Table 4, after age adjustment, QA ranks did not differ significantly among patients with schizophrenia, bipolar disorder, and depression ($F(2.51) = 0.573$, $p = 0.568$). A similar pattern was observed for KA. For the KA/QA ratio, the overall model was statistically significant; however, the diagnostic group effect remained non-significant, while age showed a significant independent association with the ratio ($p = 0.006$). Across all analyses, Bonferroni-adjusted pairwise comparisons revealed no significant differences between diagnostic groups.

Table 3. Age, sex and plasma levels of QA and KA in patients with MMI and control

Parameters	Schizophrenia	Bipolar	Depression	p-value
Age (years)	40.42 ± 11.43	30.08 ± 10.93	30.60 ± 7.29	0.000*
Sex Male Female	 17 (51.5%) 16 (48.5%)	 5 (41.7%) 7 (58.3%)	 6 (60.0%) 4 (40.0%)	0.689†
QA (mg/mL)	22.86 (7.62-53.14)	15.34 (8.37-48.21)	6.18 (2.66-43.05)	0.422
KA (μmol/L)	247.0 (208.34-290.22)	243.20 (212.85-291.12)	230.76 (182.06-263.46)	0.448
KA/QA ratio	15.99 (6.41-43.38)	16.47 (4.38-28.71)	48.03 (3.45-102.67)	0.482

Note: *significant at $p < 0.05$, †p-value from chi-square
Source: compiled by the authors

Table 4. Age-adjusted differences in the parameters in schizophrenia, bipolar disorder, and depression patients

Outcome variable	Source of variation	F (df = 2.51)	p-value	Partial η^2
QA rank	Diagnostic group	0.573	0.568	0.022
	Age (covariate)	3.784	0.057	0.069
KA rank	Diagnostic group	0.641	0.531	0.025
	Age (covariate)	0.277	0.601	0.005
KA/QA ratio rank	Diagnostic group	1.324	0.275	0.049
	Age (covariate)	8.294	0.006*	0.140

Note: *significant at $p < 0.05$. Rank-based Analysis of Covariance was used because QUIN and KYNA were non-normally distributed. Diagnostic groups included schizophrenia, bipolar disorder, and depression
Source: compiled by the authors

Correlations between plasma QA and KA levels in patients with MMI and controls are shown in Table 5. Plasma QA level had no significant correlation with plasma KA level in patients with MMI and controls.

Table 5. Correlation between plasma levels of QA and KA in patients with MMI and controls

Parameters	MMI		Controls	
	r-value	p-value	r-value	p-value
QA vs KA	0.179	0.191	0.162	0.391

Source: compiled by the authors

The pathophysiology of schizophrenia, bipolar disorder, and depression encompasses various levels of neuroimmune interactions, from the hypothalamic-pituitary-adrenal axis to elevated pro-inflammatory cytokines that interfere with neuroplasticity, neurogenesis, and the regulation of neurotransmission in different brain regions. Considering the complex crosstalk between neurotransmitter systems, neuroplasticity, and immune function, the kynurenine pathway represents a promising target for investigation [13]. QA, a known N-methyl-D-aspartate receptor agonist, is a known neurotoxic metabolite implicated in neuroinflammatory processes in MMIs such as major depressive disorder [14]. In this study, it was observed that plasma QA levels were significantly lower in MMI patients compared to healthy controls, even after adjusting for age. Although this observation supported the report of K. Skorobogatov *et al.* [15]

which showed that serum QA level was significantly lower in combined patients with schizophrenia and bipolar disorder compared to controls, it contrasts with other studies that linked elevated QA level to heightened neuroinflammation and excitotoxicity in patients with MMI [16]. These discrepancies could be due to the heterogeneity in patient characteristics, particularly the use of antipsychotic medications. In this study, all the MMI patients were on antipsychotic treatment at the time of sample collection, although medication type, dosage, or treatment duration could not be standardised or controlled for due to limited clinical documentation. S.R. Patlola *et al.* [17] reported that certain antipsychotics such as risperidone, an atypical antipsychotics, downregulate interleukin-6 and tumour necrosis factor-alpha levels in patients with chronic schizophrenia. They also reported that the duration of illness influences the extent of cytokine alteration. This antipsychotic-associated modulation of inflammatory pathways may impact kynurenine metabolism. While antipsychotic therapy may play a role, the absence of baseline QA levels precludes definitive conclusions about causality as other factors such as illness chronicity, nutritional status, or immune function may also play important roles. Taken this together with the unexpectedly elevated QA levels observed in the control group, these findings underscore the need for large population prospective studies that will explore the influence of diet, systemic inflammation, and assay variability on metabolites of the kynurenine pathway in patients with MMI.

Changes in peripheral metabolites of the kynurenine pathway, as reported by M. Brum *et al.* [10], are phase-specific and specific to disorders in patients with major depressive disorder, schizophrenia, and bipolar disorder. In this study, even after adjusting for age, the plasma levels of QA were insignificantly different in patients with schizophrenia, bipolar and depression. This observation corroborates the report of F. Cathomas *et al.* [18] which showed that the levels of QA in patients with schizophrenia and depression are similar. Observation from this study could indicate that the pattern of change in QA level in patients with MMI on antipsychotics is similar irrespective of the illness. KA, an endogenous antagonist of N-methyl-D-aspartate and α-7 nicotinic receptors, was found to be significantly elevated in MMI patients particularly, those with schizophrenia and bipolar disorder compared to controls. These observations are consistent with the results of a study by A. Trepci *et al.* [19], which reported elevated cerebrospinal fluid levels in these disorders. A.M. Myint *et al.* [20] reported an upward trend in KA after six weeks of antipsychotic therapy, which may reflect a shift in the kynurenine pathway toward neuroprotection. While observation from this study is consistent with the possibility of such a shift, the lack of baseline or drug-naïve comparisons limits the strength of this inference. A study by F. Cathomas *et al.* [18] reported that plasma KA levels in patients with schizophrenia and depression were similar. Similar trend was observed in this study as the plasma levels of KA were insignificantly different in patients with schizophrenia, bipolar disorder and depression. This observation further supports the earlier suggestion that the pattern of change in kynurenine metabolites during antipsychotics therapy is similar irrespective of the illness.

While KA levels were elevated and QA levels reduced in MMI patients relative to controls, were observed a significantly higher KA/QA ratio across all patient subgroups compared to controls. The KYNA/QUIN ratio has been proposed as a neuroprotective index, largely reflecting the balance between excitotoxic and neuroprotective arms of the kynurenine pathway [14]. Previous reports on KA/QA ratio in patients with MMI are inconsistent. B.E. Wurfel *et al.* [21] reported decreased KA/QA ratio in bipolar disorder and depression, while W. Marx *et al.* [22] observed no significant differences in schizophrenia. This variability may stem from differences in sample types, analytical methods, clinical status, and treatment exposure. The absence of significant differences in the KA/QA ratio, similar to the patterns observed for QA and KA levels, across schizophrenia, bipolar disorder, and depression groups further suggest a shared metabolic profile in treated patients with MMI.

In summary, drug-experienced patients with MMI exhibited significantly lower plasma QA levels and higher KA levels compared with healthy controls, resulting in a markedly increased KA/QA ratio, even after adjusting for age. These alterations were consistent across schizophrenia, bipolar disorder, and depression, with no significant differences among diagnostic subgroups. The findings suggest a shift in kynurenine pathway metabolism towards reduced neurotoxicity and enhanced neuroprotection in drug-experienced patients.

Conclusions

This study demonstrated that age-adjusted plasma QA levels were markedly lower in patients with MMIs than in controls (median 14.52 (7.16-48.24) vs 157.79 (68.64-464.05) mg/mL; $F(1.82) = 69.375$, $p < 0.001$, partial $\eta^2 = 0.458$). In contrast, plasma KA levels were significantly higher in patients with MMI (243.96 (210.65-283.05) vs 215.36 (185.67-243.65) μmol/L; $F(1.82) = 7.255$, $p = 0.009$), resulting in a substantially elevated KA/QA ratio (17.51 (5.46-44.21) vs 1.38 (0.25-2.47); $F(1.82) = 71.542$, $p < 0.001$). Subgroup analyses showed that patients with schizophrenia, bipolar disorder, and depression all exhibited significantly reduced QA levels and increased KA/QA ratio compared with controls, while age-adjusted differences in the parameters and their ratio were not significant among the three diagnostic categories. These observations suggested a shared kynurenine metabolic profile across MMI in drug-experienced patients. Furthermore, plasma QA and KA levels were not significantly correlated in either the patients or controls, indicating independent regulation of these metabolites. Overall, the observed pattern of reduced QA, elevated KA, and increased KA/QA ratio is indicative of a shift towards reduced excitotoxicity and enhanced neuroprotection in treated MMI patients, potentially reflecting the immunomodulatory and anti-inflammatory effects of antipsychotic

therapy. These metabolites, particularly the KA/QA ratio, may therefore serve as candidate peripheral biomarkers for monitoring treatment response in patients with MMI. Future research prioritising large-scale, longitudinal studies incorporating drug-naïve patients, standardised medication data, and baseline measurements are suggested to clarify causal relationships. Additionally, integrating dietary, inflammatory, and genetic factors will be essential for a more comprehensive understanding of the dynamics of the kynurenine pathway in MMI.

Acknowledgements

The nurses and other clinical staff of the New World Specialist Hospital, Ibadan and University College Hospital, Ibadan are appreciated for their support during the study.

Funding

The authors received no specific funding for this work.

Conflict of Interest

The authors have no competing interests to declare.

References

[1] Akinlade KS, Adedokun KA, Rahamon SK, Lasebikan VO. Elevated levels of visfatin and fetuin-A in patients with major mental disorders. Arch Bas App Med. 2020;8(1):49–53.
[2] Badawy AA. Kynurenine pathway and human systems. Exp Gerontol. 2020;129:110770. DOI: 10.1016/j.exger.2019.110770
[3] Marković M, Petronijević N, Stašević M, Stašević Karličić I, Velimirović M, Stojković T, et al. Decreased plasma levels of kynurenine and KA in previously treated and first-episode antipsychotic-naive schizophrenia patients. Cells. 2023;12(24):2814. DOI: 10.3390/cells12242814
[4] Kuuskmäe C, Philips MA, Kilk K, Haring L, Kangro R, Seppo I, et al. Kynurenine pathway dynamics in patients with schizophrenia spectrum disorders across the disease trajectory. Psychiatry Res. 2023;328:115423. DOI: 10.1016/j.psychres.2023.115423
[5] Li M, Wu Y, Xu Y, Huang X, Gao K, Hu N, et al. Peripheral tryptophan-kynurenine pathway dysfunction in first-episode schizophrenia. Sci Rep. 2025;15(1):2432. DOI: 10.1038/s41598-025-86390-4
[6] Pan Y, Xu P, Sun X. Associations between Kynurenine pathway metabolites and cognitive dysfunction in major depressive disorder. PLoS One. 2025;20(8):e0328886. DOI: 10.1371/journal.pone.0328886
[7] Colle R, Chappell K, El Asmar K, Fève B, Chanson P, David DJ, et al. Plasma kynurenine pathway metabolite levels increase in depressed patients after antidepressant treatment. Brain Behav Immun. 2025;129:92–9. DOI: 10.1016/j.bbi.2025.05.025
[8] Hebbrecht K, Morrens M, Giltay EJ, van Nuijs ALN, Sabbe B, van den Ameele S. The role of kynurenines in cognitive dysfunction in bipolar disorder. Neuropsychobiology. 2022;81(3):184–91. DOI: 10.1159/000520152
[9] Yavuz Ataşlar E, Altınbaş K. A comprehensive examination of circadian rhythm and tryptophan pathway parameters: Assessing their role in predicting bipolar disorder in patients, siblings, and controls. Chronobiol Int. 2025;42(6):755–69. DOI: 10.1080/07420528.2025.2509623
[10] Brum M, Nieberler M, Kehrwald C, Knopf K, Brunkhorst-Kanaan N, Etyemez S, et al. Phase-and disorder-specific differences in peripheral metabolites of the kynurenine pathway in major depression, bipolar affective disorder and schizophrenia. World J Biol Psychiatry. 2023;24(7):564–77. DOI: 10.1080/15622975.2023.2169348
[11] World Medical Association. Declaration of Helsinki: Ethical Principles for Medical Research Involving Human Subjects [Internet]. [cited 23 November 2025]. Available from: https://surl.li/nlbkwz
[12] Diagnostic and statistical manual of mental disorders: DSM-5. Washington: American Psychiatric Association Publishing; 2013. 96 P. DOI: 10.1176/appi.books.9780890425596
[13] Sales PMG, Schrage E, Coico R, Pato M. Linking nervous and immune systems in psychiatric illness: A meta-analysis of the kynurenine pathway. Brain Res. 2023;1800:148190. DOI: 10.1016/j.brainres.2022.148190
[14] Savitz J, Drevets WC, Smith CM, Victor TA, Wurfel BE, Bellgowan PS, et al. Putative neuroprotective and neurotoxic kynurenine pathway metabolites are associated with hippocampal and amygdalar volumes in subjects with major depressive disorder. Neuropsychopharmacology. 2015;40:463–71. DOI: 10.1038/npp.2014.194
[15] Skorobogatov K, Autier V, Foiselle M, Richard JR, Boukouaci W, Wu CL, et al. Kynurenine pathway abnormalities are state-specific but not diagnosis-specific in schizophrenia and bipolar disorder. Brain Behav Immun Health. 2023;27:100584. DOI: 10.1016/j.bbih.2022.100584
[16] Fellendorf FT, Manchia M, Squassina A, Pisanu C, Dall'Acqua S, Sut S, et al. Is poor lithium response in individuals with bipolar disorder associated with increased degradation of tryptophan along the kynurenine pathway? Results of an exploratory study. J Clin Med. 2022;11(9):2517. DOI: 10.3390/jcm11092517
[17] Patlola SR, Donohoe G, McKernan DP. Anti-inflammatory effects of 2nd generation antipsychotics in patients with schizophrenia: A systematic review and meta-analysis. J Psychiatr Res. 2023;160:126–36. DOI: 10.1016/j.jpsychires.2023.01.042
[18] Cathomas F, Guetter K, Seifritz E, Klaus F, Kaiser S. Quinolinic acid is associated with cognitive deficits in .schizophrenia but not major depressive disorder. Sci Rep. 2021;11:9992. DOI: 10.1038/s41598-021-89335-9
[19] Trepci A, Sellgren CM, Pålsson E, Brundin L, Khanlarkhani N, Schwieler L, et al. Central levels of tryptophan metabolites in subjects with bipolar disorder. Eur Neuropsychopharmacol. 2021;43:52–62. DOI: 10.1016/j.euroneuro.2020.11.018
[20] Myint AM, Schwarz MJ, Verkerk R, Mueller HH, Zach J, Scharpé S, et al. Reversal of imbalance between KA and 3-hydroxykynurenine by antipsychotics in medication-naïve and medication-free schizophrenic patients. Brain Behav Immun. 2011;25(8):1576–81. DOI: 10.1016/j.bbi.2011.05.005

[21] Wurfel BE, Drevets WC, Bliss SA, McMillin JR, Suzuki H, Ford BN, et al. Serum KA is reduced in affective psychosis. Transl Psychiatry. 2017;7(5):e1115. DOI: 10.1038/tp.2017.88
[22] Marx W, McGuinness AJ, Rocks T, Ruusunen A, Cleminson J, Walker AJ, et al. The kynurenine pathway in major depressive disorder, bipolar disorder, and schizophrenia: A meta-analysis of 101 studies. Mol Psychiatry. 2021;26(8):4158–78. DOI: 10.1038/s41380-020-00951-9

Рівень хінолінової кислоти та кінуренової кислоти в плазмі крові пацієнтів з важкими психічними захворюваннями

Шеу Рахамон
Доктор філософії, викладач
Медичний коледж, Університет Ібадана
200285, дор. Королеви Єлизавети II, Агоді, м. Ібадан, Нігерія
https://orcid.org 00-0003-0305-5026

Софіят Белло
Магістр, аспірант
Медичний коледж, Університет Ібадана
200285, дор. Королеви Єлизавети II, Агоді, м. Ібадан, Нігерія
https://orcid.org/0009-0008-7780-8020

Абдулрахамон Сікірулла
Магістр, аспірант
Медичний коледж, Університет Ібадана
200285, дор. Королеви Єлизавети II, Агоді, м. Ібадан, Нігерія
https://orcid.org/0009-0001-5579-8414

Олуремі Оладеле
Бакалавр, викладач, консультант-психіатр
Медичний коледж, Університет Ібадана
200285, дор. Королеви Єлизавети II, Агоді, м. Ібадан, Нігерія
https://orcid.org/0009-0001-0472-4714

Віктор Ласебікан
Доктор філософії, професор, консультант-психіатр
Медичний коледж, Університет Ібадана
200285, дор. Королеви Єлизавети II, Агоді, м. Ібадан, Нігерія
https://orcid.org/0000-0001-5834-9190

Анотація. З'ясування впливу антипсихотичних препаратів на метаболіти кінуренінового шляху може покращити розуміння їхньої ролі у розвитку основних психічних захворювань. Тому метою цього дослідження було оцінити рівні хінолінової та кінуренової кислот у плазмі крові пацієнтів з основними психічними захворюваннями. У дослідженні взяли участь 85 дорослих, серед яких 55 пацієнтів з основними психічними захворюваннями (шизофренія, біполярний розлад та депресія), які раніше проходили лікування, та 30 осіб з контрольної групи. Рівень хінолінової кислоти та кінуренової кислоти в плазмі крові визначали за допомогою імуноферментного аналізу. Рівень хінолінової кислоти в плазмі крові був значно нижчим у пацієнтів з тяжкими психічними захворюваннями (14,52 (7,16-48,24) мг/мл) порівняно з контрольною групою (157,79 (68,64-464,05) мг/мл). Натомість рівень кінуренової кислоти в плазмі був вищим у пацієнтів з тяжкими психічними захворюваннями (243,96 (210,65-283,05) μмоль/л) порівняно з контрольною групою (215,36 (185,67-243,65) μмоль/л). Після стратифікації на групи пацієнти зі шизофренією (22,86 (7,62-53,14) мг/мл), біполярним розладом (15,34 (8,37-48,21) мг/мл) та депресією (6,18 (2,66-43,05) мг/мл) мали значно нижчий рівень хінолінової кислоти порівняно з контрольною групою (157,79 (68,64-464,05) мг/мл). Однак рівень кінуренової кислоти був значно підвищений лише у пацієнтів із шизофренією (247,0 (208,34-290,22 μмоль/л) та біполярним розладом (243,20 (212,85-291,12) μмоль/л) порівняно з контрольною групою (215,36 (185,67-243,65) μмоль/л). Рівень хінолінової та кінуренової кислот не відрізнявся істотно у пацієнтів із шизофренією, біполярним розладом та депресією. Пацієнти, які лікувалися від серйозних психічних захворювань, мали профіль метаболітів кінуреніну, що вказував на зниження нейротоксичності та посилення нейропротекції. Ці метаболіти можуть слугувати потенційними біомаркерами для моніторингу ефектів лікування

Ключові слова: антипсихотики; запалення; метаболізм триптофану; розлади психічного здоров'я; нейропротекція