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Original Research

Cognitive Scores and Selected Polyunsaturated Fatty Acids in Adolescents With Depression in Sokoto

AI Umar[1], Yusuf Sarkingobir[2], Yusuf Yahaya Miya[3], Syed Abdul Waheed[4]

[1]Department of Biochemistry and Molecular Biology, Sokoto State University, Sokoto, Sokoto State, Nigeria
[2]Department of Environmental Education, Shehu Shagari University of Education, Sokoto, Sokoto State, Nigeria
[3]University of Port Harcourt, Rivers State, Nigeria
[4]Department of Educational Studies, University of Okara, Okara, Punjab, Pakistan

ABSTRACT

Introduction: Polyunsaturated fatty acids (PUFAs) participate in neuronal functions, but their relationship with cognitive outcomes among adolescents with depression remains uncertain.

Objective: To compare cognitive screening scores between adolescents with reported depression and healthy participants and describe available PUFA measurements in Sokoto, Nigeria.

Method: This cross-sectional comparison included 30 adolescents with reported depression and 25 comparison participants. Cognitive screening was described using a mini-mental-state instrument, and selected fatty-acid values were reported for the depression group. An independent-samples t-test was reported.

Result: Mean cognitive scores were 21.50 ± 0.94 and 26.72 ± 3.048 in the depression and comparison groups, respectively (reported $t = -8.31$; $p = 0.001$). Four PUFA summary values were available only for the depression group, without units or reference intervals.

Conclusion: Lower cognitive screening scores were reported in the depression group. The available data do not establish PUFA deficiency or an association between PUFA status and cognition; methodological and biochemical verification is required.

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CONTACT
*AI Umar
mamunetdaji@gmail.com
Department of Biochemistry and Molecular Biology, Sokoto State University, Sokoto, Sokoto State, Nigeria

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INTRODUCTION

Depression in adolescence is a major clinical and public-health concern, with possible consequences for learning, attention, memory and everyday functioning. A systematic review of 17 studies found lower average performance in several cognitive domains among children and adolescents with depression than among non-depressed peers, although associations differed by domain and study design (1). Thus, cognitive assessment may provide clinically useful complementary information, but screening results must be interpreted within the developmental context.

Adolescence is a period of continuing executive-function and emotional development. Reduced concentration and processing efficiency during a depressive episode should not automatically be labelled a persistent neurocognitive disorder. In the present study, the recorded cognitive screening scores are treated as test performance, rather than a validated diagnosis of mild cognitive impairment in adolescents (1,19).

Polyunsaturated fatty acids (PUFAs), particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have biological roles in cell membranes, neurotransmission, inflammatory signaling and synaptic plasticity (2-4). Observational literature has examined their relationship with mental health, but findings are heterogeneous. Mongan and colleagues found limited evidence of cross-sectional associations with depressive disorder at age 17 in a population cohort, whereas a United States adult survey identified associations with selected circulating PUFA fractions after covariate adjustment (5,6). These results cannot simply be generalized to adolescents receiving psychiatric care. Childhood PUFA biomarkers have also been associated with later changes in body-mass-index trajectories, making nutritional status a potential confounder to record rather than an observed explanation here (13).

Biochemical interpretation requires distinction between the essential dietary fatty acids linoleic acid (18:2n-6) and alpha-linolenic acid (18:3n-3) and their longer-chain metabolites, including arachidonic acid (20:4n-6), EPA (20:5n-3), DHA (22:6n-3), and DPA (22:5n-3). Plasma, serum, erythrocyte, and dietary measures are not interchangeable; exposure windows and laboratory units vary. Long-chain PUFAs can influence cell membranes and inflammation-related signalling but are also susceptible to lipid peroxidation. These are mechanistic hypotheses, not outcomes measured in the current study (3,5,6).

Current clinical evidence also cautions against interpreting observational PUFA differences as proof of a modifiable cause of depression. A recent Cochrane review found that randomized evidence for omega-3 supplementation in depressed children and adolescents is of very low certainty (7). More broadly, conceptual reviews propose inflammatory and neuroplastic pathways as possible explanations, but these mechanisms need direct testing in clinical samples (3,8).

Research combining reported fatty-acid measurements with cognitive screening scores among adolescents attending neuropsychiatric services in north-western Nigeria remains limited. The present study contributes a local descriptive comparison of cognitive scores and a summary of selected PUFA values in the depression group. Crucially, the existing dataset, as reported, does not contain comparable PUFA results for healthy participants or patient-level PUFA–cognition associations. This limits inference about whether PUFA status explains the group difference. The objective was therefore to compare cognitive screening scores between groups and descriptively report the available PUFA measurements, rather than determine a causal effect.

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The prespecified, document-supported objectives are limited to comparing unadjusted cognitive screening scores between the two reported groups and describing selected fatty-acid results in the depression group. Whether individual PUFA concentrations correlate with depression severity or cognition remains unknown because matched PUFA measurements and linked individual-level records were not provided.

METHOD

Study design and setting

This was a cross-sectional, single-centre observational comparison conducted among adolescents attending Federal Neuropsychiatric Hospital Kware, Sokoto, Nigeria, and a group described by the authors as healthy participants. No intervention, random allocation or longitudinal follow-up was reported. The study period, enrolment pathway and exact recruitment setting for the comparison group were not specified in the source documents.

Participants and sampling

The study included 30 adolescents reported to have a diagnosis of depression and 25 healthy participants (total n = 55). The source document does not provide participants' exact ages, sex distributions, eligibility criteria, sampling method, depression-diagnostic criteria, psychiatric medications or matching variables. These details are necessary for reproducibility and for interpreting possible confounding.

Depression and cognitive assessment

The authors reported using the Zung Self-Rating Depression Scale for depression assessment (9). The manuscript also refers to a Mini-Mental State Examination (MMSE) when describing cognitive performance (10). The MMSE was originally developed for adult clinical assessment; pediatric adaptations exist for some populations (19), but no evidence was supplied that the version, language, administration method or cut-off used here had been validated for the included adolescents. Therefore, the results are described as cognitive screening scores, not a verified diagnosis or prevalence of mild cognitive impairment. The score direction was interpreted as higher scores indicating better performance, consistent with the conventional MMSE framework, subject to authors' confirmation.

PUFA assessment

The source manuscript reports measurements for arachidonic acid (AA; 20:4n-6), EPA (20:5n-3), DHA (22:6n-3), and docosapentaenoic acid (DPA; 22:5n-3). It refers to a chromatographic approach and cites prior population biomarker research (6,11), but does not specify the biological specimen, analytical platform, processing conditions, calibration, units, or thresholds for low concentration. Because only group summary values for adolescents with depression are reported, neither a comparison of PUFA concentrations between groups nor a PUFA–cognition regression can be reconstructed from the material available.

Laboratory nomenclature was standardized provisionally to DHA (22:6n-3) and DPA (22:5n-3); the original document contains a potentially serious DHA notation error ("20:6n-3").

Statistical analysis

The original manuscript describes descriptive statistics (mean and standard deviation) and an independent two-sample t-test using SPSS version 20, with a prespecified significance threshold

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of $p < 0.05$. Group mean, standard deviation and size are reproduced unchanged from the source. The reported test statistic was $t = -8.31$ with $p = 0.001$; degrees of freedom, variance-assumption testing and the original software output were not supplied. Values reconstructed approximately from the rounded summary statistics are not identical to the reported t-value, so inferential output should be verified before publication. No adjusted comparison, correlation or mediation analysis was provided.

A transparent sensitivity calculation from the rounded source summaries (n = 30/25; means = 21.50/26.72; SD = 0.94/3.048) gives a Welch unequal-variance t statistic of approximately −8.24 (df ≈ 27.81), with a depression-minus-comparison mean difference of −5.22 and an approximate 95% CI of −6.52 to −3.92 (two-sided $p < 0.001$). This reconstructed calculation is not the authors' original SPSS output and does not replace the reported $t = -8.31$.

Ethical considerations

The authors report that permission and consent from participants and guardians were obtained, and cite a hospital administrative document numbered FNPHK/ADM/SUB/809/VOL.I/P.104. The original file does not identify the name of an independent research ethics committee or an ethics approval identifier distinct from institutional administrative permission. Given the age of participants and use of psychiatric data, the ethics authority and consent/assent procedures must be documented prior to publication.

RESULTS

The analytical sample comprised 55 participants: 30 adolescents with reported depression and 25 healthy comparison participants. Sex, age distribution and other participant characteristics were unavailable in the source manuscript and therefore cannot be tabulated reliably.

Reported PUFA measurements

Table 1 reproduces the four PUFA values originally presented for the depression group. Because units, reference intervals and participant-level paired cognitive assessments are unavailable, the values are descriptive and cannot support a classification of PUFA deficiency or cognitive impairment per analyte.

The original table associated individual fatty-acid rows with separate "mild cognitive impairment" categories. That juxtaposition has been corrected: biochemical means characterize measured analytes, whereas neurocognitive classification would require participant-level scores and developmentally validated criteria. The results do not establish that every participant had low PUFA concentrations or clinically defined impairment.

Table 1. Reported fatty-acid values in adolescents with depression (n = 30; units unreported)

Fatty-acid analyte	mean ± SD
Arachidonic acid (AA), 20:4n-6	4.60 ± 0.10
Eicosapentaenoic acid (EPA), 20:5n-3	1.80 ± 0.50
Docosahexaenoic acid (DHA), 22:6n-3	2.03 ± 0.60
Docosapentaenoic acid (DPA), 22:5n-3	5.74 ± 0.10

Comparison of cognitive screening scores

The mean reported cognitive screening score was 21.50 ± 0.94 in the depression group and 26.72 ± 3.048 among healthy participants. The original analysis reported $t = -8.31$ and $p = 0.001$

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(Table 2). These summary data describe an unadjusted difference and do not establish the contribution of any particular PUFA or confounding variable.

The between-group score difference is unadjusted. Because psychiatric recruitment, education, age, medication and nutritional status may differ between groups, confounding and selection bias are plausible explanations alongside illness-related concentration difficulties. No PUFA–cognition correlation, regression, interaction or mediation analysis is reported; therefore the three-way association suggested by the original title remains untested.

Table 2. Cognitive screening scores by study group

Group	N	Mean ± SD	Original t	Original p
Depression group	30	21.50 ± 0.94	−8.31	0.001
Healthy participants	25	26.72 ± 3.048	—	—

DISCUSSION

The central finding is that the depression group had lower cognitive screening scores than the healthy comparison group. This pattern is broadly compatible with a systematic review finding modest deficits in working memory, long-term memory, attention, executive functioning and language in children and adolescents with depression (1). Nevertheless, the present outcome is a single screening score. Without age-appropriate validity evidence and a domain-specific neuropsychological battery, it is not possible to identify which cognitive functions differed or classify individuals as having mild cognitive impairment.

PUFA biology provides a plausible research rationale but is not a tested explanation of this study's group difference. EPA and DHA are involved in neuronal membrane structure, inflammatory resolution and synaptic signaling; reviews describe possible links with neuroplasticity and mood regulation (2-4,8,12). However, these pathways were not measured here. No within-individual correlation between PUFA fractions and cognitive performance, or comparison of PUFA measurements with controls, was provided. Consequently, language implying that low PUFA levels caused depression or impaired cognition must be removed.

Studies of blood PUFA levels and psychiatric outcomes demonstrate why cautious interpretation is warranted. Mongan et al. reported little evidence for cross-sectional relationships with depression at age 17 but some associations in young adults, while Wang et al. reported specific associations in an adult United States sample using adjusted regression models (5,6). Li et al. described further associations in older populations (11). These cohorts differ from hospital-attending Nigerian adolescents in age, setting, measurement and confounder control. The current results cannot be directly compared with their PUFA concentration estimates until analytic units and matrices are established.

Interpretation should accommodate reverse causation: depressed mood and treatment may affect appetite, food selection, body composition or biochemical status, while dietary factors may also affect mood-related pathways. The temporal direction cannot be resolved in a cross-sectional design. No dietary intake, household food security, rurality, inflammatory markers, supplement use or psychiatric medication profiles were measured in the supplied data, so related contextual explanations cannot be presented as study findings.

Interventional findings are also mixed. A Cochrane synthesis of trials in young people judged the benefit of omega-3 supplementation for depressive symptoms uncertain because the evidence was very low certainty (7). A randomized placebo-controlled trial among adolescents with major

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depression did not find omega-3 monotherapy superior to placebo (14). A youth-focused scoping review reached similarly inconclusive conclusions, while later clinical reviews discuss specific patient subgroups requiring further study (17,18). Thus, dietary modification or supplementation should not be presented as a proven intervention arising from the present cross-sectional data. Nutritional assessment may be reasonable within comprehensive adolescent care, but clinical management requires independent evidence and patient-specific evaluation (21). Earlier controlled pediatric omega-3 trials further underscore differences in age, diagnosis, and follow-up periods across the evidence base (16).

Mechanistic work on synaptic plasticity, neurogenesis, membrane signaling and blood-brain barrier function provides hypotheses for future investigations, not confirmation of pathways within this sample (20,22-24). A randomized pediatric omega-3 trial conducted for a distinct neurodevelopmental condition (25) also illustrates why findings from other diagnoses should not be extrapolated to depression.

This study's contribution is principally descriptive: it highlights potentially important differences in cognitive screening performance among psychiatric patients and documents that selected fatty-acid measurements were obtained in a clinical setting in Sokoto. To convert these descriptive observations into stronger evidence, future studies should measure PUFA fractions in both groups, collect dietary and medication information, use validated adolescent cognitive tests, specify recruitment criteria and pre-register an analysis plan addressing confounding by age, sex and illness severity. Prospective or controlled designs would better inform the temporal relationships between lipid status, depression and cognition. Transparent reporting should follow observational-study principles such as STROBE (15).

Study limitations

This study was limited by its cross-sectional design, relatively small sample size, and unadjusted group comparisons. Because comparable PUFA measurements were unavailable for the control group, the study cannot establish an association between PUFA concentrations and cognitive performance or determine whether PUFA deficiency contributes to adolescent depression.

CONCLUSION

Among 55 adolescents evaluated in Sokoto, those classified as having depression had lower mean reported cognitive screening scores than healthy comparison participants. Selected PUFA values were available descriptively for the depression group, but the manuscript does not provide sufficient laboratory or statistical evidence to classify deficiency or link PUFA status to the observed score difference. Further studies with validated adolescent assessments, complete biochemical reporting and appropriately controlled designs are required before drawing etiologic or clinical-prevention conclusions.

AUTHOR'S CONTRIBUTION STATEMENT

Author contributions are not specified in the source manuscript. Each author must confirm their actual roles (conceptualization, methodology, investigation, data curation, analysis, drafting, critical revision and approval of final version) before the statement can be finalized. AI Umar (AIU); Yusuf Sarkingobir (YS); Yusuf Yahaya Miya (YYM); Syed Abdul Waheed (SAW).

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CONFLICTS OF INTEREST

The source manuscript contains no verified conflict-of-interest declaration. The authors must disclose any financial, professional, personal or institutional interests capable of influencing this work, or explicitly confirm that no such conflicts exist.

DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

AI assistance has been used to prepare language and structural revisions to this draft. All authors must review the revised text, verify its fidelity to the original study and data, ensure appropriate disclosure under journal policy, and take responsibility for the final submission. AI tools did not supply new participant-level observations or laboratory results.

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