RESEARCH PAPER

Executive Function Shows the Greatest Cognitive Benefit from Omega-3 Supplementation after Ischemic Stroke

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ABSTRACT

Background: Post-stroke cognitive impairment (PSCI) is a frequent complication following ischemic stroke and contributes to reduced functional recovery and quality of life. Omega-3 fatty acids, particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have demonstrated neuroprotective properties; however, clinical evidence regarding their role in post-stroke cognitive recovery remains limited and inconsistent.

Methods: This preliminary double-blind, placebo-controlled randomized controlled trial evaluated omega-3 supplementation on cognitive function in patients with early post-stroke cognitive impairment after ischemic stroke. Sixty-two participants were randomly assigned to omega-3 supplementation (EPA+DHA 2000 mg/day; n=31) or placebo (n=31) for six weeks plus standard management. MoCA-INA assessed global and domain-specific cognitive changes at baseline and follow-up.

Results: Baseline demographic and clinical characteristics were comparable between groups. The omega-3 group demonstrated significant improvement in MoCA-INA scores, increasing from 19.35 ± 4.86 to 21.16 ± 5.48 ($p<0.001$), whereas the placebo group showed no significant change (17.84 ± 6.01 to 17.94 ± 6.20; $p=0.499$). Between-group analysis revealed greater cognitive improvement in the omega-3 group (ΔMoCA-INA 1.77 ± 1.36 vs 0.13 ± 0.67; $p<0.001$). Domain-specific analysis showed significant benefits in executive function ($p<0.001$), memory ($p<0.001$), and visuospatial function ($p=0.011$), while attention ($p=0.108$) and language ($p=0.743$) did not differ significantly between groups.

Conclusion: Omega-3 supplementation during the post-acute phase of ischemic stroke was associated with improved global cognitive function and selective enhancement of executive, memory, and visuospatial domains. Omega-3 may represent a promising adjunctive strategy for post-stroke cognitive rehabilitation, although larger trials with longer follow-up are needed to confirm these findings.

Keywords: ischemic stroke; post-stroke cognitive impairment; omega-3; cognitive rehabilitation; MoCA-INA; randomized controlled trial

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INTRODUCTION

Stroke remains a leading cause of mortality and long-term disability worldwide and imposes a substantial socioeconomic burden and reduction in quality of life.[1] Beyond motor deficits, cognitive impairment is a common complication following stroke and may emerge during the acute phase or develop over subsequent months. Approximately 20–50% of stroke survivors experience post-stroke cognitive impairment (PSCI), ranging from mild cognitive impairment to vascular dementia, which adversely affects rehabilitation outcomes, functional independence, and long-term prognosis.[2]

Omega-3 polyunsaturated fatty acids, particularly docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), have gained attention as potential neuroprotective interventions because of their anti-inflammatory, antioxidant, and neuronal membrane-stabilizing effects.[3] Experimental studies have demonstrated that omega-3 supplementation improves white matter integrity, promotes anti-inflammatory microglial polarization, and enhances long-term neurological recovery after ischemic

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injury.[4] Additional evidence suggests that omega-3 supports angiogenesis, neurogenesis, and oligodendrogenesis, thereby facilitating neurovascular remodeling and functional recovery.[5] Furthermore, omega-3 may activate antioxidant pathways including Nrf2–HO-1 and reduce oxidative stress–related neuronal injury.[6] Neuroprotective effects have also been observed in other neurological injury models, including traumatic brain injury.[7]

Despite promising mechanistic evidence, clinical data evaluating omega-3 supplementation for cognitive recovery after stroke remain limited and inconsistent. An ancillary randomized controlled trial involving patients with cardiovascular disease, including individuals with prior stroke, found no overall benefit of omega-3 supplementation on global cognitive outcomes, although subgroup analysis suggested possible improvement in temporal orientation when combined with B-vitamin supplementation.[8,9] Similarly, a recent systematic review reported mixed findings regarding dietary interventions for cognition among stroke survivors and concluded that current evidence for omega-3 remains insufficient because of substantial heterogeneity in study design, dosage, intervention timing, and cognitive assessment tools.[10] Large randomized trials conducted in community-dwelling older adults without stroke also failed to demonstrate consistent cognitive benefit from omega-3 supplementation, although these findings may not be directly applicable to post-stroke populations with distinct pathophysiological mechanisms.[11]

Current stroke cognitive management guidelines do not recommend omega-3 supplementation as standard therapy for post-stroke cognitive recovery because of the lack of consistent clinical evidence.[12] Potential factors contributing to inconsistent findings include variability in omega-3 dosage and EPA:DHA composition, delayed initiation of supplementation after the acute inflammatory phase, and patient heterogeneity related to age, lesion characteristics, educational level, and biological response markers.[10,11,13] Therefore, this preliminary randomized controlled trial was conducted to evaluate the effect of omega-3 supplementation initiated after the acute phase of ischemic stroke on early cognitive function among patients with early post-stroke cognitive impairment. This study aims to address the current evidence gap and provide preliminary clinical data to support the development of safe, accessible, and potentially effective adjunctive strategies for cognitive rehabilitation in ischemic stroke patients.

MATERIALS AND METHODS

Study Design and Setting

This study was conducted as a double-blind, placebo-controlled randomized controlled trial (RCT) using a parallel-group design to evaluate the effect of omega-3 supplementation on cognitive function in patients with early post-stroke cognitive impairment following ischemic stroke. The study was performed at Dr. Wahidin Sudirohusodo Hospital and affiliated hospitals under the Department of Neurology, Makassar, Indonesia. Participant recruitment, intervention administration, and follow-up assessments were conducted between April 6 and June 17, 2026.

Participants

Eligible participants were adult patients aged 40–75 years with confirmed ischemic stroke diagnosed by computed tomography (CT) or magnetic resonance imaging (MRI), who had entered the post-acute phase (≥7 days and ≤1 month after stroke onset) and demonstrated early cognitive impairment defined as a Montreal Cognitive Assessment–Indonesian version (MoCA-INA) score <25. All participants provided written informed consent prior to enrollment.

Patients were excluded if they had a history of hemorrhagic stroke, pre-existing dementia or cognitive impairment before stroke onset, omega-3 supplementation or other cognitive-targeted therapies within one month before enrollment, severe comorbid conditions including end-stage renal disease, liver failure, or malignancy, or documented allergy to fish-derived products or omega-3 supplements. Participants were withdrawn from the study if they showed poor adherence to intervention, developed severe adverse events, experienced new medical conditions affecting cognitive evaluation, were unavailable for follow-up cognitive assessment, voluntarily withdrew consent, or died during the study period.

Sample Size

Sample size was calculated using the formula for comparison of two independent means based on an anticipated effect size from previous studies evaluating omega-3 supplementation and cognitive outcomes after stroke. Assuming a two-sided alpha level of 0.05 and statistical power of 80%, the minimum required sample was estimated at 27 participants per group. To account for an anticipated dropout rate of 15%, the final target sample size was increased to 31 participants per group, resulting in a total sample of 62 participants.

Randomization and Blinding

After baseline assessment, participants were randomly allocated in a 1:1 ratio into either the intervention group or placebo group. Randomization was performed using a concealed allocation sequence. Both participants and investigators responsible for intervention administration and outcome assessment remained blinded to treatment assignment throughout the study period.

Intervention

Participants assigned to the intervention group received oral omega-3 supplementation in the form of Natureline® Omega-3 Fish Oil capsules containing a combined daily dose of 2000 mg EPA and DHA in addition to standard ischemic stroke management. The control group received identical placebo capsules containing saccharin powder (Saccharin Food®) together with standard stroke therapy.

The intervention was administered once daily for six consecutive weeks.

Data Collection and Outcome Assessment

Baseline evaluation included demographic data collection, vascular risk factor assessment, neurological examination, and laboratory investigations including lipid profile, blood glucose, liver function, and renal function tests. Clinical variables collected included age, sex, education level, hypertension, diabetes mellitus, dyslipidemia, smoking history, recurrent stroke history, stroke severity, and lesion characteristics.

Cognitive function was assessed using the MoCA-INA, which evaluates multiple cognitive domains including memory, attention, language, visuospatial ability, and executive function, with total scores ranging from 0 to 30. Early post-stroke cognitive impairment was defined as a MoCA-INA score below 26. Cognitive assessment was performed at baseline, week 3, and week 6 after intervention initiation.[14]

The primary outcome was change in global cognitive function measured by differences in total MoCA-INA score between baseline and follow-up assessments. Secondary outcomes included changes across individual cognitive domains and evaluation of intervention safety and tolerability through monitoring of gastrointestinal symptoms, allergic reactions, lipid profile, and liver enzyme levels.

The independent variable was omega-3 supplementation (EPA+DHA). The dependent variable was cognitive function measured by MoCA-INA score. Potential confounding variables included age, sex, educational attainment, lesion location and size, stroke severity measured by the National Institutes of Health Stroke Scale (NIHSS), diabetes mellitus, hypertension, dyslipidemia, and concurrent cognitive rehabilitation therapy.

Statistical Analysis

Data analysis was performed using statistical software. Continuous variables were presented as mean ± standard deviation for normally distributed data or median with interquartile range for non-normal distributions, while categorical variables were expressed as frequencies and percentages. Data normality was evaluated prior to inferential analysis. Because not all variables met normality assumptions, between-group comparisons were analyzed using the Mann–Whitney U test. Within-group comparisons of cognitive outcomes before and after intervention were analyzed using the Wilcoxon signed-rank test. Cognitive improvement was calculated as the change in MoCA-INA score (ΔMoCA) between follow-up and baseline assessments. Similar analyses were performed for each cognitive domain. Statistical significance was defined as a two-tailed p-value <0.05.

Ethical Considerations

This study was approved by the Health Research Ethics Committee, Faculty of Medicine, Hasanuddin University/Dr. Wahidin Sudirohusodo Hospital, Makassar (Approval No. 466/UN4.6.4.5.3.1/PP36/2026). All procedures were conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before study enrollment.

RESULTS

Baseline Characteristics

This preliminary randomized controlled trial was conducted between April and June 2026 and enrolled 62 patients with early post-stroke cognitive impairment following ischemic stroke. Participants were randomly allocated to receive either omega-3 supplementation (n=31) or placebo (n=31). Cognitive outcomes were assessed using the MoCA-INA at baseline and after completion of the intervention.

Baseline demographic and clinical characteristics are presented in Table 1. No statistically significant differences were observed between the omega-3 and placebo groups across all baseline variables (all $p>0.05$), indicating successful randomization and balanced group allocation.

The mean age was 57.10 ± 11.33 years in the omega-3 group and 63.13 ± 12.22 years in the placebo group ($p=0.094$). Male participants accounted for 64.5% and 45.2% of subjects in the omega-3 and placebo groups, respectively ($p=0.202$). Educational level distribution was comparable between groups ($p=0.274$), with undergraduate education being the most common category in the omega-3 group and senior high school in the placebo group. Previous stroke history was reported in 25.8% of the omega-3 group and 51.6% of the placebo group ($p=0.068$). No significant between-group differences were observed in post-stroke duration, baseline NIHSS score, or baseline MoCA-INA score. Overall, these findings demonstrate comparable baseline characteristics between study groups prior to intervention.

Changes in Cognitive Function Within Each Group

Changes in cognitive function within each group were evaluated using Montreal Cognitive Assessment–Indonesian version (MoCA-INA) scores obtained before and after the intervention period. Within-group comparisons were performed to assess changes in cognitive performance over time. The results are presented in Table 2. Participants receiving omega-3 supplementation demonstrated a significant improvement in cognitive function, with mean MoCA-INA scores increasing from 19.35 ± 4.86 at baseline to 21.16 ± 5.48 after intervention ($p<0.001$). In contrast, the placebo group showed only minimal change, with mean scores increasing from 17.84 ± 6.01 to 17.94 ± 6.20, which was not statistically significant ($p=0.499$).

The distribution of MoCA-INA scores before and after intervention is illustrated in Figure 3. A visible upward shift in post-intervention scores was observed in the omega-3 group, whereas cognitive scores remained relatively unchanged in the placebo group. Overall, these

findings indicate that omega-3 supplementation was associated with significant improvement in global cognitive function during the study period, while no meaningful cognitive change was observed among participants receiving placebo.

Between-Group Comparison of Cognitive Function Changes

Between-group differences in cognitive outcomes were evaluated using the change in MoCA-INA scores from baseline to post-intervention assessment (Δ MoCA-INA). The magnitude of cognitive improvement was compared between the omega-3 and placebo groups to determine the effect of supplementation on cognitive recovery. The results are summarized in Table 3. The omega-3 group demonstrated a greater improvement in cognitive performance, with a mean Δ MoCA-INA score of 1.77 ± 1.36 points compared with 0.13 ± 0.67 points in the placebo group. Statistical analysis showed that the difference in cognitive change between groups was significant ($p<0.001$).

The distribution of Δ MoCA-INA scores is illustrated in Figure 4. Participants receiving omega-3 exhibited a higher median change and a broader shift toward positive cognitive improvement, whereas changes in the placebo group remained minimal. Overall, the mean between-group difference of 1.64 points suggests that omega-3 supplementation was associated with greater improvement in cognitive function compared with placebo among patients with early post-stroke cognitive impairment following ischemic stroke.

Effects of Omega-3 Supplementation on Cognitive Domains

Domain-specific cognitive analysis was performed using subscale scores from the MoCA-INA. Cognitive domains evaluated included memory, attention, visuospatial ability, executive function, and language. Changes in domain scores from baseline to post-intervention were compared between the omega-3 and placebo groups. The results are presented in Table 4.

Participants receiving omega-3 supplementation demonstrated improvements across most cognitive domains. The largest mean change was observed in executive function (Δ=1.10 ± 0.83), followed by memory (Δ=0.58 ± 0.72), attention (Δ=0.29 ± 0.59), and visuospatial function (Δ=0.19 ± 0.40). Language showed only minimal improvement (Δ=0.06 ± 0.25).

Between-group analysis demonstrated statistically significant differences favoring omega-3 supplementation in memory ($p<0.001$), visuospatial function ($p=0.011$), and executive function ($p<0.001$). In contrast, no significant differences were identified in attention ($p=0.108$) or language ($p=0.743$).

Figure 5 illustrates the distribution of changes in domain-specific scores (Δ score) following intervention. Greater improvement was observed in the omega-3 group for memory, visuospatial, and executive domains, whereas score distributions for attention and language remained comparable between groups.

Overall, these findings suggest that the cognitive benefits associated with omega-3 supplementation were domain-specific and appeared to predominantly affect executive function, memory, and visuospatial performance in patients with early post-stroke cognitive impairment following ischemic stroke.

DISCUSSION

This preliminary randomized controlled trial demonstrated that omega-3 supplementation administered during the post-acute phase of ischemic stroke was associated with greater improvement in cognitive function compared with placebo. Participants receiving omega-3 showed significant improvement in global cognitive performance measured by MoCA-INA, whereas cognitive changes in the placebo group were minimal. In addition, the beneficial effect of omega-3 appeared to be domain-specific, with the greatest improvement observed in executive function, followed by memory and visuospatial performance.

The absence of significant differences in baseline demographic and clinical characteristics suggests that randomization produced comparable groups and reduces the likelihood that post-intervention differences were attributable to confounding factors. This is particularly relevant because age, educational level, stroke severity, and baseline cognitive status are recognized determinants of post-stroke cognitive outcomes through their influence on cognitive reserve and neuroplasticity.[15,16]

The improvement in global cognitive function observed in the omega-3 group supports the hypothesis that nutritional modulation may contribute to cognitive recovery after ischemic stroke. These findings are in line with Shahinfar et al. (2025), who reported beneficial effects of omega-3 supplementation on cognitive performance across several domains, particularly at higher doses.[17] Similarly, Suh et al. (2024) demonstrated that omega-3 supplementation may improve cognitive outcomes, with more consistent effects on higher-order cognitive processes.[18] Mechanistically, DHA and EPA may support post-stroke recovery by reducing neuroinflammation, attenuating oxidative stress, preserving neuronal membrane integrity, and promoting neuroplasticity through increased expression of brain-derived neurotrophic factor (BDNF) and enhancement of synaptic remodeling.[19–21]

Importantly, the mean change in MoCA-INA score was significantly greater in the omega-3 group than in the placebo group (Δ=1.77 vs 0.13 points), suggesting that the observed improvement exceeded expected spontaneous recovery during the study period. These findings differ from those reported by Andreeva et al. (2011), who found no overall cognitive benefit of omega-3 supplementation in patients with cardiovascular disease.[8] Several factors may explain this discrepancy, including differences in study population, intervention timing, omega-3 dosage, and outcome assessment. Unlike previous studies

conducted predominantly in stable cardiovascular populations, the present study initiated supplementation during the early post-stroke phase, a period characterized by active neuroplasticity and neural reorganization, which may represent a therapeutic window for intervention.[22,23]

Domain-specific analysis revealed that executive function demonstrated the largest improvement following omega-3 supplementation. These findings are consistent with Suh et al. (2024), who reported executive function as one of the most responsive cognitive domains to omega-3 intervention [18]. Executive processes rely heavily on fronto-subcortical network integrity and white matter connectivity, both of which may be particularly vulnerable after ischemic injury and potentially responsive to mechanisms promoted by omega-3, including synaptic stabilization and improved neuronal communication.[4,24]

Significant improvements were also observed in memory and visuospatial domains. These findings are in line with Shahinfar et al. (2025) and Welty (2023), who reported favorable effects of omega-3 on memory-related outcomes.[17,25] DHA is highly concentrated within hippocampal and cortical neuronal membranes and may facilitate learning and memory through enhancement of long-term potentiation and BDNF-mediated neuroplasticity.[26,27] Improvements in visuospatial function may additionally reflect preservation of white matter integrity and enhanced cortical connectivity involved in visual information processing and spatial orientation.[7,28]

In contrast, no significant effects were observed in attention and language domains. These findings are also consistent with previous literature showing heterogeneous effects of omega-3 across cognitive domains.[17,18] Recovery of attention and language after stroke may depend more strongly on lesion-specific network reorganization, rehabilitation intensity, and factors not evaluated in the present study, including fatigue, mood status, and sleep quality. Additionally, the language and attention components of MoCA-INA provide relatively brief screening measures and may be less sensitive to subtle changes over short follow-up periods.

Several limitations should be considered when interpreting these findings. First, this was a preliminary RCT with a relatively small sample size and short intervention duration. Second, cognitive outcomes were assessed using a screening instrument rather than comprehensive neuropsychological testing. Third, biomarkers potentially associated with omega-3 response, including DHA/EPA status, inflammatory markers, and neurotrophic factors, were not evaluated. Nevertheless, the randomized and double-blind design strengthens internal validity and provides early clinical evidence supporting omega-3 as a potential adjunctive strategy for cognitive rehabilitation after ischemic stroke.

Overall, the present findings suggest that omega-3 supplementation may improve early post-stroke cognitive recovery, with more pronounced effects on executive, memory, and visuospatial domains. Larger multicenter trials with longer follow-up and mechanistic biomarkers are warranted to confirm these findings and determine long-term clinical relevance.

LIMITATIONS

This study has several limitations that should be considered when interpreting the findings. First, as a preliminary randomized controlled trial, the relatively small sample size (n=62) may have limited statistical power, particularly for detecting subtle differences across cognitive domains and reduced generalizability. Second, the intervention duration was limited to six weeks, which may not fully capture the long-term effects of omega-3 supplementation on post-stroke cognitive recovery, as neuroplastic changes may continue beyond the study period. Third, cognitive assessment was performed exclusively using MoCA-INA. Although validated for post-stroke cognitive screening, MoCA-INA has limited sensitivity for detailed domain-specific evaluation compared with comprehensive neuropsychological testing [29].

Finally, biological markers related to omega-3 response (e.g., DHA/EPA levels, BDNF, inflammatory markers) and several potential confounders, including lesion characteristics, post-stroke depression, sleep quality, and rehabilitation intensity, were not evaluated, limiting mechanistic interpretation of the findings [19]. Despite these limitations, the randomized double-blind placebo-controlled design and balanced baseline characteristics support the internal validity of the study and provide preliminary evidence for further investigation of omega-3 as an adjunctive strategy for post-stroke cognitive rehabilitation.

CONCLUSIONS

In this preliminary randomized controlled trial, omega-3 supplementation administered during the post-acute phase of ischemic stroke was associated with significant improvement in global cognitive function among patients with early post-stroke cognitive impairment. Participants receiving omega-3 demonstrated greater cognitive improvement compared with those receiving placebo. Domain-specific analysis showed that the beneficial effects of omega-3 were more prominent in executive function, memory, and visuospatial performance, whereas no significant improvements were observed in attention and language domains. These findings suggest that omega-3 supplementation may serve as a potential adjunctive strategy for cognitive rehabilitation after ischemic stroke. However, larger studies with longer follow-up periods and more comprehensive cognitive assessment are needed to confirm these findings and determine their long-term clinical relevance.

DECLARATIONS

Consent for Publication

Not applicable.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Competing Interests
The authors declare that they have no competing interests.

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Authors' Contributions
APK contributed to conceptualization, study design, data collection, data analysis, interpretation of results, and manuscript drafting. AM contributed to study supervision, methodology development, and critical revision of the manuscript. MA contributed to participant recruitment, clinical assessment, and data interpretation. YAF contributed to investigation and manuscript review. YG contributed to methodology, supervision, and manuscript revision. MIB contributed to statistical analysis, interpretation of findings, and final manuscript approval. All authors read and approved the final manuscript.

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TABLES AND FIGURE

Table 1. Baseline Characteristics of Study Participants According to Treatment Group

Variable	Omega-3 (n=31)	Placebo (n=31)	p-value
Age (years)	57.10 ± 11.33	63.13 ± 12.22	0.094
Sex			0.202
Male	20 (64.5%)	14 (45.2%)	
Female	11 (35.5%)	17 (54.8%)	
Education Level			0.274
Elementary School	3 (9.7%)	2 (6.5%)	
Junior High School	1 (3.2%)	5 (16.1%)	
Senior High School	10 (32.3%)	12 (38.7%)	
Diploma (D2/D3)	1 (3.2%)	2 (6.5%)	
Bachelor's Degree (D4/S1)	16 (51.6%)	9 (29.0%)	
Master's Degree (S2)	0 (0%)	1 (3.2%)	
Previous Stroke History			0.068
Yes	8 (25.8%)	16 (51.6%)	
No	23 (74.2%)	15 (48.4%)	
Post-stroke Duration (days)	12.16 ± 6.50	11.74 ± 6.56	0.932
Baseline NIHSS Score	5.42 ± 3.06	5.61 ± 3.15	0.733
Baseline MoCA-INA Score	19.35 ± 4.86	17.84 ± 6.01	0.355

Continuous variables are presented as mean ± standard deviation, whereas categorical variables are presented as frequency and percentage.

Table 2. Changes in MoCA-INA Scores Before and After Intervention Within Each Group

Group	Pre-Intervention	Post-Intervention	p-value
Omega-3 (n=31)	19.35 ± 4.86	21.16 ± 5.48	<0.001
Placebo (n=31)	17.84 ± 6.01	17.94 ± 6.20	0.499

Data are presented as mean ± standard deviation. Statistical analysis was performed using the Wilcoxon Signed-Rank test.

Table 3. Between-Group Comparison of Changes in MoCA-INA Scores

Variable	Omega-3 (n=31)	Placebo (n=31)	p-value
Δ MoCA-INA Score	1.77 ± 1.36	0.13 ± 0.67	<0.001

Δ MoCA-INA score was calculated as the difference between post-intervention and baseline MoCA-INA scores. Data are presented as mean ± standard deviation.

Table 4. Between-Group Comparison of Changes in Cognitive Domain Scores

Cognitive Domain	Omega-3 (Δ score)	Placebo (Δ score)	p-value
Memory	0.58 ± 0.72	0.00 ± 0.73	<0.001
Attention	0.29 ± 0.59	0.00 ± 0.58	0.108
Visuospatial	0.19 ± 0.40	0.00 ± 0.26	0.011
Executive Function	1.10 ± 0.83	−0.03 ± 0.55	<0.001
Language	0.06 ± 0.25	0.06 ± 0.25	0.743

Δ score was calculated as the difference between post-intervention and baseline scores. Data are presented as mean ± standard deviation.

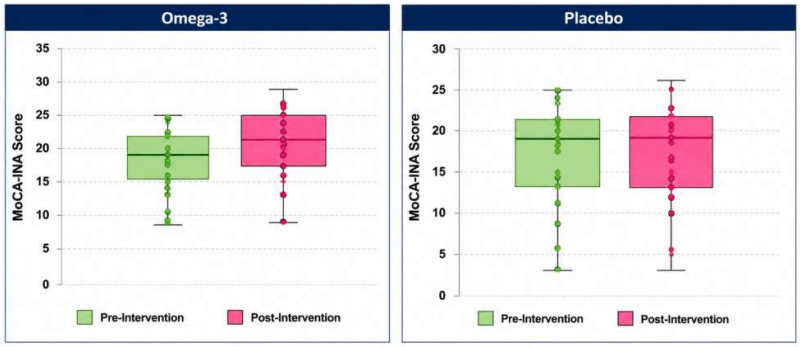

Figure 1. Box Plot of MoCA-INA Scores Before and After Intervention in the Omega-3 and Placebo Groups

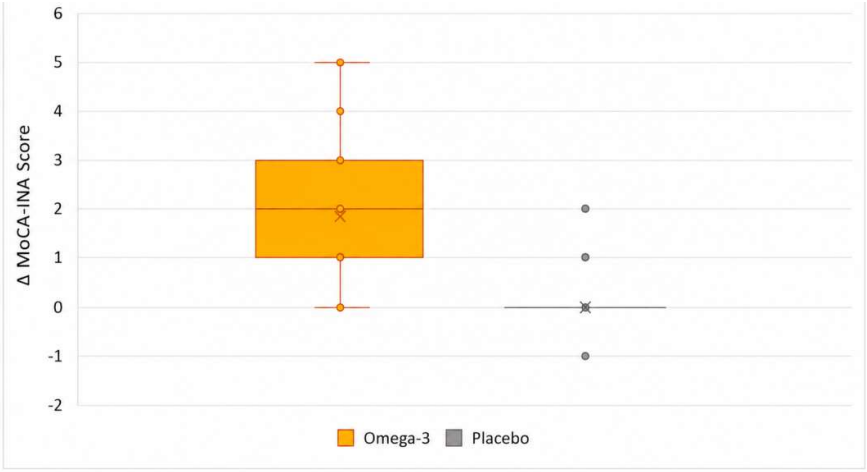

Figure 2. Box Plot of Changes in MoCA-INA Scores Between the Omega-3 and Placebo Groups

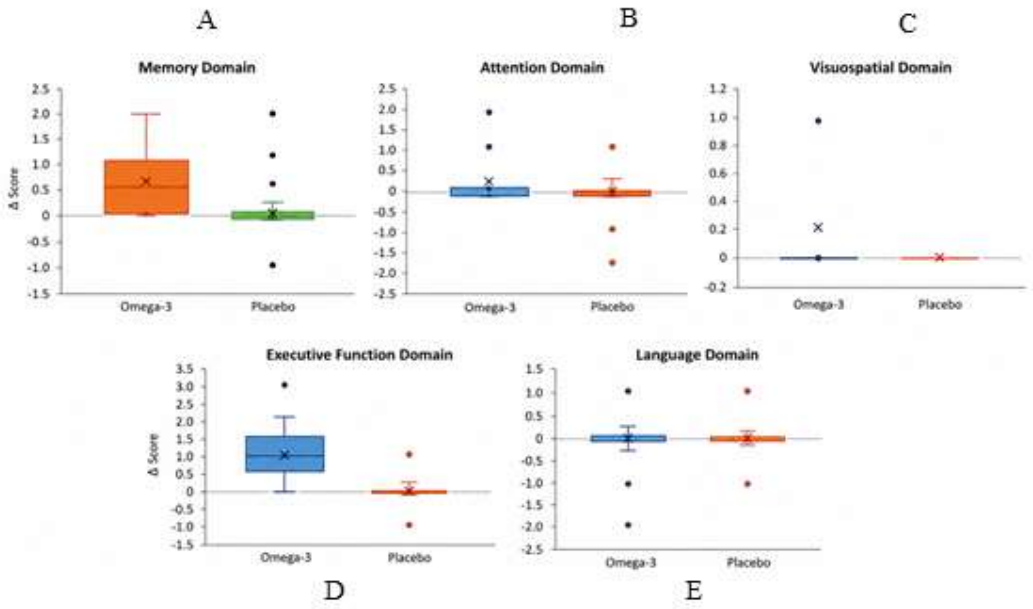

Figure 3. Illustrates the distribution of changes in domain-specific cognitive scores: (A) memory, (B) attention, (C) visuospatial, (D) executive function, and (E) language. Greater improvement was observed in the omega-3 group for memory, visuospatial, and executive function, whereas attention and language showed comparable score distributions between groups.