

## Omega-3 Polyunsaturated Fatty Acids and Cognition in a College-Aged Population

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The cognitive influences of omega-3 polyunsaturated fatty acids (n-3 PUFA) remain unclear throughout the life span. Dietary n-3 PUFA appear cognitively beneficial prenatally and neuroprotective at later age; however, researchers using supplementation designs have reported disparate findings across age groups. Few studies have examined the cognitive impact of n-3 PUFA during young adulthood. This study assessed the cognitive effects of fish oil supplementation at college age, hypothesizing benefits on affect, executive control, inhibition, and verbal learning and memory. College-aged participants were assigned to active ( $n = 20$ , 5 men;  $\bar{x}_{\text{age}} = 19.9$ ,  $s_{\text{age}} = 1.8$ ) or placebo ( $n = 21$ , 7 men;  $\bar{x}_{\text{age}} = 20.4$ ,  $s_{\text{age}} = 1.6$ ) treatments, receiving fish oil (480 mg DHA/720 mg EPA) or coconut oil, respectively. Both groups completed four weeks of supplementation. At baseline and posttreatment, the researchers administered the Rey Auditory Verbal Learning Test (RAVLT; Lezak, 1995), Stroop Color and Word Test (SCWT; Golden & Freshwater, 2002), Trail Making Test (TMT; Corrigan & Hinkeldey, 1987; Gaudino, Geisler, & Squires, 1995; Lezak, 1995), and Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). Repeated-measures ANOVAs indicated no benefits of fish oil on the SCWT, RAVLT Stages 1 to 5, or PANAS. An interaction occurred between condition and time of measurement (i.e., baseline and posttreatment) on RAVLT Stages 6 and 7, and placebo significantly improved TMT performance over fish oil. The benefits of n-3 PUFA on RAVLT performance derived more from depreciated placebo performance than improved performance due to fish oil. The placebo gain on TMT performance likely derived from a learning effect. Together, these results present limited cognitive benefits of n-3 PUFA at college age; however, the treatment may have been subtherapeutic, with a larger sample needed to generalize these results.

**Keywords:** omega-3 fatty acids, DHA, EPA, cognition, young adulthood

The essential fatty acids have gained the attention of researchers examining dietary influences on the brain. These fatty acids include omega-3 (n-3) and omega-6 (n-6) polyunsaturated fatty acids (PUFA), which appear mutually necessary for a healthful human diet (Karr, Alexander, & Winningham, 2011). Most cognitive research has examined the effects of two specific n-3 PUFA, docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA). These n-3 PUFA have important roles in the central nervous system, with 50% of neuronal membranes and 70% of myelin composed of lipids (Yehuda, Rabinovitz, & Mostofsky, 2005a). Researchers have observed the influence of EPA on myelination (Salvati et al., 2008) and as a precursor to DHA (Heinrichs, 2010), which accounts for 40% of PUFA in the brain (Singh, 2005). These n-3 PUFA notably impact the activity of dopamine and

other relevant neurotransmitters (Chalon, 2006; Yehuda et al., 2005a). The neurophysiology of n-3 PUFA may explain the potential impacts of the nutrients on cognition throughout the life span.

The cognitive influences of n-3 PUFA differ across age groups, with the requirements of n-3 PUFA for cognition amplified at the extremities of the life span (Karr et al., 2011). During the last trimester of pregnancy and first postnatal months, the infant experiences a growth spurt in the brain, with a large increase in the cerebral content of n-6 PUFA and DHA (Helland, Smith, Saarem, Saugstad, & Drevon 2003). At older ages, individuals consuming more n-3 PUFA appear resistant against neurodegeneration, as those reporting high n-3 PUFA diets experience lower rates of dementia and cognitive declines (Albanese et al., 2009; Eskelinen et al., 2008; Solfrizzi et al., 2006). From ages 45 to 70, marine n-3 PUFA in the diet were inversely related to risk for impaired cognitive performance (Kalmijn et al., 2004). These later-life studies indicate consistent neuroprotective effects of dietary n-3 PUFA; however, researchers supplementing the diets of older adults with n-3 PUFA report more disparate findings (Dangour et al., 2010; Quinn et al., 2010; Yurko-Mauro et al., 2010). This ambiguity of results in supplementation research transcends to other stages of the life span as well.

Despite an apparent requirement of PUFA for infants, researchers have reported both beneficial (Helland et al., 2003) and no influence (Makrides et al., 2010) of n-3 PUFA supplementation on

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early cognitive development. Among children, researchers have reported similarly inconsistent findings. Some researchers have reported no cognitive benefit of n-3 PUFA through supplementation (Kennedy et al., 2009) or fortified diet (Muthayya et al., 2009), although Ryan and Nelson (2008) found a mild link between DHA and comprehension and vocabulary. Another research group examined children's cheek cell phospholipids and reported a weak correlation between DHA concentration and nonverbal IQ, but interpreted these results with caution (Kirby, Woodward, Jackson, Wang, & Crawford, 2010).

Beyond childhood, the influence of supplementation remains similarly unclear. Assessing college students, Yehuda, Rabinovitz, and Mostofsky (2005b) found that three weeks of 1:4 n-3 to n-6 PUFA supplementation reduced test anxiety. Examining healthy adults, Fontani et al. (2005) observed improvement in attention and mood after five weeks of n-3 PUFA supplementation. However, this study has notable design-related concerns, including a wide age range (i.e., 22 to 51), unreported placebo values in the analyses, and regularly exercising participants in the sample, which may produce an interacting influence of n-3 PUFA and exercise on cognition (Chytrova, Ying, & Gomez-Pinilla, 2010; Wu, Ying, & Gomez-Pinilla, 2008). Antypa, van der Does, Smelt, & Rogers (2009) partially replicated the Fontani et al. design with a 4-week treatment period and roughly similar dosage, but found little influence of n-3 PUFA over placebo on most measures of cognition and mood. These results cumulatively show potential cognitive benefits of n-3 PUFA from prior to late adulthood; however, further research must determine if any benefit truly exists.

Cognitive scientists have mostly focused on n-3 PUFA prenatally and at later age, resulting in very little understanding of the cognitive value of n-3 PUFA among young adults (Karr et al., 2011). Confounding this issue, researchers using n-3 PUFA supplementation have reported inconsistent results across the life span

(see Table 1, for a summary of studies using an n-3 PUFA supplementation design). Although Fontani et al. (2005) remains the only study reporting cognitive benefits of n-3 PUFA for an adult sample prior to later age, their results cannot be disregarded, as few studies have investigated similar populations.

The incongruent results surrounding young adult populations (Antypa et al., 2009; Fontani et al., 2005; Yehuda et al., 2005b) have led the current investigation to focus on a college-aged sample and the possible benefits of n-3 PUFA supplementation on their cognitive performance and mood. Considering the psychophysical role of n-3 PUFA (Heinrichs, 2010; Salvati et al., 2008; Singh, 2005; Yehuda et al., 2005a) and positive findings of Fontani et al. (2005) on cognition and mood, we hypothesized that college-aged participants supplemented with fish oil capsules containing n-3 PUFA would show greater improvement from baseline than participants supplemented with a coconut oil placebo on measures of affect, executive control, inhibition, and verbal learning and memory.

## Method

### Participants

A total of 43 college students enrolled in the study, with 21 receiving n-3 PUFA fish oil supplements and 22 receiving coconut oil placebos. Two participants, one from each condition, were removed from analyses due to problems with following supplementation procedures. This resulted in 41 participants ( $N = 41$ ) eligible for analysis, with 20 (5 men,  $\bar{x}_{age} = 19.90$ ,  $s_{age} = 1.83$ ) in the active fish oil treatment and 21 ( $n = 21$ ; 7 men,  $\bar{x}_{age} = 20.43$ ,  $s_{age} = 1.63$ ) in the coconut oil placebo condition (see Table 2, for more participant information). Exclusion criteria included colorblindness, pregnancy, regular consumption of fish or coconut oil supplements, and allergies to fish or coconut. Participants were

Table 1  
*Cited N-3 PUFA Studies Using Supplementation Designs*

	Citation	N	Treatment duration	Sample diagnosis	Dosage	Benefit over placebo
Prenatal	Helland et al. (2003)	84	18th week of gestation till 12 weeks after birth	—	1180 mg DHA/800 mg EPA	Yes, Cognitive
	Makrides et al. (2010)	726	Before 21st week till birth	—	800 mg DHA	No
Childhood	Kennedy et al. (2009)	86	8 weeks	—	400 mg or 1000 mg DHA	No
	Muthayya et al. (2009)	548	1 year of modified diet	—	900 mg ALA or 140 mg ALA/100 mg DHA	No
	Ryan & Nelson (2008)	175	4 weeks	—	400 mg DHA	Yes, Cognitive
Adulthood	Antypa et al. (2009)	54	4 weeks	—	250 mg DHA/1740 mg EPA	No
	Fontani et al. (2005)	33	5 weeks	—	800 mg DHA/1600 mg EPA/400 mg other n-3s	Yes, Mood & Cognitive
	Yehuda et al. (2005b)	196	3 weeks	Test anxiety	225 mg ALA/900 mg LA	Yes, Anxiety
Later Adulthood	Dangour et al. (2010)	748	24 months	—	400 mg DHA/200 mg EPA	No
	Quinn et al. (2010)	295	18 months	Alzheimer's disease	2000 mg DHA	No
	Yurko-Mauro et al. (2010)	437	24 weeks	Age-related cognitive decline	900 mg DHA	Yes, Cognitive

Note. ALA =  $\alpha$ -linolenic acid; LA = linoleic acid; DHA = docosahexaenoic acid; EPA = eicosapentaenoic acid.

Table 2  
Participant Demographic Characteristics

	Fish oil (N = 20)		Placebo (N = 21)	
	n	%	n	%
Sex				
Male	5	25.00	7	33.33
Female	15	75.00	14	66.66
Race				
White	19	95.00	17	80.95
Hispanic	1	5.00	2	9.52
Black	0	—	1	4.76
Non-specified	0	—	1	4.76
Drug Use (Freq. not reported)				
Nicotine	4	20.00	1	4.76
Marijuana	0	—	1	4.76
Hormonal Birth Control*				
(n and % of female participants only)	3	20.00	8	57.14
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age	19.90	1.83	20.43	1.63
Years of Education	14.05	1.05	14.38	1.12
Hours of Exercise/Week	6.10	3.27	7.02	4.29
Alcoholic Drinks/Week	1.24	2.11	2.65	3.48
Caffeinated Drinks/Day	0.88	0.65	0.69	0.56
Fish/Week				
Baseline	1.00	0.95	0.76	1.30
Post-Treatment	1.03	1.29	1.33	1.79
Physical Condition				
Baseline	5.10	0.97	5.26	0.70
Post-Treatment	5.10	1.17	5.43	0.81
Mental Condition				
Baseline	5.60	1.10	5.43	0.87
Post-Treatment	5.30	0.98	5.19	1.17
Sleep Quality				
Baseline	4.70	1.13	3.95	1.12
Post-Treatment	4.45	1.39	4.19	1.44

Note. Fish consumption per week was self-rated on a 5-point Likert scale in average times per week (i.e., 0, 1–2, 3–4, 5–6, 7 or more). Physical condition, mental condition, and sleep quality for the day of assessment were self-rated on a 7-point Likert scale (1 = low rating and 7 = high rating).

\* At  $p < .05$ , hormonal birth control use among female participants yielded the only significant group difference at baseline.

recruited from a university subject pool and the treatment of participants conformed to the guidelines of the American Psychological Association. This research was approved by an institutional review board.

## Materials

The cognitive test battery included the Rey Auditory Verbal Learning Test (RAVLT; Lezak, 1995), the Stroop Color and Word Test (SCWT; Golden & Freshwater, 2002), and the Trail Making Test (TMT), Parts A and B (Corrigan & Hinkeldey, 1987; Gaudino, Geisler, & Squires, 1995; Lezak, 1995). The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) was also included to assess affect.

**RAVLT.** This measure consisted of seven stages, plus an interference stage. Stages 1 through 5 required recall of a word list read to participants before each stage, collectively measuring verbal learning and memory (Lezak, 1995). The interference stage

involved recall of a second list, while stage 6 entailed recall of the initial list directly after the interference stage. Stage 7 required recall of the initial list after a 20-min delay. Two summary measures derived from the difference between two stages. Summary measures 6–5 and 7–5 indicated the influence of the interference word list and the 20-min delay on recall of the initial list, respectively.

**SCWT.** This measure served as a traditional measure of inhibition (MacLeod, 1991; Stroop, 1935). All stages involved reading as many items as possible within 45 seconds. In the word stage, all items were color words (e.g., red, blue) written in black ink. In the color stage, all items were identical (i.e., XXXX), but written in different colors of ink. In the color-word stage, all items were color words written in an incongruent color of ink. Participants read the color of the ink for the latter two stages. Interference scores were based on raw and predicted color-word scores. *T* scores were adjusted scores based on age and years of education.

**TMT Parts A and B.** This task was included as a general measure of executive control (Arbuthnott & Frank, 2000). In the TMT, shorter times indicated better performance. The TMT Part A required participants to connect 25 numbered dots in numerical order, and Part B required them to alternate between 12 alphabetized and 13 numbered dots (i.e., 1-A-2-B-3-C).

**PANAS.** The PANAS consisted of 20 words describing feelings and emotions, with 10 positive (e.g., determined, strong) and 10 negative (e.g., hostile, scared) words. Each item was rated on a 5-point Likert scale (1 = very slightly or not at all and 5 = extremely) and the positive and negative scores were established by summing their respective items, providing a summary measure for both positive and negative affect (Watson et al., 1988).

**Supplementation materials.** The two supplement conditions consisted of 1000 mg fish oil capsules (240 mg DHA/360 mg EPA) and 1000 mg coconut oil placebo capsules, aligning with the placebo used by Amminger et al. (2007). Each participant received enough capsules to consume two capsules per day for four weeks of treatment. Participants also received a personal log of dated boxes for them to mark daily after supplementation.

## Procedure

Individuals interested in participation contacted the researchers via email to schedule an appointment for a baseline assessment. In response, the researchers informed participants of the two supplement conditions and exclusion criteria. Eligible participants scheduled an appointment on campus where they met with the researchers. Upon giving signed informed consent, participants completed a short demographic questionnaire and underwent a baseline assessment consisting of the RAVLT, SCWT, TMT Parts A and B, and PANAS.

All results were coded randomly by participant number, which indicated the experimental condition of the participants. Odd numbers resulted in a placebo condition and even numbers resulted in a fish oil condition, resulting in an equal chance of assignment to either condition. The researchers and participants were blinded of their supplement conditions. Each participant received 56 capsules for the 4-week supplementation period. The researchers adopted this length of supplementation from research on populations of similar ages (Antypa et al., 2009; Fontani et al., 2005; Yehuda et

al., 2005b). Posttreatment assessments were scheduled exactly 28 days after the baseline assessments.

The participants consumed two capsules of their supplement each day before 7:00 p.m. at a consistent time convenient for their schedule throughout the 4-week period. Participants monitored their own supplementation and contacted the researchers daily via text message to confirm supplementation. The researchers logged all text confirmations. If confirmation was not received by 7:00 p.m., the researchers contacted nonreporting participants once via text message as a reminder. Each participant also kept a daily log as another reference point for confirming treatment compliance.

Exactly 28 days after baseline, participants came to their follow-up appointments at a similar time of day to their initial assessment. Participants completed the same tests and measures from the baseline assessment, using alternative wordlists for the RAVLT (as cited in Lezak, 1995). The researchers then acquired participants' personal logs and asked whether the participants consumed all supplements, recording their responses. Thereafter, the participants were debriefed and informed of their supplement condition.

## Results

Between text message confirmations and personal logs, participants accounted for 99% of the supplementation. Participants included in analysis confirmed taking all 56 capsules over the 28-day treatment through either their personal logs or verbal confirmation (see Table 3, for mean scores and standard deviations for all measures used in the analysis). Due to sphericity issues,

Greenhouse-Geisser adjustments were used on all data analyzed. Each separate analysis involved a repeated-measures ANOVA, with each analysis including two conditions (i.e., fish oil and coconut oil), two measurement times (i.e., baseline and posttreatment), and varying numbers of test stages or scales (e.g., RAVLT Stages 1 to 5) depending on the assessment tool. Therefore, analyses were univariate or multivariate depending on the outcome measure.

## Outcome Measures

**RAVLT.** There was no significant main effect for supplement condition on RAVLT performance in Stages 1 through 5,  $F(1, 39) = 1.38, p = .25$ , and no interaction between condition, test stage, and time of measurement,  $F(3.71, 144.61) = .766, p = .54$ . Due to their unique meaning, Stages 6 and 7 were analyzed separately, each through a univariate repeated-measures ANOVA. This analysis of Stage 6 yielded an interaction between condition and time of measurement,  $F(1, 39) = 4.45, p = .04, \eta^2 = .10$ , but did not yield a main effect for condition,  $F(1, 39) = 1.94, p = .17$ . The same analysis for Stage 7 resulted in similar outcomes, with an interaction between condition and time of measurement,  $F(1, 39) = 5.65, p = .02, \eta^2 = .13$ , but no main effect for condition,  $F(1, 39) = .42, p = .52$ . For both interactions, fish oil performance improved from baseline, but placebo performance declined.

The RAVLT summary measures also have unique meanings and were analyzed in the same way as Stages 6 and 7. A significant interaction between condition and time of measurement occurred for Summary Measure 7–5,  $F(1, 39) = 4.30, p = .045, \eta^2 = .10$ ,

Table 3  
*Mean and Standard Deviations of Participant Performances and Ratings*

Measure	Score type	Submeasure	Baseline				Post-treatment			
			Fish oil		Placebo		Fish oil		Placebo	
			<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
RAVLT	Raw	1	6.55	2.09	7.10	1.41	6.75	1.45	7.00	1.38
		2	9.20	2.17	9.52	2.20	9.30	1.89	9.90	1.70
		3	11.15	2.58	11.24	2.34	10.60	2.41	11.62	1.94
		4	11.50	2.59	12.38	2.11	11.50	2.56	12.38	2.04
		5	12.05	2.16	12.62	1.94	12.35	2.37	12.86	1.56
	Interference	6	6.00	1.86	5.71	1.38	6.50	2.21	6.05	1.77
		7	9.85	3.28	11.57	2.36	10.80	3.14	11.33	2.06
		7	10.00	3.43	11.48	2.96	10.50	3.61	10.29	3.26
	Summary	6–5	–2.20	2.33	–1.05	1.88	–1.55	1.39	–1.52	1.66
		7–5	–2.05	2.37	–1.14	2.48	–1.85	1.79	–2.57	2.54
SCWT	Raw	Word	102.35	14.88	105.10	10.86	110.25	17.55	110.52	12.01
		Color	77.80	12.94	78.29	11.06	83.25	14.75	82.00	10.44
		Color-Word	50.85	11.06	49.38	9.44	56.00	10.10	53.43	10.55
	Interference	Interference	6.95	6.87	4.86	6.99	9.20	6.62	6.76	8.73
		Word	47.35	10.36	48.33	7.21	52.95	11.94	52.67	7.63
		Color	48.15	10.66	48.24	8.76	52.65	12.18	51.43	8.11
		Color-Word	53.85	10.62	52.19	8.89	58.80	9.77	56.10	9.97
	T-Score	Interference	56.95	6.87	54.86	6.99	59.20	6.62	56.76	8.73
		Part A	15.83	3.05	16.78	3.87	14.27	3.00	16.01	4.66
		Part B	32.57	9.36	43.32	18.08	28.61	6.95	35.54	14.04
TMT	Raw	Part A	0.11	0.32	0.05	0.22	0.21	0.42	0.10	0.44
		Part B	0.35	0.59	0.48	0.75	0.55	1.10	0.33	0.73
	Errors	Part A	36.95	5.74	35.71	5.73	37.10	4.61	36.00	5.72
		Part B	19.50	5.61	17.90	4.86	18.35	6.24	16.95	5.23
PANAS	Summary	Positive	36.95	5.74	35.71	5.73	37.10	4.61	36.00	5.72
		Negative	19.50	5.61	17.90	4.86	18.35	6.24	16.95	5.23



where fish oil performance improved from baseline, but placebo performance declined. No main effect occurred for condition on summary measure 7–5,  $F(1, 39) = .02, p = .88$ . The same analysis for the 6–5 summary measure yielded no interaction between condition and time of measurement,  $F(1, 39) = .229, p = .14$ , and no main effect for condition,  $F(1, 39) = 1.79, p = .19$ .

**TMT.** For TMT performance analysis, an additional participant's performance data was removed due to failure to follow testing directions. Both groups improved their performance on the TMT from baseline,  $F(1, 38) = 5.47, p = .03, \eta^2 = .13$ . A main effect was observed for condition on TMT performance: Placebo condition improved performance significantly over fish oil,  $F(1, 38) = 5.92, p = .02, \eta^2 = .14$ , as mean placebo completion time decreased significantly more than mean fish oil completion time. The analysis indicated no interaction for condition, test stage, and time of measurement,  $F(1, 38) = 1.31, p = .26$ .

**SCWT.** No main effect occurred for condition,  $F(1.92, 74.96) = .12, p = .88$ , and no interaction occurred between condition, test stage, and time of measurement,  $F(1, 39) = .01, p = .926$ . The same analysis of *T* scores indicated similar results to that of the raw scores, with no main effect for condition,  $F(1, 39) = .10, p = .75$ , and no interaction between condition, test stage, and time of measurement,  $F(1.89, 73.78) = .01, p = .99$ . Identical analyses of raw and *T* interference scores yielded equivalent results, with no main effect for condition,  $F(1, 39) = 1.22, p = .28$ , and no interaction between condition and time of measurement,  $F(1, 39) = .03, p = .87$ .

**PANAS.** Analyzing the positive and negative summary scales of the PANAS, no main effect was observed for condition,  $F(1, 39) = 2.12, p = .15$ , nor was there an interaction between condition, scale (i.e., positive and negative), and time of measurement,  $F(1, 39) = .001, p = .98$ .

## Discussion

Most results indicated no cognitively beneficial properties of fish oil over placebo, and the few positive results of this study must be interpreted with caution. The only main effect observed for condition occurred on the TMT, where the placebo condition improved significantly over the fish oil condition. However, this result occurred with moderate significance (i.e.,  $p = .02$ ) and likely derived from a learning effect, as no previous research has attributed a cognitive benefit to coconut oil. The other positive results occurred in favor of fish oil, but occurred via an interaction rather than a main effect for condition. Further confounding these findings, the predominant number of participants were women, and considering the 4-week treatment period, the menstrual cycle may have influenced cognitive performance.

Previous research has identified inconsistent menstrual-cycle influences on verbal memory and inhibition (Gordon & Lee, 1993; Hatta & Nagaya, 2009; Keenan, Lindamer, & Jong, 1995). In turn, significant group differences in hormonal contraceptive use at baseline make hormone levels a potential confounding variable. Unfortunately, contraceptive use did not serve as a covariate in analyses, as information pertaining to the hormonal contraceptives used was unavailable. Without biochemical data on hormone levels, any interaction between hormones and cognition could not be determined, but this variable should be monitored in future n-3 PUFA research using similar treatment durations.

Aside from hormonal influences, the predominantly null results may derive from design-related concerns. The 4-week timeframe and the daily dosage of DHA and EPA may have been subtherapeutic, a common problem with n-3 PUFA supplementation studies (Heinrichs, 2010). Although comparable study durations have procured positive results (Fontani et al., 2005; Yehuda et al., 2005b), the study design did not verify biochemical absorption of the nutrients, and relied on self-supplementation. Previous researchers have related small changes in phospholipids to cognitive changes (Kirby et al., 2010; Ryan & Nelson, 2008), and such measurements, had they been used, may have secured different results.

Without biochemical data, the baseline n-3 PUFA level in participants was unknown. If participants already had healthy n-3 PUFA supplies, supplementation effects may have been minimal. Previous studies on n-3 PUFA and attention-deficit/hyperactivity disorder have described similar design-related shortcomings (Sinn & Bryan, 2007). As n-3 PUFA deficiency correlates with attention-deficit and hyperactive symptoms (Colquhoun & Bunday, 1981; Burgess, Stevens, Zhang, & Peck, 2000), a population with sufficient n-3 PUFA may not gain from supplementation. In the current design, without baseline n-3 PUFA data, any participant deficiencies could not be determined.

## Cognitive Results

The cognitive impact of n-3 PUFA supplementation remains unclear across the life span (Antypa et al., 2009; Dangour et al., 2010; Fontani et al., 2005; Helland et al., 2003; Kennedy et al., 2009; Makrides et al., 2010; Ryan & Nelson, 2008; Yurko-Mauro et al., 2010). The current study results showed limited cognitive effects of n-3 PUFA, as interactions between condition and RAVLT measures (i.e., Stages 6 and 7, summary Measure 7–5) seemed to derive more from performance declines with placebo than improvement with fish oil. Similar to the results of Antypa et al. (2009), few measures indicated any effect of the fish oil treatment, bringing into question the results of Fontani et al. (2005). Previous research is consistent with this trend, as few positive effects of n-3 PUFA have been reported prior to later age (Karr et al., 2011). More extensive studies must examine a young-adult population in order to verify this claim.

## Affective Results

The current findings present no influence of n-3 PUFA on either the positive or negative affect of nonclinical college-aged students. A recent meta-analysis reported no evidence of mood alteration through n-3 PUFA treatment among populations without depression (Appleton, Rogers, & Ness, 2010). The current study did not have participant psychiatric information available, which limits the interpretation of mood-related results. Patients with depression have significantly lower n-3 PUFA in their cell phospholipids (Maes et al., 1999), and although clinical populations might respond to n-3 PUFA treatments, nonclinical populations may have sufficient n-3 PUFA, making them insensitive to nutritional interventions.

## Future Directions in Neuro-Nutritional Research on n-3 PUFA

Although n-3 PUFA produced no positive effect in this study, most supplementation research designs yield null results (Antypa et al., 2009; Dangour et al., 2010; Kennedy et al., 2009; Makrides et al., 2010; Quinn et al., 2010); however, designs measuring reported diet have established neuroprotective qualities of n-3 PUFA at later age (Eskelinen et al., 2008; Kalmijn et al., 2004; Solfrizzi et al., 2006). Supplementation research on younger adult populations have used consistently short treatment durations, although lengthier treatments with larger samples have led to null results in different age groups (Kennedy et al., 2009; Quinn et al., 2010). Fontani et al. (2005) stands as the longest supplementation study examining an adult population prior to later age, with just a 5-week treatment and 33 participants. Although they reported positive findings, the results may have derived from design-related flaws (Antypa et al., 2009). Neuro-nutritional researchers have yet to extensively study young adults, but the research trend indicates limited cognitive benefits of n-3 PUFA after childbirth and prior to later age (Karr et al., 2011).

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