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Neurobehavioral Effects of Consuming Dietary Fatty Acids

G Lindseth, RN, PhD, FADA, FAAN[1] and **T Petros, PhD**[1]

[1]University of North Dakota, Grand Forks, ND, USA

Abstract

Background—Research results on the neurobehavioral effects of consuming dietary fatty acids are mixed. Therefore, this study examined the effects of consuming dietary fatty acids on depression, mood, and anxiety.

Methods—In this randomized crossover-design study, 37 university students served as their own controls, consuming each of the following diets for a 4-day period with a 2-week washout period between diets: (1) low fatty acid, (2) high saturated fatty acid (SFA), (3) high polyunsaturated fatty acid (PUFA), and (4) control. The order of sessions was counterbalanced across dietary groups. Following consumption of each diet, participants were examined for within-subject differences in depression, mood, and anxiety. Measures included weighed dietary fat intakes, Zung's Self-Rating Anxiety and Depression Scales, and the Positive and Negative Affect Schedule.

Results—Participants had significantly higher positive affect scores ($p < .007$) and were significantly less irritable ($p < .04$) when they consumed diets rich in SFAs and PUFAs than when they consumed a low fatty acid or control diet. However, depression, anxiety, and negative affect scores did not differ significantly among diets. Analysis of participants' serum lipid levels following their intake of the fatty acid and control diets indicated significantly higher levels of total cholesterol ($p = .006$) and serum triglycerides ($p = .003$) with the control diet.

Conclusions—These results highlight the neurobehavioral benefits of consuming dietary fatty acids among healthy individuals. By concentrating on the positive effects of diet on affective processes, health professionals can also provide support for at-risk individuals.

Keywords

fatty acids; anxiety; mood; depression

The National Institute of Mental Health (NIMH) indicates that 15.7 million adults in the United States experienced a major depressive disorder in 2014 (NIMH, 2015a), and it is

Corresponding Author: Glenda Lindseth, RN, PhD, FADA, FAAN, University of North Dakota, Grand Forks, ND, USA. glenda.lindseth@und.edu.

Author Contribution

Glenda Lindseth contributed to conception, design, and acquisition; drafted and critically revised the manuscript; gave final approval; and agrees to be accountable for all aspects of work ensuring integrity and accuracy. Thomas Petros contributed to acquisition, analysis, and interpretation; critically revised the manuscript; gave final approval; and agrees to be accountable for all aspects of work ensuring integrity and accuracy.

Declaration of Conflicting Interests

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estimated that 18% of U.S. adults were affected by mental illness in 2014 (NIMH, 2015b). As a result, researchers have sought to elucidate the causes of mental illnesses and to identify modifiable risk factors, such as diet, to determine effective approaches for addressing the prevalence and negative effects of mental illness.

Over the last decade, studies have increasingly recognized the role that dietary lipids play in neuronal functions of the brain (Brown & Murphy, 2009). Lipids are a class of organic compounds that include fatty acids. Some studies have shown that consuming diets deficient in fatty acids may be associated with affective mood disorders (Brinkworth, Buckley, Noakes, Clifton, & Wilson, 2009; Moran et al., 2013; Stahl, Albert, Dew, Lockovich, & Reynolds, 2014; Sublette, Hibbeln, Galfalvy, Oquendo, & Mann, 2006). However, other studies have indicated that consumption of fatty acids such as polyunsaturated fatty acids (PUFAs) including omega-3 (n-3) fatty acids resulted in no significant neurobehavioral effects (Hakkarainen et al., 2004; Nanri et. al., 2014; Rogers, 2008).

Membrane lipids, a group of compounds that includes cholesterol, serve important functions in the brain. They form a double-layered cell surface that separates the inner and outer cellular environments (Van Meer, Voelker, & Feigenson, 2008; Trebaticka & Durackova, 2014). Membrane lipids are also involved in cell signaling. However, because some fatty acids are not synthesized by the human body, they must be obtained directly from the diet (e.g., linoleic and linolenic acid; King, 2015). Dietary fatty acid supplementation may prevent oxidative neuronal injury that can result in abnormal mood and emotional behavior. For example, n-3 (PUFA) fatty acids are thought to mediate membrane fluidity and affect neurotransmitter transmission. Neurotransmitter pathway dysfunction is thought to be the molecular basis for depression (Trebaticka & Durackova, 2014). Research has shown that changes in the diet can alter the lipid composition of the brain, which may have long-term consequences on mood and emotional behavior (Müller et al., 2015). Effects of PUFA (specifically, n-3 fatty acid) supplementation on mood and behavior may be attributed to cell membrane fluidity that can modulate brain neurotransmitter function (Puri, Counsell, Hamilton, Richardson, & Horrobin, 2001; Rogers et al., 2008). Thus, mood may be susceptible to dietary fatty acids.

Another study examined the possible cerebral hemodynamic effects of PUFA supplementation, specifically an n-3 fatty acid-rich fish oil supplement, on 22 healthy adults (Jackson, Reay, Scholey, & Kennedy, 2012). Investigators measured changes in prefrontal cortex oxygenation, while the participants completed computerized cognitive tasks. The n-3 supplementation with docosahexaenoic acid-rich fish oil, in comparison to the placebo, resulted in significantly increased oxygen-hemoglobin levels, indicating increased cerebral blood flow during the tasks.

In a recent study of 106 participants (mean age 50 years), a sustainable decrease in both anxiety and depression scores resulted from a low-fat diet used for weight loss in comparison to a high-fat diet after 8 weeks (Brinkworth et al., 2009; Moran et al. 2013). Over the long term (8–52 weeks), subjects on the low-fat diet maintained low anxiety scores and improved depression levels, but those on the high-fat diet reverted to baseline levels of anxiety and depression. Meanwhile, using the Stress Profile Questionnaire, Ness et al.

(2003) found no differences in anxiety or depression in 452 male participants from baseline to 6 months after they began consuming either fatty fish or fish oil capsules (high in PUFAs) to ameliorate their cardiac conditions.

Using health surveys and self-reported diet history questionnaires, Nanri et al. (2014) found no significant relationship between depressive symptoms and fatty acid (PUFA) intakes of 1,794 Japanese male workers. In a Finnish study of 29,000 men, Hakkarainen et al. (2004) similarly found no association between fatty acid consumption and low mood levels when using self-reported measures of mood, hospital discharge data, and diet history questionnaires. Also, in a 12-week double-blind, randomized trial, 190 mildly to moderately depressed individuals received either one of two essential PUFA (n-3) supplements or a placebo daily (Rogers et al., 2008). Using the Beck Depression Inventory to measure mood states, researchers found no significant differences between subjects taking the fatty acid treatments and those taking the placebo.

In contrast to the previous findings, Whitaker, Sharpe, Wilcox, and Hutto (2014) found that symptoms of depression were positively associated ($p < .05$) with dietary intakes of saturated fats as measured by 24-hr dietary recalls. In a study of 33 medication-free patients with depression, Sublette, Hibbeln, Galfalvy, Oquendo, and Mann (2006) found that low fatty acid levels were predictive of suicidal tendencies. In total, seven patients with low n-3 (PUFA) lipid profiles committed suicide over a 2-year period.

Previous studies have thus had mixed results when examining high PUFA (Hakkarainen et al., 2004; Nanri et al, 2014; Rogers et al., 2008; Stahl et al., 2014; Sublette et al., 2006) and saturated fatty acid (SFA) diets (Whitaker, Sharpe, Wilcox, & Hutto, 2014) and diets deficient in fatty acids for effects on mood and neurobehavioral conditions such as depression and anxiety. In addition, less work has been focused on the neuro-behavioral effects of consuming diets high in SFAs, despite the significant consumption of diets high in saturated fats in our society. Therefore, our study examined the effects of consuming diets high in PUFAs; diets high in SFAs; and diets low in fatty acid on depression, mood, and anxiety in healthy young adults.

Method

Study Design

In this counterbalanced study with a within-subjects design, we examined differences in mood, depression, and anxiety of participants following their consumption of an SFA diet, a PUFA diet, and a low fatty acid diet. We compared observations for each of these dietary intakes to baseline observations (control diet). To ensure that order of treatment was not a concern for the interpretation of findings, we mitigated any potential effects by (1) allowing a full 2 weeks of ‘‘washout’’ time between the dietary treatments and (2) randomly assigning order of dietary treatments to the participants. During the washout periods, participants resumed their typical diets. There were 24 possible orders in which the 4-day dietary treatments and control diet could be given. We assigned the treatment and control diets randomly, without replacement to the 36 participants, using the following procedure: (1) We typed two copies of each order on slips of paper; (2) We placed both copies of each

order into a container and mixed them well; (3) As participants enrolled in the study, we drew a slip from the container, and; (4) We recorded the selected order for each participant and implemented diets accordingly. After enrolling all of the participants, we discarded the 12 slips of paper that remained.

During the first week, participants met with the nurse-researcher to complete demographic questionnaires and determine baseline health, anthropometric, and indirect calorimetry measurements as well as dietary and health histories. We instructed all participants on the proper protocols for completing each dietary treatment session; the assessments of mood, depression, and anxiety; and the laboratory tests. We also provided instructions to enhance compliance in completing the study diets. Times for consuming the study meals were arranged according to the scheduling requirements of the study participants.

Population Description and Sampling Plan

Study participants were Midwestern university students in their third semester of study. The academic programs targeted for this study cumulatively accommodated about 350 potential participants each year. In an initial meeting, we explained the study to potential participants and invited them to participate. We answered their questions and had eligible participants sign the consent form. To be included in the study, students had to meet the following selection criteria: (1) be enrolled within the first 4 weeks of the third term of study at the university; (2) have given consent to participate in the study; (3) be 18–35 years of age; and (4) be able to read, understand, and speak English.

We excluded potential participants from consideration if any of the following conditions were present: pregnancy diagnosis, diabetes diagnosis, circulatory limitations, respiratory limitations, neurosensory limitations, nutritional or metabolic limitations such as gluten or lactose intolerance, elimination limitations, mobility limitations, current skin problems, reproductive (sexuality) limitations, endocrine limitations, or history of mental disorders. In addition, taking prescription or over-the-counter medications was an exclusion criterion. However, taking aspirin or noncodeine acetaminophen was allowed if prescribed by the study's health-care provider.

Based upon a plan to use repeated-measures analysis of variance (ANOVA) statistics for the study, we calculated statistical power using the *Power and Precision* software package (Version 3) by Borenstein, Rothstein, and Cohen (2001). We used previous study information with similar sample populations to determine the potential effects of the dietary intervention plan for this study (Lindseth et al., 2011; Lindseth, Lindseth, & Thompson, 2013). Our goal was to achieve a statistical power of .80, an a of .05, and a ''medium'' effect size of .30. To meet this goal, we needed an estimated sample size of 30 participants. Investigators in similar neurobehavioral studies also calculated a medium effect size to be . 25 to .30 (Lindseth et al., 2011; Lupien, Gillin, & Hauger, 1999). Taking into account a possible 20% attrition rate, we estimated that our actual sample size would need to be a minimum of 36 participants. Over three semesters, we recruited 40 participants into the study. A total of three participants did not complete the study, two due to time constraints and one whom we eliminated because of a medical condition. Therefore, a total of 37 participants completed the study.

The institutional review boards of both the university and the Human Research Protections Office of the U.S. Army Medical Research and Materiel Command, the primary funding agency for this study, approved this study. We provided a small stipend of US$25 to each participant following completion of study interviews as an expression of appreciation for their participation.

Dietary Treatments

Using indirect calorimetry, we determined food portions for each individual in order to provide daily kilocalorie requirements with dietary fat calculated as percentages of the total daily kilocalorie intakes. We took a dietary history into consideration for each participant along with an assessment of their likes and dislikes as we planned the study diets. In addition, we used advice from a consulting dietitian with extensive experience in conducting intervention studies in planning the study diets. As described above, we randomly assigned participants to different 4-day dietary treatment schedules, with each participant serving as his or her own control. Table 1 presents the nutrient compositions of the four study diets: (1) control diet, (2) low fatty acid diet, (3) high saturated fat diet, and (4) high polyunsaturated fat diet.

The university dining services prepared the food under the guidance of the study's research dietitian and a dietary consultant. While being monitored by the study staff, participants consumed all meals in the study's metabolic dining room at the Northern Plains Center for Behavioral Research located on the university's campus. This dining room was specifically designed to conduct behavioral research studies, such as this one, that require close monitoring of research participants. The study dietitian provided all recipes and exact portion sizes. In addition to planning the meals to meet the nutrient composition of each study diet, the dietitian calculated dietary micronutrient intakes to meet the U.S. Recommended Dietary Allowances (U.S. RDA), within 5% error, to minimize potential effects of micronutrient deficiencies. We monitored both food preparation and food consumption carefully to maintain high-quality analyses of dietary intakes. The identification of the study diet was double blinded to both the participants and the psychologist who administered the study affective measures, and the meals were sufficiently similar among diets to ensure that participants could not distinguish between the control and the treatment diets.

To measure food consumption, the study dietitian and staff weighed each food serving, both before and after the meals. Weighed food intakes are considered the best method for measuring food consumption (Gibson, 1990). Participants received beverages with their meals and between meals to maintain hydration. These included (1) water and calorie-free drinks, with a limit of 200 mg of caffeine per day; (2) juices or milk containing fat, protein, and carbohydrate calories that contributed to the meal; and (3) liquid snacks. Snacks were planned for consumption during the evening and distributed with instructions to ensure that they were only eaten as prescribed. We provided bottled water with the snacks and recorded its consumption with the fluid intake data. We instructed all participants to consume only food, water, and other beverages provided by the study staff. Participants provided daily written confirmation that they had not consumed any food or beverages outside of the

prescribed study diets for that treatment week. In addition, we reminded participants that violations could result in dismissal from the study.

Tests and Measurements

In addition to daily food consumption, we measured the participants' mood, depression, irritability, and anxiety levels and related demographic, anthropometric, health assessments, and biochemical lab test data 4 times during the study, as described below. We validated and tested instrumentation in a pilot study to ascertain reliability. Both the original authors of the tools and the present study investigators tested the assessment tools for both reliability and validity. We deemed this process necessary to justify the inclusion of the proposed instruments and variables into the study.

Neurobehavioral measures—We used the Positive Affect Negative Affect Schedule (PANAS) to measure mood (Watson, Clark, & Tellegen, 1988). The PANAS comprises 20 words related to how respondents are feeling: 10 describing positive mood states (positive affect) such as pleasure, joy, and self-assurance; and 10 describing negative mood states (negative affect) such as sadness, guilt, hostility, fear, and anxiety. Respondents indicate how closely each word described their current mood on a scale ranging from 1 (*very slightly or not at all*) to 5 (*extremely*). Two scores, one for each scale, are calculated, with higher scores indicating greater affect. When Watson and Clark (1994) measured the internal consistency of the PANAS in an Upper Midwestern sample population, they observed an α coefficient of .90.

We used Zung's Self-Rating Anxiety Scale (SAS) to quantify the participants' anxiety levels (Zung, 1980). Respondents rate 20 items that describe their anxiety within the past 24 hr on a scale ranging from 1 (*not during that time period*) to 4 (*most or all of the time*). The index score is calculated by dividing the total score by 80 and then multiplying by 100. The reliability coefficient (α) for this scale was .85 for the present study. In previous studies, the reliability coefficients have ranged from .61 to .80 (Bucky & Spielberger, 1973).

To measure depression levels, we used Zung's Self-Rating Depression Scale (SDS; Zung, 1965). Respondents rate 20 items that describe depressive symptoms, indicating to what extent they had experienced each symptom over the past 24 hr, ranging from 1 (*least depressed*) to 4 (*most depressed*). An index score is determined by dividing the total score by 80 and then multiplying by 100. The reliability coefficient for this scale was .91 for the present study. A previous study showed a reliability coefficient of .85 for depression measurements in a study involving 415 undergraduate students (Campbell, Maynard, Roberti, & Emmanuel, 2012). Authors calculated a validity coefficient of .70 in a study involving 152 patients compared to the Minnesota Multiphasic Personality Inventory (MMPI; Zung, 1967).

We used the Undergraduate Affective Irritability subscale to measure irritability. Sakamoto Kijima, Tomoda, and Kambara (1998) selected, modified, and tested five questions related to irritability from Zung's (1965) original SDS to develop this subscale. Responses range from 1 (*least irritable*) to 4 (*most irritable*). The index score is determined by dividing the total score by 20 and then multiplying by 100 (Sakamoto Kijima, Tomoda, & Kambara, 1998).

The reliability coefficient for the Undergraduate Affective Irritability subscale was .86 for the present study.

Demographics and health assessments—We collected demographic data from participants', including ethnicity, age, educational levels, marital/living status, employment status, and place of residence.

We used the Block Food Frequency Questionnaire (Block, Wakimoto, Jensen, Mandel, & Green, 2006) to obtain qualitative, descriptive information about food consumption patterns and nutrient intakes for the participants. These data provided information about the participants' diet history during the year prior to the study.

To assess each participant's baseline health status, we used the Doenges' Health Assessment Checklist (modified from Doenges, 1989). The checklist comprises a medical history examination to determine the following factors: (1) circulatory limitations, (2) respiratory limitations, (3) neurosensory limitations, (4) nutrition limitations, (5) elimination limitations, (6) mobility limitations, (7) current skin problems, (8) reproductive (sexuality) limitations, (9) endocrine limitations, and (10) history of mental disorders. These assessments identified symptoms that might compromise performance outcomes.

We calculated body mass index (BMI, kg/m^2) to analyze associations with other study variables. We measured body weight a mechanical balance-beam scale and height with a wall-mounted stadiometer. We weighed participants 8 times on the first and last days of each dietary treatment week. The anthropometric measurements (weight and height) had reliabilities in excess of 0.97 (Marks, Habicht, & Mueller, 1989), according to the Second National Health and Nutrition Examination Survey.

At the end of each study week and within 2 hr following consumption of the last SFA, PUFA, low fatty acid, or control diet study meal, we drew blood for serum lipid measures and analyzed them for each study participant. Biochemical and laboratory measures included serum triglycerides, total cholesterol, and high-density lipoprotein (HDL), and low-density lipoprotein (LDL) levels. We drew these samples immediately prior to or following the neurobehavioral measurements. The same licensed personnel performed all lab tests in order to ensure consistency and to facilitate validity of the laboratory measures.

Data Collection and Analysis

We provided participants with verbal directions for completing the dietary treatment sessions; mood, depression, anxiety, and irritability assessments; and laboratory tests to enhance compliance with completing the study protocols. We carefully monitored study meals to ensure that participants ate all of the foods served. On the fourth day of each treatment diet, participants met with the nurse-researcher and psychology researchers to complete the mood, anxiety, irritability, and depression scales; health assessments; and laboratory tests.

We recorded data from the psychological and health assessments on spreadsheets using a double entry procedure to minimize data-entry errors. For data analyses, we used the SPSS

statistical program (Version 23) and the Explore Procedure, taking an α coefficient of ≤ .05 as the criterion for statistical significance. We analyzed the weighed food intakes with the Food Processor System (Elizabeth Stewart Hands and Associates [ESHA] Research, 2010). We selected this system for its ability to measure intakes of micronutrients and macronutrients. It also includes intake of caffeine. Food intake analyses were based on the U.S. RDA. The primary statistics that we used for this study include descriptive statistics and repeated-measures ANOVA calculations.

Results

Demographic and Anthropometric Data

The study participants had a mean age of 20.9 years (SD = 1.9) and had completed an average of 14.2 years of education (SD = 0.9). The participants' BMIs had a mean of 24.7 kg/m^2 (SD = 4.2) just prior to the first week of the study and a mean of 24.8 kg/m^2 (SD = 4.0) on the last day of the study. Table 2 presents the demographic and anthropometric data on the participants.

Effects of Fatty Acid Consumption on Anxiety

We measured anxiety levels of the participants upon completion of each dietary treatment week using Zung's SAS and analyzed anxiety scores using repeated-measures ANOVA calculations. Anxiety scores did not differ significantly across study diets (Table 3).

Positive and Negative Affect and Irritability Scores Following Fatty Acid Consumption

We measured the effects of fatty acid consumption on mood using the PANAS and evaluated these scores using repeated-measures ANOVA calculations. We found significant differences in the basic positive affect (mood) scores (F = 4.3, p = .007) among the SFA, PUFA, low fatty acid, and control diet periods (Table 3). Further analysis using the Tukey's post hoc test indicated that the SFA and PUFA diets resulted in significantly (p < .05) higher basic positive affect scores in comparison to the low fatty acid and control diets. The negative affect scores, however, did not significantly change with the different diets (Table 3).

We measured the irritability scores of the study participants using the Undergraduate Affective Irritability Scale. Repeated-measures ANOVA revealed that irritability scores differed significantly (F = 3.0, p = .035) among study diets (Table 3). The Tukey's post hoc analysis showed no significant differences in irritability scores between the individual diets.

Effects of Fatty Acid Consumption on Depression

Participants' scores on Zung's SDS did not differ significantly across the high SFA, high PUFA, low fatty acid, and control diets (Table 3).

Effects of Fatty Acid Consumption on Serum Lipid Levels

We drew serum samples for laboratory analysis of total cholesterol, HDL, LDL, and triglyceride levels at the end of each dietary treatment week. Total cholesterol (F = 4.4, p = .006) and serum triglyceride levels (F = 4.9, p = .003) differed significantly across the four study diets (Table 4). A post hoc Tukey's test indicated that the control diet resulted in

significantly higher total cholesterol and serum triglyceride ($p < .05$ for both) values in comparison to when the participants consumed the low fatty acid, SFA, and PUFA diets. However, HDL and LDL levels did not differ significantly among the study diets.

Discussion

Our findings support the results of previous studies showing that diets high in fatty acids improve affect scores (Stahl et al., 2014; Sublette et al., 2006). As discussed above, other studies have concluded that consumption of fatty acids such as PUFAs, including n-3 fatty acids, results in no significant neurobehavioral effects (Hakkarainen et al., 2004; Nanri et al., 2014; Rogers, 2008).

Depression and Fatty Acids

Our finding that depression was not significantly affected by consumption of dietary fatty acids was consistent with those of other studies. Hakkararainen et al. (2004) examined 29,000 men to determine whether n-3 fatty acid (PUFA) intake was associated with low mood levels or depression and found no association. Nanri et al. (2014) reported similar findings in a study of 1,794 Japanese male workers that examined the relationship between depressive symptoms and fatty acid (PUFA) intake. Two other studies involving SFAs reached different conclusions than our research. Le Port et al. (2012) found that, over 10 years, 9,272 men had an increased probability of depressive symptoms when consuming high-SFA ‘‘Western diets.’’ In Whitaker et al. (2014), symptoms of depression correlated significantly ($p < .05$) with increases in saturated fat intake. Yet Sublette et al. (2006) found that low fatty acid levels were associated with depression in medication-free patients with suicidal tendencies. This finding was similar to those of other studies in which increased intakes of n-3 and n-6 fatty acids were associated with a decreased likelihood of depression (Daley, Patterson, Sibbritt, & MacDonald-Wicks, 2015; Stahl et al., 2014). A recent meta-analysis of data from 23 different studies involving almost 5,000 participants with different oxidative stress markers suggested that oxidative stress is increased and antioxidant defenses are decreased in depression (Palta, Samuel, Miller, & Szanton, 2014). Oxidative stress has been associated with increased lipid peroxidation (Puri, Tsaluchidu, & Treasaden, 2009). Clinical evidence has shown that n-3 fatty acids have the potential to serve as a dietary treatment for oxidative stress; thus, dietary supplementation with fatty acids may be beneficial in reducing depression. Some recent research also pointed to dairy products (which often contain higher amounts of SFA) having a beneficial effect on both oxidative stress and reduction of oxidative LDL levels. However, there has been no consensus on the most appropriate biomarkers of oxidative stress; thus, more research is needed.

Anxiety and Positive and Negative Affect and Fatty Acids

In the present study, neither the amount nor the type of fatty acid in the diet significantly affected anxiety scores. In contrast, Brinkworth, Buckley, Noakes, Clifton, and Wilson (2009) found that there was a sustainable decrease in both anxiety scores and depression scores after 8 weeks with a low-fat diet in comparison to a high-fat diet. However, over the long term (8–52 weeks), participants on the high-fat diet reverted to baseline levels of anxiety and depression. In a large Australian epidemiological study, Daley, Patterson,

Sibbritt, and MacDonald-Wicks (2015) reported results that contradict those of both the present study and Brinkworth's study, finding that participants who increased intakes of n-3 (linoleic) and n-6 fatty acids had decreased anxiety.

In contrast to our findings on anxiety, we did find that the participants in the present study had significantly different basic positive affect (mood) scores ($p = .007$) when they consumed the SFA and PUFA diets in comparison to the time periods in which they consumed the low fatty acid and control diets. Although the repeated measures ANOVA indicated significant differences in irritability among diets, the post hoc test indicated no significant differences between particular diets.

Serum Lipid Levels and Dietary Fatty Acids

Given that the fat content of the SFA and PUFA study diets was 65% of the daily kilocalories consumed, it is surprising that consumption of these diets did not significantly affect the participants' HDL or LDL levels. Previous studies have indicated that when dietary fat intake (especially intake comprising SFAs) reached 40% or more of the daily kilocalories consumed, there was a significant effect on serum lipid levels (Keys, Anderson, & Grande, 1957; Mensink & Katan, 1990). There were, however, significant differences in the present study in participants' total cholesterol ($p = .006$) and serum triglyceride ($p = .003$) levels following their consumption of the study diets, with the post hoc test indicating that the control diet resulted in significantly higher cholesterol and triglyceride levels than the other study diets. These results may be explained by the differing amounts of carbohydrates in the control diet compared to the high-fat SFA and PUFA diets. The glycemic load and fiber content of these diets could also have impacted cholesterol levels (Hare-Bruun, Nielsen, Grau, Oxlund, & Heitmann, 2008). Because dietary fiber can affect serum lipid levels and glycemic load, the protein and carbohydrate content of the control diet may have also affected the serum lipid levels. On the other hand, this explanation may be confounded by the fact that the low-fat diet was more similar on these measures to the SFA and PUFA diets than it was to the control diet. Thus, further study with considerations for the dietary fiber and glycemic content of the study meals is recommended.

Strengths and Limitations

This study had several strengths. Most previous studies used fatty acid supplements (Rogers et al., 2008; Sublette et al., 2006) or self-reported dietary intakes (Hakkarainen et al., 2004; Le Port et al., 2012; Tsai et al., 2014) when examining the neurobehavioral effects of fatty acids. In contrast, in the present study, we carefully controlled food intake by monitoring and weighing the prepared food items consumed by each participant. Also, this study is one of the few to focus on the effects of dietary fatty acid in healthy individuals. Moreover, previous studies have produced mixed results and some conflicting conclusions. Therefore, although the outcomes of the present study were consistent with those reported in some previous studies (Hakkarainen et al., 2004), our conclusions are based on strong evidence due to our study controls.

An important limitation of this study was the small, homogeneous sample. Thus, our conclusions may have limited generalizability. However, to mitigate this effect, we had the

participants serve as their own controls. While our sample size of 37 participants is smaller than those of some previous studies (Hakkarainen et al., 2004; Nanri et al., 2014; Ripoll et al., 2015; Rogers et al., 2008), our study was based on the direct weighing and controlling of the nutritional intakes of the study participants. As mentioned above, other studies have involved the use of nutritional supplements (Nanri et al., 2014; Rogers et al., 2008) or self-reported food consumption questionnaires (Daley et al., 2015; Hakkarainen et al., 2004; Ripoll et al., 2015). Although these methods are less expensive and enable a larger sample size, they also tend to produce data that are less conclusive than data obtained by weighed, controlled food intake. In addition, the short time period for each dietary treatment session may be a limitation of the present study. Other limitations may include the lack of the use of biomarkers to demonstrate separate alterations in the fatty acid composition of cell membranes and the absence of a quantification of how much fatty acid made up the total plasma fatty acid pool or use of gas chromatography to assist in determining how dietary changes in cell membranes can influence fatty acid composition.

Conclusion

The results of our study indicate that increased intake of dietary fatty acids (particularly n-3 enriched PUFAs and SFAs) had beneficial effects on irritability and mood among healthy participants. Also, surprisingly, consumption of the SFA and PUFA diets imposed in this study resulted in significantly lower serum cholesterol and triglyceride levels in comparison to the control diet. These findings, along with the Food and Drug Administration's recommendation that SFA consumption be limited to less than 10% of the total daily caloric intake (McGuire, 2011), suggest that further research would be useful. We also recommend additional research on the neurobehavioral effects of consuming dietary fatty acids and the possible confounding effects of inflammation that may be associated with consuming high levels of dietary fatty acid is also recommended.

The impact that diet has on the mental and emotional health of human beings makes additional research vital to promoting a healthier society. The results of this and prior studies suggest that diets rich in fatty acids may help prevent neurobehavioral disorders. Health professionals can provide evidence-based support and informed counseling regarding dietary intake to both healthy individuals and individuals at risk for affective disorders.

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Table 1

Nutrient Content of Study Diets

Diet	Fat, %	Carbohydrate, %	Protein, %
Control diet	35	50	15
Low fatty acid diet	10	80	10
High saturated fat diet (35% SFA)	65	25	10
High polyunsaturated fat diet (1.3 mg PUFA)	65	25	10

Note. PUFA = polyunsaturated fatty acid; SFA = saturated fatty acid.

Table 2

Sample Characteristics

Variable	Mean (SD)
Age (years)	20.9 (1.9)
Education (years)	14.2 (0.9)
Height (in.)	69.6 (3.6)
Baseline weight (lb)	170.4 (33.3)
Baseline BMI (kg/m^2)	24.72 (4.2)
Final weight (lb)	170.6 (32.9)
Final BMI (kg/m^2)	24.75 (4.0)

Note. $N = 37$. BMI = body mass index.

Table 3

Within-Subject Differences in Depression, Anxiety, Irritability, and Mood Scores Following Consumption of the Four Study Diets.

	Control Diet	Low-Fat Diet	High-SFA Diet	High-PUFA Diet		
Measure	**Mean (*SD*)**	**Mean (*SD*)**	**Mean (*SD*)**	**Mean (*SD*)**	***F***	***p***
Depression	40.1 (8.9)	41.0 (10.0)	39.1 (6.7)	39.1 (6.7)	1.6	.189
Anxiety	37.8 (8.1)	37.8 (6.4)	37.1 (6.0)	36.8 (6.3)	0.75	.525
Irritability	34.0 (9.6)	34.2 (9.8)	31.9 (7.2)	31.6 (6.9)	3.0	.035
Basic positive affect	17.5 (4.1)	16.1 (4.1)	18.0 (3.9)[a]	18.1 (3.6)[a]	4.3	.007
Basic negative affect	7.8 (2.1)	8.1 (2.7)	7.8 (2.4)	7.7 (2.2)	0.48	.695

Note. $N = 37$. $df = 3$. $p < .05$ indicates significance. PUFA = polyunsaturated fatty acid; SFA = saturated fatty acid.

[a]Post hoc test results: High-SFA and PUFA diets resulted in significantly ($p < .05$) higher basic positive affect scores.

Table 4

Within-Subject Differences in Serum Lipid Levels Following Consumption of Control, Low-Fat, High Saturated Fatty Acid (SFA), and High Polyunsaturated Fatty Acid (PUFA) Diets

	Control Diet	Low-Fat Diet	High-SFA Diet	High-PUFA Diet		
Serum Lipid	**Mean (*SD*)**	**Mean (*SD*)**	**Mean (*SD*)**	**Mean (*SD*)**	***F***	***p***
Total cholesterol	153.3 (27.5)[a]	145.0 (23.0)	144.2 (22.4)	143.7 (23.2)	4.4	.006
High-density lipoprotein	44.9 (13.5)	43.2 (18.4)	43.0 (13.3)	43.0 (13.8)	0.37	.773
Low-density lipoprotein	82.0 (26.1)	78.1 (16.0)	79.3 (21.1)	80.0 (20.0)	0.43	.733
Triglycerides	136.2 (77.5)[a]	101.2 (63.7)	111.1 (64.7)	110.6 (65.5)	4.9	.003

Note. $N = 37$. $df = 3$. $p < .05$ indicates significance.

[a]Post hoc test results: The control diet resulted in significantly ($p < .05$) higher total cholesterol and serum triglyceride levels in comparison to the low-fat, high-SFA, and high-PUFA diets.