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Allen K.L., Mori T.A., Beilin L., Byrne S.M., Hickling S. & Oddy W.H. (2013) Dietary intake in population-based adolescents: support for a relationship between eating disorder symptoms, low fatty acid intake and depressive symptoms. *J Hum Nutr Diet.* 26, 459–469 doi:10.1111/jhn.12024

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Dietary intake in population-based adolescents: Support for a relationship between eating disorder symptoms, low fatty acid intake, and depressive symptoms

Abstract

Background: Relatively little is known about the dietary intake and nutritional status of community-based individuals with eating disorders. The current research aimed to (i) describe the dietary intake of population-based female adolescents with an eating disorder, and (ii) examine associations between eating disorder symptoms, fatty acid intake, and depressive symptoms in adolescents with and without an eating disorder. **Methods:** Participants (N=429) were female adolescents aged between 16 and 18 years, drawn from the Western Australian Pregnancy Cohort (Raine) Study. **Results:** Adolescents with an eating disorder (n=66) reported a significantly lower intake of total fat, saturated fat, omega-6 fatty acid, starch, Vitamin A, retinol, and Vitamin E when compared to adolescents without an eating disorder (n=363). Adolescents with an eating disorder and pronounced depressive symptoms (n=23) also reported a significantly lower intake of polyunsaturated fat and omega-3 and omega-6 fatty acid than adolescents with an eating disorder but no marked depression (n=43). In the eating disorder sample, but not the control sample, omega-3 and omega-6 fatty acid correlated significantly and negatively with eating disorder symptoms and with depressive symptoms. **Conclusions:** Support is provided for a relationship between low omega-3 and omega-6 fatty acid intake and depressive symptoms in adolescents with eating disorders. Research is needed to examine the feasibility and effectiveness of fatty acid supplementation in this high-risk group.

Introduction

Eating disorders, including anorexia nervosa (AN), bulimia nervosa (BN), and eating disorders not otherwise specified (EDNOS), are serious mental illnesses that affect up to 10% of women across their lifetime, as well as a smaller percentage of men (Wade et al., 2006). Eating disorders are associated with considerable psychosocial and physiological morbidity and have the highest mortality rate of any psychiatric disorder, equal with substance abuse (Berkman et al., 2007).

By definition, eating disorders are associated with disordered eating practices. However, relatively little is known about the dietary intake and nutritional status of community-based individuals with eating disorders. This is problematic for two reasons. First, if nutritional deficiencies are common amongst individuals with eating disorders, this information should be disseminated so that appropriate monitoring and management (e.g., supplementation) can be undertaken when an eating disorder is suspected or diagnosed. Second, there is growing support for an association between diet, particularly fatty acid (FA) intake, and depressive symptoms. As major depressive disorder frequently co-occurs with eating disorders (Lewinsohn et al., 2000; Zaider, et al., 2002), identifying possible associations between dietary variables and depressive symptoms in this population is important.

Studies with clinical samples have found intake of protein, calcium, iron, ascorbic acid, retinol, Vitamin D, Vitamin B12, folate, zinc, magnesium, and copper may be below recommended levels in AN (Beumont et al., 1981; Hadigan et al., 2000), whilst intake of iron and zinc may be below recommended levels in BN (Gendall et al., 1997; Woell et al., 1989). Low cholesterol levels have also been documented in AN, and found to correlate significantly with depressive symptoms and suicidality (Favaro, et al., 2004). The ratio between tryptophan (necessary for serotonin synthesis) and other large neural amino acids appears to increase in AN as caloric intake, body mass index (BMI) and body fat decrease (Favaro et al., 2000). More recently, investigation of omega-3 (n-3) and omega-6 (n-6) FA status in adolescents with eating disorders revealed that adolescents with eating disorders and pronounced depressive symptoms had significantly lower levels of n-3 FA, and a significantly higher ratio of n-6 to n-3, than adolescents with eating disorders but no pronounced depressive symptoms (Svenne et al., 2011).

Associations between FA status and depressive symptoms have also been identified in the general population. Population-based cohort studies show a high intake of 'whole' foods rich in n-3 FA (e.g., nuts, fish) to be protective against depression in adulthood (Akbaraly et al., 2009; Sanchez-Villegas et al., 2009), whilst a low intake of these foods, and/or a high intake of processed foods (e.g., refined grains, fried foods), may

prospectively predict depression in mid-life (Akbaraly et al., 2009; Sanchez-Villegas et al., 2009). Recently, Oddy and colleagues (Oddy et al., 2011) used data from the Western Australian Pregnancy Cohort (Raine) Study to extend these findings to an adolescent sample. Significant negative associations were found between FA intake at age 14 and depressive symptoms at ages 14 and 17. However, these relationships ceased to reach statistical significance after BMI and other covariates were controlled for, making further research important. Information on dietary intake at age 14 was also obtained via parent-report, which may have led to under- or over-reporting for adolescents with disordered eating practices.

The current study aimed to integrate research on dietary intake in eating disorders with research on FA intake and depression. Specifically, it aimed to:

1. Examine the dietary intake of population-based adolescents with eating disorders, relative to controls,
2. Compare FA intake across eating disordered adolescents with and without depressive symptoms,
3. Examine associations between eating disorder symptoms, FA intake and depression, and
4. Test a mediational model that positions FA intake as a partial mediator of associations between eating disorder and depressive symptoms.

Method

Design

As with Oddy et al. (Oddy et al., 2011), this research utilised data from the Raine Study. This study has been described previously (Allen et al., 2009; Newnham et al., 1993; Oddy et al., 2011). In brief, 2900 women were recruited from the antenatal booking clinics at King Edward Memorial Hospital for women (KEMH), the only public maternity hospital in Western Australia, between May 1989 and November 1991. Of the 2,900 women enrolled, 2,804 delivered live birth babies. Due to 64 multiple births, the initial cohort included 2,868 children. These children were assessed at birth and ages one, two, three, five, eight, 10, 14, and 17 years.

Unlike Oddy et al. (2011), the current study focused exclusively on data from the 17-year follow-up and from female participants. The decision to focus on the 17-year assessment stemmed from all information collected at this time point being adolescent self-report. The decision to exclude male participants stemmed from expected sex differences in the nature and degree of association between eating disorder symptoms and dietary intake. Further, only 11 of the 455 male Raine Study participants who provided eating disorder and dietary data at age 17 met criteria for an eating disorder.

Participants

Participants were female adolescents who took part in the 17-year Raine Study follow-up and who completed questionnaire measures regarding dietary intake, eating disorder symptoms, and depression. This equated to 454 participants, or 46% of the female Raine Study sample eligible to take part at age 17. Non-participation at the 17-year follow-up was not significantly associated with 14-year eating disorder status ($\chi^2[3]=1.86, p=.603$) and a one-way ANOVA confirmed that 14-year eating disorder and depressive symptom scores did not differ significantly across participants who did and did not provide data at age 17 ($p=.486 - .805$).

Of the 454 participants who provided data, 66 (15%) met full ($n=18$; 4%) or partial ($n=48$; 11%) criteria for a *DSM-IV* eating disorder at age 17. These adolescents formed the eating disorder sub-group. An additional 23 participants (5%) reported sub-threshold eating disorder symptoms (e.g., occasional binge eating or purging without other symptoms) and were excluded from analyses. The remaining 363 participants (80%) did not report significant eating pathology and formed the non-eating disorder control group.

The mean age of the sample was 16.92 (SD 0.12, range=15.7–18.2yrs). This did not differ significantly across the eating disorder ($M[SD]=16.92 [0.19]$) and control ($M[SD]=16.92 [0.26]$) groups ($p=.881$).

Measures

Eating disorder symptoms. Eating disorder symptoms were assessed using 24 items adapted from the Child Eating Disorder Examination (Bryant-Waugh et al., 1996) and Eating Disorder Examination-Questionnaire (Fairburn & Beglin, 1994). Items assessed for *DSM-IV* diagnostic criteria for AN, BN and EDNOS, and for dietary restraint and eating, weight and shape concerns more generally. The eating disorder group included participants who met full or partial criteria for a *DSM-IV* eating disorder on the basis of their responses to these items, in conjunction with measured BMI and self-reported menstruation status.

Eating disorder cases were further classified according to diagnostic sub-type: full and partial AN, BN, Binge Eating Disorder (BED), or Purging Disorder (PD). Although BED and PD are classified under the broader EDNOS category in *DSM-IV*, they are increasingly used for research purposes (Stice et al., 2009; Wade et al., 2006). Provisional diagnostic criteria for BED are included in the *DSM-IV* and provisional criteria for PD are provided by Keel and colleagues (Keel et al., 2005). The number of participants in each diagnostic sub-group (including full and partial cases) was as follows: $n=6$ for AN, $n=38$ for BN, $n=6$ for BED, and $n=15$ for PD.

A global eating disorder symptom score was calculated by taking the mean of items relating to dietary restraint and eating, weight and shape concerns.

Additional information about the eating disorder items and classification process is available in Allen et al. (2009).

Depressive symptoms. Depressive symptoms were assessed using the Beck Depression Inventory for Youth (BDI-Y) (Beck et al., 2001). This self-report measure has been established as reliable and valid for children and adolescents aged 7 to 18 years (Beck et al., 2001; Stapleton et al., 2007; Steer et al., 2001), and assesses for cognitive and behavioural depressive symptoms experienced over the past two weeks.

Dietary variables. Dietary information was obtained using the Commonwealth Scientific and Industrial Research Organisation (CSIRO) Food Frequency Questionnaire (FFQ), which has been previously applied and validated in the Raine Study cohort (Ambrosini et al., 2009; Oddy et al., 2011). This self-report measure asks respondents to indicate their typical intake of 212 individual foods, mixed dishes, and beverages over the previous year. Consumption frequencies are made against standard serving sizes and response options range through never, rarely, number of times per month, number of times per week, and number of times per day. Respondents are also asked to indicate if their typical serving size differs from the example provided, which was measured in household units (e.g., cups, slices). Information was also collected about foods eaten outside of the FFQ, cooking methods, fat types, and whether or not low-fat versions and fresh, frozen or canned foods were consumed.

The questionnaire generates estimates of intake of all macro- and micronutrients, including specific polyunsaturated FA (total n-3, total n-6, and individual n-3 and n-6 FA). The database for composition of FA in foods was compiled by the CSIRO, drawing on information from the Australian nutrient database (NUTTAB), British food tables, the US Department of Agriculture food tables, and the food industry (Yearsley et al., 1999).

The FFQ has been validated in adults and children (Hodge et al., 1996; Oddy et al., 2004; Rohan et al., 1987). In addition, 14-year FFQ data in the Raine Study cohort were validated against a 3-day, comprehensive diet record, with good convergence, providing further support for the utility of the measure in this sample (Ambrosini et al., 2009).

Procedure

Questionnaire packages were posted to adolescents for at-home completion prior to attendance at a face-to-face assessment session. Height and weight were measured during this assessment. If required, research nurses were also available to assist with questionnaire completion at this time.

Data collection occurred in accordance with Australian National Health and Medical Research Council Guidelines for Ethical Conduct in Human Research and was approved by the ethics committees of KEMH and Princess Margaret Hospital for Children.

Statistical analyses

Although global eating disorder scores and BDI-Y scores were positively skewed, skewness values were in the low to moderate range (range=0.81 – 1.04), and were consistent with the application of clinically-focused measures to a community sample. Thus, data were not transformed prior to analysis.

To address Aim 1, intake of macro and micronutrients was compared across the eating disorder and non-eating disorder groups. Intake of specific FA was considered in addition to total fat and total n-3 and n-6. Non-parametric tests were used in recognition of uneven group sizes.

For micronutrient intake, the proportion of participants in each group who reported intake less than two-thirds below the Australian RDI (where available) was also considered for each vitamin and mineral. The use of two-thirds the RDI is consistent with previous studies (e.g., Gendall et al., 1997), and allows for some acceptable variation around the absolute RDI value.

For Aim 2, the eating disorder sample was divided into high and low depression groups on the basis of BDI-Y scores, using established cut-points (Beck et al., 2001; Stapleton et al., 2007). Subsequently, non-parametric tests were used to compare fat intake across participants with scores in the normal-mild range (0 – 20) and those with scores in the moderate-high range (≥ 21).

For Aim 3, correlations were examined between global eating disorder symptom scores, fat intake, and BDI-Y scores. Again, non-parametric tests were utilised. Correlations were examined separately for participants with and without eating disorders.

For Aim 4, a series of regression analyses was conducted to determine if FA intake mediated the relationship between eating disorder and depressive symptoms. Consistent with previous studies (Oddy et al., 2011; Sanchez-Villegas et al., 2009), BMI and family income were included as covariates. Analyses followed the steps outlined by Baron and Kenny (Baron & Kenny, 1986) and are consistent with recommendations regarding mediational analyses in population-based research (Hafeman & Schwartz, 2009).

Results

Preliminary analyses: Eating disorder diagnostic differences

One-way ANOVAs confirmed there were no significant differences in reported intake of any macro- or micronutrient group across the full and partial eating disorder cases ($p=.100 - .964$), or across the eating disorder diagnostic subgroups ($p=.559 - .996$).

Only 17% ($n=11$) of the eating disorder group reported being diagnosed or treated for an eating disorder in their lifetime, suggesting that dietary data are relatively unaffected by clinical intervention.

Aim 1: Dietary intake for eating disordered and control participants

Macronutrients. The eating disorder group reported a significantly lower intake of total, monounsaturated and saturated fat, total n-6 FA, alpha linolenic acid (ALA) 18:3 FA, and starch when compared to the control group. They reported a significantly greater intake of alcohol (see Tables 1 and 2). With the exception of alcohol intake (which was elevated in the BN, BED and PD subgroups), between-group differences in macronutrient intake were largely accounted for by the AN and BN subgroups. All differences remained significant after adjusting for BMI

Individuals with BN also reported a significantly greater intake of refined sugars than the control group, and a significantly lower intake of long-chain FA (see Table 1).

Micronutrients. The eating disorder group reported a significantly lower intake of Vitamin A, retinol, and Vitamin E than the control group (see Table 3). Again, these differences remained significant after adjusting for BMI. Group-specific differences included a lower intake of sodium and cholesterol in the AN group, a lower intake of Vitamin B12 in the BN group, and a lower intake of Vitamin C in the BED group, when compared to control participants (see Table 3).

Chi square tests indicated that a significantly greater proportion of eating disordered than control participants scored less than two-thirds the Australian RDI for zinc (8% vs. 1% respectively, $p < .001$), thiamine (14% vs. 4%, $p < .001$), Vitamin B6 (12% vs. 4%, $p = .012$), and phosphate (17% vs. 7%, $p = .008$).

Aim 2: Fat intake across eating disordered participants with and without depressive symptoms

When applying BDI-Y cut-points, 35% ($n=23$) of the eating disorder sample were classified as having moderate or high depressive symptoms. Compared to participants with an eating disorder and scores in the normal-mild range ($n=43$), these adolescents reported a significantly lower intake of polyunsaturated fat ($p=.015$), total n-3 FA ($p=.012$), total n-6 FA ($p=.004$), and six specific n-3 and n-6 FA ($p=.003 - .039$). Median fat intake is shown in Figure 1 for each group, for polyunsaturated fat and total n-3 and n-6 FA.

Aims 3 and 4: Associations between eating disorder symptoms, fat intake, and depressive symptoms

Spearman Rho correlations between eating disorder symptom scores, BDI-Y scores, and fat intake are summarised in Table 4.

For the non-eating disorder sample, correlations between fat intake and eating disorder and depressive symptoms were weak and non-significant. Thus, no further analyses were conducted and possible mediational relationships were not considered.

For the eating disorder sample, eating disorder scores correlated significantly and negatively with most fat variables (see Table 4). Scores on the BDI-Y correlated significantly and negatively with polyunsaturated fat, total n-3 FA, total n-6 FA, and four specific FA. In light of these results, mediational models were tested in the eating disorder sample for polyunsaturated fat, total n-3 FA, and total n-6 FA. Eating disorder symptom scores were the predictor variable in each instance and BDI-Y scores formed the dependent variable (see Table 5).

None of the models was able to satisfy the first criteria for mediation. After accounting for BMI and family income, eating disorder symptom scores were no longer significantly associated with polyunsaturated fat, total n-3 FA, or total n-6 FA. Polyunsaturated fat and total n-6 FA did continue to significantly predict BDI-Y scores after covariates were considered.

Discussion

This study examined associations between eating disorder symptoms, FA intake, and depressive symptoms in a population-based sample. Female adolescents who met full or partial criteria for an eating disorder reported a significantly lower intake of total fat, saturated fat, total n-6 FA, starch, Vitamin A, retinol, and Vitamin E when compared to control participants from the same population. Adolescents with an eating disorder and pronounced depressive symptoms also reported a significantly lower intake of polyunsaturated fat, total n-3 FA, and total n-6 FA when compared to participants with an eating disorder but no marked depressive symptoms. In the eating disorder sample, but not the control sample, eating disorder symptoms and depressive symptoms correlated significantly and negatively with n-3 and n-6 FA intake.

This is the first study to provide comprehensive data on dietary intake in a population-based sample of adolescents with eating disorders. Consistent with clinical studies, participants with an eating disorder reported a significantly lower intake of several macro and micronutrient groups when compared to control participants. A significant minority of the eating disorder sample also reported intakes of zinc, thiamine, Vitamin B6, and phosphate that were more than two-thirds below the Australian RDI. As most participants in the eating disorder group were in the healthy weight range, and between-group differences persisted when BMI was controlled for,

these differences cannot be attributed to low weight status. This highlights the importance of attending to possible physiological consequences of disordered eating in adolescence, even when weight appears normal.

Intake of alcohol in the eating disorder group was significantly higher than in the control group. This finding is consistent with research on impulsive behaviour in eating disorders, and the documented overlap between eating (and depressive) disorders and drug and alcohol misuse (Krug et al., 2009; Patton et al., 2003). The results suggest that screening for alcohol abuse may be warranted when assessing or treating binge eating or purging in adolescence.

As with Swenne et al.'s (2011) recent findings with clinical participants, adolescents with an eating disorder and pronounced depressive symptoms reported a significantly lower intake of polyunsaturated FA, including n-3 and n-6 FA, than adolescents with an eating disorder but no marked depression. This pattern of results has implications for the assessment and management of depression in individuals with eating disorders. Supplementation of FA may, for example, serve to improve mood, even in the early stages of eating disorder treatment when patients may be unwilling to engage in broader dietary change. Further research is required to assess this possibility, and to determine if changes in FA intake predict changes in depression.

In the control group, fat intake did not correlate significantly with eating disorder or depressive symptoms. This contrasts with previous theoretical and empirical work (Akbaraly et al., 2009; Blakaj, 2010; Sanchez-Villegas et al., 2009). In the eating disorder sample, however, low fat intake correlated significantly with eating disorder and depression scores. This may suggest that associations between FA intake and depression are evident only when FA intake is particularly low, as seen in eating disorders. This would make attention to disordered eating an important consideration when conducting research in this area.

The associations between eating disorder symptoms and FA intake in the eating disorder sample did not remain statistically significant when covariates were controlled for. As such, support was not provided for a mediational relationship between eating disorder symptoms, FA intake, and depressive symptoms. Notably, however, the negative relationship between n-6 FA and depressive symptoms did persist when covariates were considered. This lends further support to the potential relevance of FA intake to depressive symptoms in individuals with eating disorders. The finding also suggests that further research to test the proposed mediational relationship, including research with larger samples, may be justified.

This study has a number of strengths, including the use of a population-based sample and comprehensive dietary data. It is the first study to examine dietary intake in population-based adolescents with eating disorders. Limitations of the research include the reliance on self-reported dietary intake, and combining

FFQ data with biochemical markers of nutrient intake and FA status (e.g., blood samples) would be optimal for addressing this issue. The use of self-report data for eating disorder and depressive symptoms also deserves note, and small group sizes for the eating disorder diagnostic subgroups prevent conclusions being drawn about possible diagnostic differences. The modest overall eating disorder sample size may have contributed to insufficient power for the mediational analyses. Finally, prospective analyses would be optimal for extending the findings reported here, and are essential if any causal relationships are to be delineated.

In summary, new evidence is provided for a relationship between low n-3 and n-6 FA intake and depressive symptoms in adolescents with eating disorders. A similar relationship was not identified for participants without eating pathology. Although FA intake did not reach statistical significance as a mediator of associations between eating disorder and depressive symptoms in this sample, additional research to explore this relationship in a larger sample of individuals with eating disorders seems warranted.

Acknowledgements

We are extremely grateful to all the families who took part in this study; to the whole Raine Study team, which includes data collectors, cohort managers, data managers, clerical staff, research scientists and volunteers; to the National Health and Medical Research Council of Australia for their long term contribution to funding the study; and to the Telethon Institute for Child Health Research for their long term support of the study.

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Figure 1.

Median and upper ranges for polyunsaturated, omega-3 (n-3) and omega-6 (n-6) fatty acid (FA) intake, for female adolescents with an eating disorder and either low-mild scores on the Beck Depression Inventory for Youth (BDY-Y) (n=43) or moderate-high scores on the BDI-Y (n=23).

Table 1.

Median scores (and ranges) for Body Mass Index (BMI), kilocalorie day, and macronutrient intake/day (in grams), for the eating disorder and non-eating disorder groups

	1. Non-eating disorder controls (n=363)	Eating disorder cases					Differences ¹
		2. All eating disorders (n=66)	2a. AN (n=6)	2b. BN (n=38)	2c. BED (n=6)	2d. PD (n=15)	
Body Mass Index	21.89 (16.09 - 44.31)	23.10 (16.26 - 50.16)	17.80 (16.26 - 18.47)	23.20 (18.61 - 50.16)	23.33 (19.61 - 32.76)	24.15 (19.98 - 36.26)	2 > 1; 2a < 1; 2b, 2c, 2d > 1
Total kilocalories	1956.3 (1202.8 - 4836.5)	1786.6 (812.9 - 4183.7)	1557.4 (1107.0 - 1975.3)	1818.8 (812.9 - 4183.7)	2202.5 (1559.2 - 3114.5)	1783.3 (978.1 - 3301.7)	
Protein	79.7 (27.4 - 198.4)	75.6 (22.6 - 222.2)	71.9 (39.5 - 95.6)	71.1 (22.6 - 222.2)	100.1 (70.7 - 126.1)	81.1 (29.2 - 179.8)	
Total carbohydrate	228.6 (91.4 - 590.6)	216.3 (104.7 - 465.8)	218.5 (144.2 - 310.3)	206.3 (124.7 - 465.8)	275.7 (163.3 - 370.6)	246.3 (104.7 - 368.4)	
Natural Sugars	63.7 (13.1 - 245.7)	60.7 (10.4 - 177.0)	52.3 (20.1 - 161.4)	61.6 (10.4 - 177.0)	61.8 (31.0 - 131.4)	61.3 (28.8 - 109.6)	
Refined sugars	58.6 (1.2 - 300.9)	65.4 (28.9 - 74.0)	52.3 (12.8 - 71.2)	67.9 (0.6 - 157.1)	64.2 (56.0 - 132.2)	71.1 (11.6 - 156.8)	2b > 1
Starch	100.6 (34.5 - 231.6)	87.7 (28.1 - 175.0)	95.6 (54.8 - 110.33)	83.9 (48.3 - 175.0)	127.1 (76.1 - 169.8)	87.0 (28.1 - 167.1)	2 < 1 2b < 1
Total fat ²	72.1 (21.7 - 217.3)	61.5 (10.4 - 160.0)	39.5 (36.9 - 61.3)	63.2 (10.4 - 143.1)	67.8 (52.0 - 160.1)	61.7 (24.2 - 128.3)	2 < 1 2a, 2b < 1
Polyunsaturated	9.8 (2.5 - 50.5)	8.6 (3.0 - 23.4)	7.4 (5.1 - 15.7)	8.8 (3.3 - 21.9)	9.9 (3.5 - 22.1)	9.0 (3.0 - 23.4)	
Monounsaturated	24.7 (8.2 - 71.4)	20.8 (2.7 - 57.9)	13.6 (11.8 - 20.6)	20.9 (2.7 - 48.1)	23.2 (18.3 - 57.9)	21.5 (8.6 - 47.7)	2 < 1 2a, 2b < 1
Saturated	31.6 (7.0 - 105.0)	26.9 (2.5 - 69.8)	16.7 (6.7 - 28.5)	28.9 (2.5 - 69.8)	31.5 (26.9 - 67.4)	26.2 (9.0 - 59.9)	2 < 1 2b < 1
Alcohol	1.3 (0.01 - 125.6)	5.8 (0.0 - 59.9)	1.9 (0.0 - 20.3)	6.6 (0.0 - 59.9)	13.9 (0.0 - 32.6)	4.5 (0.0 - 35.9)	2 > 1 2b, 2c > 1

Note. Differences are significant at $p < .05$, based on non-parametric tests.

¹ As identified through Mann-Whitney U and Kruskal-Wallis non-parametric tests.

² Specific fatty acids are shown in Table 2.

Table 2.

Median scores (and ranges) for intake of polyunsaturated fatty acids/day (in grams), for the eating disorder and non-eating disorder groups.

	1. Non-eating disorder controls (n=363)	Eating disorder cases					Differences ¹
		2. All eating disorders (n=66)	2a. AN (n=6)	2b. BN (n=38)	2c. BED (n=6)	2d. PD (n=15)	
Total omega-3	0.98 (0.31 - 5.24)	0.93 (0.11 - 2.79)	0.71 (0.56 - 1.01)	0.95 (0.11 - 2.55)	1.09 (0.73 - 2.20)	0.97 (0.19 - 2.29)	
Alpha linolenic acid (ALA) 18:3	0.74 (0.16 - 5.18)	0.60 (0.11 - 2.28)	0.54 (0.50 - 0.65)	0.59 (0.11 - 2.28)	0.79 (0.52 - 2.04)	0.62 (0.25 - 1.80)	2 < 1
Eicosapentanoic acid (EPA) 20:5	0.05 (0.01 - 0.20)	0.05 (0.01 - 0.29)	0.05 (0.01 - 0.08)	0.04 (0.01 - 0.13)	0.06 (0.03 - 0.07)	0.08 (0.01 - 0.29)	2b < 1
Docosapentaenoic acid (DPA) 22:5	0.07 (0.00 - 0.27)	0.06 (0.00 - 0.45)	0.03 (0.02 - 0.09)	0.06 (0.00 - 0.18)	0.08 (0.05 - 0.12)	0.06 (0.02 - 0.45)	2a, 2b < 1
Docosahexanoic acid (DHA) 22:6	0.06 (0.01 - 0.43)	0.05 (0.01 - 0.27)	0.07 (0.01 - 0.27)	0.05 (0.01 - 0.23)	0.05 (0.03 - 0.18)	0.07 (0.01 - 0.21)	2b < 1
Long chain fatty acids (EPA + DPA + DHA)	0.18 (0.01 - 0.68)	0.16 (0.01 - 0.83)	0.18 (0.05 - 0.37)	0.14 (0.01 - 0.41)	0.21 (0.11 - 0.30)	0.27 (0.03 - 0.83)	2b < 1
Total omega-6	7.97 (2.57 - 59.07)	6.98 (2.51 - 20.44)	5.60 (4.68 - 8.54)	6.98 (2.51 - 19.46)	8.33 (3.47 - 16.40)	8.92 (2.58 - 20.44)	2 < 1
Linoleic acid (LA) 18:2	7.83 (2.42 - 39.79)	6.74 (2.39 - 20.03)	5.36 (4.56 - 8.48)	6.57 (2.46 - 19.13)	8.12 (3.14 - 16.16)	7.93 (2.39 - 20.03)	
Eicosadienoic acid (EA) 20:2	0.02 (0.00 - 0.08)	0.02 (0.00 - 0.09)	0.01 (0.00 - 0.01)	0.01 (0.00 - 0.09)	0.02 (0.01 - 0.05)	0.02 (0.00 - 0.08)	2a < 1
Dihomo-gamma-linolenic acid (dGLA) 20:3	0.03 (0.00 - 0.09)	0.03 (0.00 - 0.18)	0.01 (0.01 - 0.03)	0.03 (0.00 - 0.07)	0.03 (0.03 - 0.06)	0.03 (0.01 - 0.18)	2a < 1
Arachidonic acid (AA) 20:4	0.13 (0.00 - 0.46)	0.12 (0.00 - 0.69)	0.06 (0.03 - 0.12)	0.12 (0.00 - 0.34)	0.14 (0.13 - 0.33)	0.12 (0.02 - 0.69)	2a < 1

Note. Differences are significant at $p < .05$, based on non-parametric tests.

¹ As identified through Mann-Whitney U and Kruskal-Wallis non-parametric tests.

Continued below

Table 2 (cont.)

	1. Non-eating disorder controls (n=363)	Eating disorder cases					Differences ¹
		2. All eating disorders (n=66)	2a. AN (n=6)	2b. BN (n=38)	2c. BED (n=6)	2d. PD (n=15)	
Bossepentaenoic acid (BA) 20:5	0.06 (0.00 - 0.43)	0.00 (0.00 - 0.00)	0.00 (0.00 - 0.00)	0.00 (0.00 - 0.00)	0.00 (0.00 - 0.00)	0.00 (0.00 - 0.00)	
Adrenic acid 22:4	0.02 (0.00 - 0.06)	0.01 (0.00 - 0.08)	0.01 (0.00 - 0.01)	0.01 (0.00 - 0.05)	0.14 (0.13 - 0.33)	0.01 (0.00 - 0.08)	2a, 2c < 1
AA:EPA	2.51 (0.40 - 6.98)	2.67 (0.34 - 5.21)	1.85 (0.53 - 4.26)	2.76 (0.34 - 5.14)	2.97 (1.87 - 5.21)	2.37 (1.38 - 4.93)	
AA:DHA	2.47 (0.09 - 8.20)	2.75 (0.12 - 7.34)	1.08 (0.16 - 5.23)	2.96 (0.12 - 7.12)	2.95 (0.73 - 4.67)	2.41 (0.53 - 7.34)	
Omega-6:Omega-3	7.61 (2.98 - 21.96)	7.39 (3.45 - 32.82)	8.36 (5.96 - 8.79)	7.12 (3.65 - 32.82)	7.45 (4.75 - 8.39)	7.11 (3.45 - 14.23)	

Note. Differences are significant at $p < .05$, based on non-parametric tests.

¹ As identified through Mann-Whitney U and Kruskal-Wallis non-parametric tests.

Table 3.

Median scores (and ranges) for micronutrient intake (in mg), across the eating disorder and non-eating disorder groups

	1. Non-eating disorder controls (n=363)	Eating disorder cases					Differences ¹
		2. All eating disorders (n=66)	2a. AN (n=6)	2b. BN (n=38)	2c. BED (n=6)	2d. PD (n=15)	
Fibre	20.9 (6.6 - 58.9)	18.8 (7.9 - 60.4)	18.9 (10.0 - 60.4)	18.2 (7.9 - 48.4)	22.7 (11.9 - 39.2)	19.4 (7.9 - 27.7)	
Nitrogen	12.9 (4.7 - 31.8)	12.1 (3.6 - 35.7)	11.5 (6.6 - 15.6)	11.4 (3.6 - 35.7)	16.2 (11.4 - 20.7)	13.2 (4.8 - 28.8)	
Sodium	2607.0 (976.8 - 6261.6)	2482.3 (575.6 - 6729.3)	1841.6 (1585.1 - 2014.8)	2516.1 (575.6 - 6729.3)	3113.7 (2357.9 - 5319.8)	2565.7 (962.6 - 4990.2)	2a < 1
Potassium	3126.3 (1220.7 - 8050.0)	3101.4 (1388.4 - 6997.0)	2861.8 (1811.5 - 6561.4)	2942.2 (1472.1 - 6997.1)	3083.4 (2032.4 - 5258.0)	3569.9 (1388.4 - 4915.6)	
Calcium	924.2 (176.0 - 2962.8)	942.9 (242.0 - 2448.0)	1020.6 (242.0 - 1368.3)	951.6 (248.2 - 2448.0)	912.6 (655.9 - 1870.2)	907.7 (292.5 - 1791.8)	
Magnesium	268.0 (127.4 - 1028.0)	260.1 (112.4 - 608.8)	260.5 (139.3 - 487.4)	242.9 (125.6 - 608.8)	315.7 (185.7 - 498.1)	278.0 (112.4 - 437.9)	
Phosphorous	1339.9 (498.5 - 3374.5)	1294.6 (455.3 - 3690.8)	1375.5 (607.5 - 1968.2)	1215.2 (482.3 - 3690.8)	1686.6 (1021.7 - 2457.0)	1298.6 (455.3 - 2630.8)	
Iron	11.6 (3.7 - 31.7)	10.6 (4.8 - 24.3)	10.1 (6.8 - 21.7)	10.1 (4.8 - 24.3)	16.1 (9.1 - 17.9)	11.7 (5.4 - 21.1)	
Copper	1.7 (0.8 - 5.0)	1.5 (0.8 - 3.5)	1.4 (1.0 - 3.0)	1.5 (0.9 - 3.5)	1.7 (1.1 - 2.3)	1.7 (0.8 - 2.4)	
Zinc	10.9 (3.3 - 26.1)	9.7 (2.7 - 26.6)	9.1 (4.4 - 13.6)	9.1 (2.7 - 24.0)	11.1 (8.5 - 16.8)	9.9 (4.2 - 26.6)	
Retinol	331.1 (36.6 - 8007.3)	275.2 (56.8 - 708.6)	199.5 (113.5 - 425.7)	278.9 (56.8 - 644.5)	273.1 (227.3 - 708.6)	305.7 (57.6 - 575.2)	2 < 1; 2b < 1

Note. Differences are significant at $p < .05$, based on non-parametric tests. ¹ As identified through Mann-Whitney U and Kruskal-Wallis non-parametric tests.

Continued below

Table 3 (cont.)

	1. Non-eating disorder controls (n=363)	Eating disorder cases					Differences ¹
		2. All eating disorders (n=66)	2a. AN (n=6)	2b. BN (n=38)	2c. BED (n=6)	2d. PD (n=15)	
Carotene	3597.7 (307.0 - 16871.3)	3272.5 (749.1 - 12653.4)	1587.1 (1331.5 - 12653.3)	3330.5 (950.7 - 8459.5)	3031.5 (749.1 - 5819.9)	3225.5 (1438.9 - 6289.6)	
Vitamin D	1.3 (0.01 - 7.5)	0.9 (0.1 - 7.9)	0.8 (0.3 - 7.9)	1.0 (0.1 - 4.2)	0.8 (0.4 - 3.4)	1.4 (0.1 - 3.6)	
Thiamine	1.4 (0.4 - 5.0)	1.4 (0.5 - 3.3)	1.2 (0.8 - 2.1)	1.3 (0.5 - 3.3)	1.9 (1.0 - 2.2)	1.4 (0.6 - 2.33)	
Riboflavin	1.9 (0.4 - 5.4)	2.0 (0.7 - 5.3)	1.9 (0.8 - 3.0)	1.9 (0.8 - 5.3)	2.1 (1.4 - 3.7)	2.2 (0.7 - 3.9)	
Niacin	15.6 (4.6 - 35.6)	15.7 (6.4 - 35.9)	12.1 (10.9 - 25.3)	15.6 (6.5 - 35.9)	22.2 (11.4 - 27.1)	17.3 (6.4 - 35.6)	
Total folic acid	218.7 (60.5 - 852.5)	213.8 (79.8 - 498.2)	230.4 (111.1 - 498.1)	213.8 (79.8 - 420.8)	170.1 (103.5 - 347.9)	219.8 (99.6 - 342.4)	
Biotin	17.9 (3.0 - 64.9)	18.2 (5.6 - 86.8)	15.3 (5.9 - 86.8)	19.2 (5.6 - 31.4)	17.4 (11.5 - 38.4)	18.9 (7.5 - 48.7)	
Cholesterol	227.0 (22.4 - 1114.5)	202.1 (2.9 - 760.6)	134.1 (93.4 - 182.5)	202.1 (2.9 - 452.2)	225.6 (211.4 - 723.2)	240.8 (55.5 - 760.6)	2a < 1
Vitamin A	955.9 (176.4 - 9145.8)	841.4 (369.3 - 2534.6)	464.0 (440.5 - 2534.6)	637.4 (443.2 - 1665.0)	836.6 (398.0 - 1328.9)	887.7 (380.7 - 1354.0)	2 < 1
Vitamin C	140.1 (13.3 - 607.3)	127.2 (32.9 - 465.4)	117.3 (103.2 - 305.6)	141.9 (32.9 - 444.4)	82.03 (41.7 - 136.2)	137.9 (51.9 - 465.4)	2c < 1
Vitamin E	6.3 (1.2 - 21.4)	5.2 (1.8 - 18.5)	4.8 (3.0 - 18.5)	4.9 (2.6 - 16.0)	6.7 (2.0 - 7.3)	6.2 (1.8 - 15.5)	2 < 1
Vitamin B6	1.5 (0.3 - 3.7)	1.4 (0.6 - 3.2)	1.5 (0.8 - 3.2)	1.3 (0.6 - 3.1)	1.4 (1.0 - 2.2)	1.6 (0.7 - 2.3)	
Vitamin B12	3.6 (0.8 - 30.4)	3.3 (0.1 - 10.1)	3.1 (1.1 - 6.5)	3.1 (0.1 - 8.0)	4.7 (2.8 - 5.3)	4.0 (1.1 - 10.1)	2b < 1

Note. Differences are significant at $p < .05$, based on non-parametric tests.

¹ As identified through Mann-Whitney U and Kruskal-Wallis non-parametric tests.

Table 4.

Spearman Rho correlations between fat intake, eating disorder scores and depression scores, for eating disordered and non-eating disordered participants

	Eating disorder score		BDI-Y	
	Non-eating	Eating	Non-eating	Eating
	disorder controls (n=363)	disorder cases (n=66)	disorder controls (n=363)	disorder cases (n=66)
Eating disorder score	-	-	.24**	.39**
BDI-Y	.24**	.39**	-	-
Total fat	-.03	-.30*	.03	-.20
Saturated fat	-.06	-.28*	.04	-.13
Monounsaturated fat	-.03	-.32*	.04	-.18
Polyunsaturated fat	.07	-.29*	-.02	-.29*
Total omega-3	.02	-.29*	-.01	-.30*
Alpha linolenic acid (ALA) 18:3	.04	-.26*	-.01	-.30*
Eicosapentanoic acid (EPA) 20:5	-.01	-.34**	.01	-.08
Docosapentaenoic acid (DPA) 22:5	-.06	-.34**	.02	-.17
Docosahexanoic acid (DHA) 22:6	.05	-.36**	.07	-.03
Long chain FA (EPA + DPA + DHA)	.01	-.30*	.05	-.09
Total omega-6	.08	-.35*	-.01	-.35*
Linoleic acid (LA) 18:2	.09	-.22	-.01	-.36*
Eicosadienoic acid (EA) 20:2	-.03	-.27*	-.03	-.27*
Dihomo-gamma-linolenic acid (dGLA) 20:3	-.04	-.29*	.01	-.22
Arachidonic acid (AA) 20:4	-.03	-.32*	.02	-.27*
Bossepentaenoic acid (BA) 20:5	.07	-.20	.02	-.17
Adrenic acid 22:4	-.04	-.35*	.03	-.20
AA:EPA	-.03	-.02	-.02	-.19
AA:DHA	-.10	.26*	-.05	-.06
Omega-6:Omega-3	.10	.13	.01	-.14

Note. ED=Eating disorder; BDI-Y=Beck Depression Inventory-Youth; YSR=Youth Self Report; FA=Fatty acid

* $p < .05$ ** $p < .01$

Table 5.

Linear regression analyses of mediation for the eating disorder sample (n=66), controlling for Body Mass Index and family income

Mediational chains (IV → MV → DV)	IV → MV	MV → DV	IV → DV	IV + MV → DV ¹
Eating disorder symptoms → polyunsaturated fat intake → BDI-Y depressive symptoms	-0.14	-0.30*	0.32**	0.31*
Eating disorder symptoms → omega-3 fatty acid intake → BDI-Y depressive symptoms	-0.19	-0.19	0.32**	0.32*
Eating disorder symptoms → omega-6 fatty acid intake → BDI-Y depressive symptoms	-0.19	-0.33*	0.32**	0.29*

Note. IV=independent variable; MV=mediator variable; DV=dependent variable; BDI-Y=Beck Depression Inventory-Youth

¹ Reported β is for the IV * $p < .05$ ** $p < .01$