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Omega-3 supplementation lowers ApoB100 levels in obese adolescents: a controlled intervention study

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ABSTRACT

Background: Obesity is a risk factor associated with cardiovascular disease. Apolipoprotein B (ApoB) is the main protein in atherogenic lipoproteins and a strong predictor of cardiovascular risk. Omega-3 fatty acids have been shown to reduce plasma triglycerides and total ApoB levels in individuals with metabolic syndrome. This study aims to analyze the effect of omega-3 administration on ApoB-100 levels in adolescents with obesity.

Methods: This study was an experimental study with a pre- and post-test design, conducted by health students from February to April 2025 in Makassar. A total of 90 individuals met the inclusion criterion of a body mass index (BMI) ≥ 25 kg/m². ApoB-100 levels were measured using the Human Apolipoprotein B1 ELISA Kit, and lipid profiles were assessed using an automated clinical chemistry analyzer with enzymatic colorimetric methods. 37 participants completed all procedures, divided into two groups: the control group (n=20), which received nutritional counseling, and the intervention group (n=17), which received 1200 mg omega-3 supplements daily for four weeks.

Results: The average age was 18.59 ± 0.62 years in the intervention group and 18.30 ± 0.57 years in the control group. In the intervention group, ApoB-100 levels significantly decreased from 1768.23 ± 335.18 to 1410.20 ± 191.03 ug/ml ($p < 0.001$). The control group showed an increase from 1582.29 ± 426.95 to 1760.29 ± 507.65 ug/ml ($p = 0.156$).

Conclusion: This study showed Omega-3 supplementation significantly reduced ApoB-100 levels in obese adolescents, suggesting its potential role in reducing cardiovascular risk.

KEYWORDS

Cardiovascular risk; lipid metabolism; nutritional intervention; polyunsaturated fatty acids; adolescent obesity.

INTRODUCTION

Obesity has become a growing public health concern, affecting over 600 million individuals globally, including more than 100 million children. Indonesia has also experienced a rising trend in adult obesity. According to *Riset Kesehatan Dasar* (Riskesdas) in 2018, the prevalence of obesity among individuals aged 18 years and older increased from 10.5% in 2007 to 21.8% in 2018. The data further revealed a prevalence of 17.9% in rural areas and 24.2% in urban regions[1–3]. Obesity can occur due to lack of physical activity and high consumption of ultra-processed foods[4,5].

Obesity directly affects several organs responsible for metabolism, i.e. the pancreas, liver and adipocytes[6]. The severity of dyslipidemia often escalates with the degree of obesity, making it common for lipid metabolism disorders and dyslipidemia to coexist with excess body weight. This condition frequently results from a chronic imbalance between energy intake and expenditure. Disruptions in lipid absorption may contribute to dyslipidemia, characterized by elevated levels of low-density lipoprotein (LDL) and triglycerides (TGs),

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along with reduced levels of high-density lipoprotein (HDL)[7,8]. High triglyceride levels are a significant risk factor for cardiovascular disease[9]. Apolipoprotein B (ApoB), a key structural protein of atherogenic lipoproteins, serves as a reliable marker for the number of these particles in circulation[10]. There is a strong correlation between the likelihood of atherosclerosis and vascular disease and the accumulation of these particles[7,8].

Fish oil (FO), which contains the long-chain omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), has shown efficacy in lowering fasting and non-fasting plasma triglycerides as well as total ApoB-lipoprotein levels in individuals with metabolic syndrome. FO also helps reduce postprandial triglyceride spikes after high-fat meals. Meta-analyses have reported reductions of 15% to 45% in fasting TG and ApoB levels following fish oil supplementation[11]. Therefore, this study aims to analyze the effect of omega-3 supplementation on ApoB-100 levels in adolescents with obesity as part of cardiometabolic risk assessment.

METHODS

Study Design

This study is an experimental study, parallel clinical trial with pre-post test design. This study was conducted at Hasanuddin University, Makassar, with the collection of research subjects in February to March 2025, then an intervention was carried out for 4 weeks from March 2025 to April 2025.

Participant

A total of 770 adolescent individuals were initially screened for eligibility to participate in this study. Following the screening process, 90 individuals met the inclusion criteria, which required participants to be new undergraduate students in a health-related program at Hasanuddin University for the 2024/2025 academic year, have a BMI ≥25 kg/m², and provide written informed consent to participate. Exclusion criteria included the presence of chronic illnesses, use of medications that affect lipid metabolism, current pregnancy or breastfeeding, recent participation in weight loss or insulin resistance programs (within the past 3 months), consumption of fish oil supplements in the past month, allergies to fish or fish oil, and diagnoses of diabetes mellitus or chronic heart or kidney disease. However, 50 participants were drop out.

As a result, 40 participants remained and were randomly assigned to one of two groups: 20 to the intervention group and 20 to the control (education) group. Participants were randomly assigned to intervention and control groups using the sealed opaque envelope method. Each envelope contained a pre-generated random allocation number, and was identical in appearance to ensure allocation concealment. Envelopes were shuffled and opened sequentially by the research team only after participant enrollment, maintaining blinding and minimizing selection bias. During the course of the study, three participants in the intervention group declined to proceed with the research procedures, reducing the final number of participants in the intervention group to 17. Despite this attrition, data collection and analysis were completed for all remaining participants in both groups.

Research Ethics

The Health Research Ethics Commission of the Faculty of Medicine, Hasanuddin University, Makassar, as well as the Education and Research Department of Dr. Wahidin Sudirohusodo General Hospital and Makassar Satellite/Network Hospital, approved this study with number 226/UN4.6.4.5.31/PP36/2025. All research participants signed an *informed consent* to participate with anonymity, and the confidentiality of the data was guaranteed.

Data collecting

At baseline, all participants completed the International Physical Activity Questionnaire (IPAQ) to evaluate their initial physical activity levels. However, throughout the duration of the study, no structured interventions were introduced to modify physical activity or regulate caloric intake. As a result, any effects of physical activity or dietary energy consumption on the study outcomes were not actively controlled or influenced.

Measurements were taken at both the beginning of the study and after the four-week intervention period. These included assessments of body weight, height, blood pressure, body mass index (BMI), waist and hip circumference, and waist-to-hip ratio, along with repeated physical activity assessments. In addition, body composition specifically visceral fat, muscle mass, bone mass, and fat mass was evaluated at the same two time points. Blood samples were collected to determine ApoB-100 levels. Lipid profile parameters, including triglycerides, HDL, LDL, and total cholesterol, were also measured at both time points.

Anthropometric Measurements

Height measurement was performed using GEA microtoise, and weight measurement was performed using a body weight scale. Body composition measurement used BIA (Bioelectric impedance analysis) (Tanita Body Fat Analyzer; Tanita Corporation of America Inc, Arlington Heights, IL, USA, model 730). All participants should to stand upright without shoes, and wearing clothes without carrying items (phone, wallets, keys, bags, or hats) facing forward, the back of the shoulders, buttocks and heels facing the wall with hand position at the side. Height and weight values were used to obtain BMI results. Waist and hip circumferences were measured using a measuring tape

Blood Pressure

Blood pressure data collection was obtained by measuring blood pressure using a digital sphygmomanometer (Omron, Schaum-berg, IL, USA).

Measurement of ApoB-100

ApoB-100 levels were measured using Human Apolipoprotein B1 ELISA Kit Cat. No. E3768Hu from BT LAB with the Enzyme-Linked Immunosorbent Assay (ELISA) method. The measurement results were interpreted in microgram per milliliter (µg/ml). All participants had to fast for 12 hours before blood sampling. Blood sampling of participants is carried out by laboratory staff certified in phlebotomy. Then ApoB-100 analysis is carried out in the laboratory.

Biochemical Measurements

Fasting blood samples were obtained after 12 hour overnight fast, collected by certified medical personnel. Lipid profile parameters, including total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), were measured using an automated clinical chemistry analyzer (e.g., Cobas 6000, Roche Diagnostics, Basel, Switzerland) employing enzymatic colorimetric methods, following the manufacturer's protocol. All assays were conducted in a certified clinical laboratory in compliance with established internal quality control procedures**.**

Omega-3 Supplementation

After the participants were enrolled, they were randomly assigned to one of two groups: the education group and the intervention group. The intervention group received an omega-3 supplement for a duration of four weeks, with a daily dosage of 1200 mg, which also included 400 mg of Fish Oil (18/12 EPA/DHA) and 15 mg of 100% d-alpha-tocopherol. In contrast, the control group received general nutritional guidance at the beginning of the study. The intervention group will receive reminder messages to consume an omega-3 and receive the monitoring paper that needs to be checked every day and collected every week.

Data analysis

Statistical Package for the Social Sciences (SPSS) version 29.0 was used for data analysis. Categorical variables were interpreted with frequency and percentage. Numerical variables were interpreted with mean and standard deviation. To analyze the effect of omega-3 administration on ApoB-100, bivariate analysis was used. First, Shapiro-Wilk normality tests were performed on numerical variables. To evaluate the effect of omega-3 supplementation on ApoB-100 and other metabolic parameters, both intra-group (within-group) and inter-group (between-group) comparisons were conducted. For intra-group comparisons (pre- and post-intervention within the same group), the paired t-test was used for normally distributed data, and the Wilcoxon signed-rank test for non-normally distributed data. For inter-group comparisons (between intervention and control groups), the independent t-test was used for normally distributed data, and the Mann-Whitney U test for non-normally distributed data. In addition, a repeated measures analysis was performed to assess the interaction between time (pre- and post-intervention) and group (intervention vs. control), allowing for evaluation of intra-group and inter-group changes over time. The 95% Confidence Interval (CI95%) was used in this study, and a p-value of less than 0.05 was considered significant.

RESULTS

The study included 17 participants in the intervention group and 20 participants in the control group. The process of selecting study subjects is illustrated in Figure 1, while Table 1 presents the demographic and baseline characteristics of the respondents.

There were no significant differences between the intervention and control groups in terms of age, BMI, waist circumference, hip circumference, waist-to-hip ratio, fat mass, muscle mass, bone mass, physical activity level, blood pressure, and obesity classification. However, significant differences were observed between the two groups in body weight (p=0.018), height (p=0.040), visceral fat (p=0.033), cellular age (p=0.043), and parental history of metabolic syndrome (p=0.048), as presented in Table 1. At baseline there were no significant differences ApoB-100,HDL-C,LDL-C,LDL-C and TG level between the intervention and control groups. However, there was a significant differences in total cholesterol (p=0.028) in table 2.

Table 3 showed different results between the groups, before receiving omega-3 supplementation, the intervention group had a mean ApoB-100 level of 1768.23 ± 335.18, which significantly decreased to 1410.20 ± 191.03 after the intervention (p = 0.000). In contrast, the control group showed an increase in mean ApoB-100 levels from 1582.29 ± 426.95 at the first measurement to 1760.29 ± 507.65 at the second, with no statistically significant difference observed (p = 0.156). These results indicate a significant association between omega-3 supplementation and changes in ApoB-100 levels (p = 0.000), as levels declined in the intervention group but increased in the control group. Regarding lipid profiles, the intervention group exhibited significantly lower levels of total cholesterol, LDL cholesterol, and triglycerides compared to the education group (p=0.039, p=0.001, and p=0.045, respectively). However, the increase in HDL cholesterol levels in the intervention group compared to the control group narrowly missed statistical significance (p=0.050).

Table 4 showed significant differences in body composition and metabolic parameters between the intervention and con-

Table 1. Participants Characteristics

Variacble		Group		Total	p-value
		Intervention (n=17)	Control (n=20)		
		Mean ± SD	Mean ± SD		
Age (17-20 years)		18.59±0.62	18.30±0.57	18.3±0.60	0.189[a]
Weight (kg)		69.06±9.56	78.5±13.59	74.16±12.68	0.018[b*]
Height (cm)		154.22±3.98	157.69± 5.61	156.09±5.17	0.04 [a*]
Body Mass indes (kg/m^2)		28.94±3.84	30.70±3.93	29.89±3.94	0.059 [a]
Waist Circumference (cm)		85.79±7.57	90.83±12.19	88.62±9.88	0.251[b]
Hip Circumference (cm)		103.06±7.05	106.8±7.87	104.93±7.46	0.183[b]
Waist-Hip Ratio		0.93±0.19	0.90±0.17	0.915±0.18	0.988[b]
Body composition (BIA)					
Fat Mass %		38.88±3.90	41.32±4.92	40.20±4.62	0.080[b]
Muscle Mass %		39.55±3.15	41.76±3.88	40.74±3.69	0.069[a]
Visceral Fat %		6.79±2.15	8.07±1.88	7.48±2.08	0.033[b*]
Bone Mass %		2.53±0.30	2.74±0.37	2.64 ± 0.35	0.076[a]
Biological Age (years)		29.35±3.52	31.50±2.61	30.51 ± 3.20	0.043[b*]
History of metabolic syndrome in parents	Yes	5	5	10	0.048[b*]
		29.4%	25.0%	27.0%	
	No	8	3	11	
		47.1%	15.0%	29.7%	
	Don't Know	4	12	16	
		23.5%	60.0%	43.2%	
Physical activity (IPAQ)	Light	0	4	4	0.062[b]
		0.0%	20.0%	10.8%	
	Moderate	9	12	21	
		52.9%	60.0%	56.8%	
	Heavy	8	4	12	
		47.1%	20.0%	32.4%	

[a] Independent T-Test; [b] Mann-Whitney test; * p ≤0,05 considered Significant.

Table 1 continuation. Participants Characteristics

Variacble		Group		Total	p-value
		Intervention (n=17)	Control (n=20)		
		Mean ± SD	Mean ± SD		
Blood pressure (mmHg)	Normal	16	18	34	1.00[b]
		94.1%	90.0%	91.9%	
	High	1	2	3	
		5.9%	10.0%	8.1%	
Obesity Category	Obesity 1	12	12	24	0.744[b]
		70.6%	60.0%	64.9%	
	Obesity 2	5	8	13	
		29.4%	40.0%	35.1%	
Total		17	20	37	
		100.0%	100.0%	100.0%	

[a] Independent T-Test; [b] Mann-Whitney test; * p ≤0,05 considered Significant.

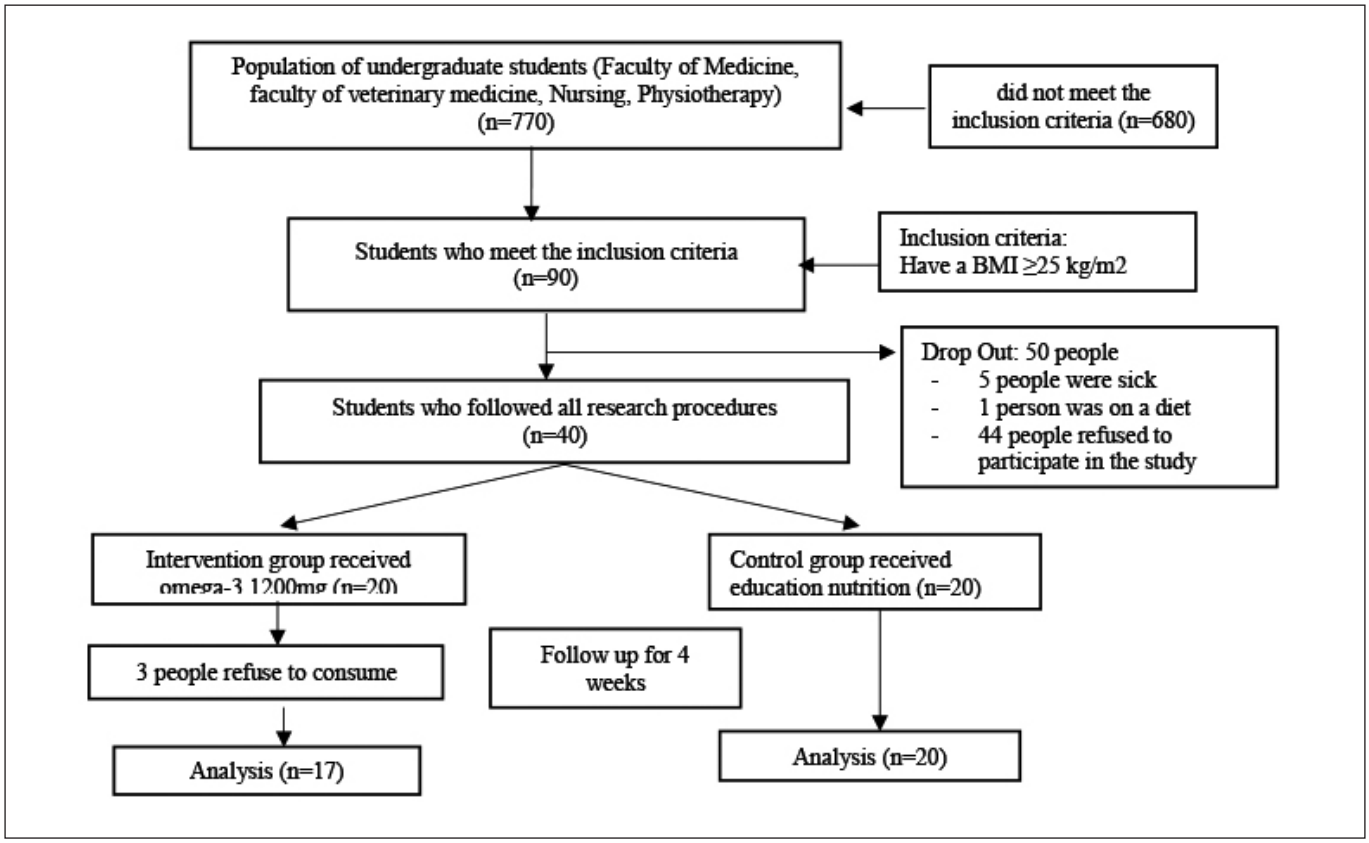

Figure 1. Selection of Research Subjects

Table 2. Apolipoprotein B100 and lipid profile level at baseline

Variable	Intervention (n=17)	Control (n=20)	p-value
	Mean ± SD	Mean ± SD	
ApoB-100	1768.23 ± 335.18	1582.29 ± 426.95	0.155[a]
Total Cholesterol	171.12±29.62	193.55±29.66	0.028[a*]
HDL-C	44.47±7.70	48.45±18.75	0.963[b]
LDL-C	109.00±30.42	126.80±30.87	0.087[a]
TG	96.06±27.32	119.35±59.49	0.352[b]

[a] Independent T-Test; [b] Mann-Whitney test; * p-value ≤ 0.05 was considered significant; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TG: triglycerides.

Table 3 . Effect of Omega-3 Administration on Apolipoprotein B100 and Lipid Profile

	Group	Pre-Test	Post-Test	p-value within group	p-value between group [c]
		Mean ± SD	Mean ± SD		
ApoB-100	Intervention	1768.23 ± 335.18	1410.20 ± 191.03	0.000[a*]	0.000*
	Control	1582.29 ± 426.95	1760.29 ± 507.65	0.156[b]	
Total Cholesterol	Intervention	171.12 ± 29.62	169.65 ± 24.45	0.585[a]	0.039*
	Control	193.55 ± 29.66	206.05 ± 32.83	0.021[a]	
HDL-C	Intervention	44.47 ± 7.70	50.24 ± 7.99	0.000[a]	0.050
	Control	48.45 ± 18.75	48.10 ± 9.17	0.025[b]	
LDL-C	Intervention	109.00 ± 30.42	102.35 ± 25.14	0.004[a]	0.001*
	Control	126.80 ± 30.87	141.70 ± 33.08	0.022[a]	
TG	Intervention	96.06 ± 27.32	89.82 ± 26.50	0.275[a]	0.045*
	Control	119.35 ± 59.49	126.85 ± 46.66	0.084[b]	

[a] Paired T-Test; [b] Wilcoxon Signed Rank Test; [c] Mann-Whitney U with correction; * p-value ≤ 0.05 was considered significant; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TG: triglycerides.

trol groups. The intervention group displayed significant reduction in visceral fat and fat mass compared to the education group (p<0.001 for both parameters). However, no significant differences were observed between the groups regarding muscle mass (p=0.152) or bone mass (p=0.057), indicating that these parameters remained relatively stable across both interventions. Cell age measurements revealed a significantly lower value in the intervention group compared to the control group (p=0.004), suggesting improved cellular health outcomes. Anthropometric measurements indicated that the intervention group achieved significantly smaller waist and hip circumferences than the control group (p<0.001 for both measurements), although the waist-hip ratio showed no significant difference between groups (p=0.620).

DISCUSSION

The study indicates a significant link between omega-3 supplementation and ApoB-100 levels in adolescent with obesity. Specifically, ApoB-100 levels continue to rise in the group receiving only educational intervention, whereas a re-

Table 4. Effect of Omega-3 Administration on anthropometric measurements and body composition

	Group	Pre-Test	Post-Test	p-value within group	p-value between group
		Mean ± SD	Mean ± SD		
Weight	Intervention	69.06±9.56	68.22±9.70	0.000[d*]	0.000[b*]
	Control	78.50±13.59	77.22±13.19	0.093[d]	
BMI	Intervention	28.94±3.85	28.55±3.97	0.001[d*]	0.000[a*]
	Control	30.71±3.94	30.88±4.00	0.074[d]	
Fat Mass	Intervention	38.88±3.90	38.45±3.88	0.002[d*]	0.000[b*]
	Control	4132±4.98	41.37±5.04	0.206[c]	
Muscle Mass	Intervention	39.55±3.15	39.64±3.04	0.416[c]	0.152[b]
	Control	41.76±3.88	41.78±3.85	0.330[c]	
Visceral fat	Intervention	6.79±2.15	6.63±2.07	0.008[c*]	0.001[b*]
	Control	8.07±1.88	8.14±1.97	0.285[d]	
Bone Mass	Intervention	2.53±0.30	2.55±0.28	0.104[c]	0.057[b]
	Control	2.74±0.37	2.74±0.37	1.000[d]	
Biological Age	Intervention	29.35±3.52	28.94±3.51	0.014[c*]	0.004[b*]
	Control	31.50±2.61	31.50±2.61	1.000[d]	
Waist	Intervention	85.79±7.57	83.61±12.80	0.026[d*]	0.000[b*]
	Control	90.83±12.19	91.04±12.30	0.000[c*]	
Hip	Intervention	103.06±7.05	102.84±7.03	0.006[c]	0.000[b*]
	Control	106.80±7.87	106.92±7.96	0.027[d]	
Waist-Hip Ratio	Intervention	0.93±0.19	0.81±0.12	0.723[d]	0.620[a]
	Control	0.90±0.17	0.85±0.08	0.550[d]	

[a] Independent T-Test; [b] Mann-Whitney; [c] Paired T-Test; [d] Wilcoxon Signed Rank Test; * p-value ≤ 0.05 was considered significant; BMI: Body mass index.

duction is observed following omega-3 supplementation. These results are in line with those of Ferchaud et al., who reported that omega-3 intake may decrease ApoB-100 levels ($p = 0.028$)[12]. Similarly, Jun et al. found that omega-3 treatment lowered ApoB-100 levels in individuals with type 2 diabetes, along with changes in LDL and adiponectin levels ($p = 0.028$)[11]. However, it is important to note that several studies have questioned the immediate impact of omega-3 supplementation on blood cholesterol levels, particularly total cholesterol and LDL-C. Meta-analyses have shown that significant lipid-lowering effects often require higher doses (≥2 g/day) or longer durations of supplementation. For example, Wang et al.[13] found that higher doses of omega-3 was associated with a near-linear reduction in triglycerides, similarly Lu et al.[14] reported omega-3 (fish oil) supplementation significantly reduced triglycerides after 12 weeks administration. Another meta-analysis by Yang et al.[15] found that omega-3 monotherapy significantly reduced triglycerides and non-HDL cholesterol but increased LDL-C levels, suggesting a complex lipid response. These findings suggest that the effects observed in this study may be dose-dependent and should be interpreted with caution.

ApoB-100, a protein composed of 4,536 amino acids, plays a key role as the primary ligand for LDL receptor-mediated clearance of LDL particles from the bloodstream and is essential for the liver's production of VLDL[16]. Substantial research has shown that elevated levels of ApoB-100 are a significant risk factor for coronary heart disease (CHD), with higher concentrations correlating with an increased likelihood of developing the condition[17]. Therefore, reducing ApoB-100 levels is a critical strategy in lowering CHD risk[18]. In this study, participants in the intervention group received a daily dose of 1,200 mg of omega-3 for four weeks. Omega-3 polyunsaturated fatty acids (ω3 PUFAs) including docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA), and alpha-linolenic acid (ALA) have been recognized as an effective means of lowering cholesterol[13,19].

Omega-3 fatty acids may lower ApoB-100 levels by reducing triacylglycerol synthesis and enhancing mitochondrial fatty acid oxidation[20]. In hepatocytes, ApoB-100 production is largely influenced by the availability of liver fatty acids and triacylglycerols[21]. Omega-3s inhibit triacylglycerol formation primarily by decreasing the activity of acyl-CoA:1,2-diacylglycerol acyltransferase and promoting the oxidation of triacylglycerols. Their triglyceride-lowering effect is associated with reduced production of VLDL, especially the VLDL1 subclass[16].

The intake of omega-3 fatty acids stimulates an increase in α receptors via activation by peroxisome proliferators, which enhances hepatic uptake and oxidation of free fatty acids, as well as boosts fatty acid oxidation in skeletal muscle. This process leads to a reduced supply of free fatty acids, consequently decreasing ApoB-100 synthesis[22]. Furthermore, high doses of omega-3 over prolonged periods may induce endoplasmic reticulum stress, which can limit ApoB-100 production by suppressing its translation or promoting its degradation through both proteasomal and non-proteasomal pathways[21].

Liver levels of sphingosine, sphinganine, and ceramide play a key role in ApoB-100 production. When the levels of these lipids are reduced an effect that can be achieved through omega-3 supplementation ApoB-100 synthesis also declines[12]. Docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), components of omega-3, exert atheroprotective effects through several mechanisms: enhancing the intracellular breakdown of ApoB-100-containing lipoproteins, inhibiting hepatic ApoB-100 production, promoting the clearance of plasma triglycerides via lipoprotein lipase, accelerating the conversion of VLDL to LDL, reducing LDL synthesis, and lowering postprandial lipemia[23].

In addition, multiple studies have demonstrated that omega-3 fatty acids regulate gene expression involved in limiting fatty acid synthesis. Since the formation of triacylglycerols is essential for VLDL secretion, inhibiting fatty acid and triacylglycerol synthesis may lead to increased degradation of ApoB-100[24]. These findings are consistent with this study, which observed significant changes in triglyceride, LDL, and total cholesterol levels after omega-3 supplementation. However, HDL levels remained relatively unchanged, possibly because HDL particles do not carry ApoB-100[16]. Fish oil supplements also aid in LDL clearance, which may be due to reduced expression of LDL receptors or decreased binding affinity of LDL for its receptor. Additionally, omega-3 fatty acids can alter LDL's physicochemical properties, making it more prone to oxidation[24].

Rukman et al. reported a possible link between apolipoprotein B levels and visceral obesity[25]. Data from a larger cross-sectional study showed that a reduction in visceral fat was associated with normalization of apolipoprotein B levels. In the current study, omega-3 supplementation led to a significant reduction in both visceral fat and overall fat mass. A decrease in apolipoprotein B100 which plays a role in fat accumulation can contribute to the reduction of visceral fat and total fat mass[26]. As fat accumulation decreases, there is also a corresponding decline in body weight and body measurements, including waist and hip circumference[27].

However, this study has several limitations, one notable limitation of this study is the high attrition rate. Although 770 adolescents were initially screened, only 90 met the inclusion criteria, and ultimately just 37 participants completed the study procedures. This substantial reduction in sample size may limit the generalizability of the findings and reduce statistical power. The dropout was primarily due to refusal to participate, illness, and other personal reasons. Future studies should consider strategies to improve participant retention, such as extended follow-up, incentives, or more flexible scheduling. Another limitation of this study is the composition of the supplement used in the intervention group, which contained not only omega-3 fatty acids but also vitamin E (d-alpha-tocopherol). While the study aimed to evaluate the effects of omega-3 on ApoB-100 and lipid profiles, the presence of vitamin E a known antioxidant may have contributed to the observed outcomes. Therefore, the effects cannot be attributed exclusively to omega-3 supplementation. Future studies should consider using pure omega-3 formulations or include a separate group receiving vitamin E alone to better isolate the specific effects of each component. Other limitations of this study include its short duration and the lack of assessment of participants' caloric intake, both of which could influence ApoB-100 levels and potentially affect the study outcomes. Further research with longer intervention periods and controlled dietary intake is necessary to confirm these findings.

CONCLUSION

This study demonstrates that omega-3 supplementation significantly reduces ApoB-100 levels, total cholesterol, LDL cholesterol, triglycerides, visceral fat, and overall fat mass in obese adolescents, suggesting its potential role in lowering car-

diovascular risk. However, the findings should be interpreted with caution due to several limitations, including the small final sample size, short intervention duration, and the inclusion of vitamin E in the supplement, which may have contributed to the observed effects. Additionally, the lack of dietary intake assessment and the absence of repeated measures analysis may affect the robustness of the conclusions. Future studies with larger sample sizes, longer intervention periods, and more controlled designs are needed to confirm these results and isolate the specific effects of omega-3 fatty acids.

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