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Correlation of Dietary Intake and Body Composition with Circulating Betatrophin Levels in Overweight Young Women

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ABSTRACT

Overweight in women of reproductive age increases the risk of insulin resistance. Betatrophin, a hormone related to lipid and glucose metabolism, is considered a potential metabolic associated with insulin resistance risk. Despite increasing evidence linking betatrophin with insulin resistance and obesity, data regarding its association with dietary intake, body composition, and lipid profile among young overweight women in Indonesia remain limited. This study aimed to examine the correlation between dietary intake, physical activity, body composition, lipid profile, and menstrual cycle with betatrophin levels in overweight women aged 20–30 years. A cross-sectional study was conducted on overweight participants within this age group. Dietary intake was collected using repeated 24-hour recalls, while physical activity and menstrual patterns were obtained through questionnaires. Body composition, including fat and muscle mass, was measured using bioelectrical impedance analysis (BIA). Serum lipid profile and betatrophin concentrations were analyzed by ELISA. Spearman's correlation test was applied to determine the relationships among variables. Betatrophin levels showed positive correlations with energy intake (r=0.566; p=0.0001) and fat intake (r=0.366; p=0.028). Fat intake and body fat percentage were positively correlated with total cholesterol and LDL, while muscle mass showed negative correlations with both. Although women with irregular menstrual cycles tended to have higher betatrophin levels, the difference was not significant. These findings suggest that dietary fat, energy intake, and body composition correlated to metabolic markers related to insulin resistance risk in overweight young women.

INTRODUCTION

Overweight and obesity refer to an excessive accumulation of body fat, a condition that is increasingly prevalent among adults and adolescents[1]. According to the 2023 Indonesian Health Survey (SKI), 46,5% of adult women and 29,3% of adult men were classified as overweight or obese[2]. Obesity in women has been linked to insulin resistance and hyperlipidaemia[3]. Among women of reproductive age, insulin resistance is further associated with reproductive problems such as irregular menstrual cycles, polycystic ovary syndrome (PCOS), and infertility[4,5]. Therefore, obesity management in women should focus on addressing insulin resistance risk factors, including dietary habits, physical activity, and body composition. Biomarker like Betatrophin could play an important role as therapy target since its synthesized process is dysregulated by insulin resistance[6].

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Betatrophin, also known as angiopoietin-like protein 8 (ANGPTL8), is a hormone involved in glucose and lipid metabolism. It is secreted primarily in the liver and adipose tissue, both key organs in insulin regulation. Experimental studies have shown that betatrophin may contribute to insulin resistance by stimulating β-cell proliferation in the pancreas[7]. Research by Wang et al. reported that excess adipose tissue in obese women could exacerbate insulin resistance by altering betatrophin production[8,9]. However, findings from previous studies remain inconsistent, with some reporting decreased[10] and others increased betatrophin levels in obesity[11].

Given these inconsistencies, further investigation is needed to clarify betatrophin concentrations in overweight women. Since abnormal betatrophin levels may be influenced by determinants of obesity, such as excessive dietary intake, eating behaviours[12], lipid profile[13], and physical inactivity, these factors should be examined together[14,15]. Previous studies suggest that calorie restriction can lower betatrophin levels, while excessive intake may increase them[7]. Other interventions have shown that moderate-intensity physical activity can improve betatrophin levels[16]. Despite its potential role as a biomarker of insulin resistance, evidence regarding determinants of betatrophin, particularly dietary intake, body composition, and lipid profile, remains limited and inconsistent.

This gap in evidence complicates the development of effective interventions for preventing insulin resistance, especially among young overweight women. Previous studies by Hu et al. (2019) and Guo et al. (2022) explored the relationship between betatrophin, diet, and physical activity[6,7], but their results were inconsistent and have not been replicated in Indonesian populations. More recently, Susanto et al. (2023) observed dynamic changes in betatrophin after moderate-intensity exercise, yet did not examine associations with lipid profiles or menstrual cycles[17].

This study aimed to find a correlation between lifestyle factors including dietary intake, physical activity, body composition, and lipid profile and betatrophin levels among overweight young women. We hypothesized that higher energy intake, fat intake, adiposity, and unfavorable lipid profiles would be associated with elevated circulating betatrophin concentrations among overweight young women.

MATERIALS AND METHODS

The study is cross-sectional involved female university students who met the eligibility criteria. Participants were screened through body weight and height measurements to determine their Body Mass Index (BMI). Those classified as overweight or obese based on the WHO Asia-Pacific criteria (BMI ≥23 kg/m²) were recruited using purposive sampling. Participants were recruited using purposive sampling who met the inclusion criteria. The inclusion criteria were as follows: (i) aged 19–23 years,

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(ii) not diagnosed with cardiovascular disease, diabetes, or hypertension, (iii) not currently following a weight-loss diet, and (iv) not pregnant or breastfeeding. Participants with diagnosed metabolic diseases were excluded.

The minimum sample size was calculated using the formula for correlation analysis with an expected moderate effect size (r = 0.40), alpha = 0.05, and power = 80%, resulting in a minimum of 30 participants. A total of 36 participants were included in the final analysis.

Dietary intake data were collected through face-to-face interviews using two non-consecutive 24-hour dietary recalls, consisting of one recall on a weekday and one on a weekend day. Physical activity levels were assessed using the International Physical Activity Questionnaire (IPAQ). Body composition was measured using a Bioelectrical Impedance Analysis (BIA) device (OMRON HBF-375) to estimate body fat percentage, visceral fat level, and skeletal muscle percentage.

All participants were instructed to fast overnight prior to venous blood sampling for biochemical analysis. Serum lipid profiles, including total cholesterol, triglycerides, LDL-cholesterol, and HDL-cholesterol, were analyzed using standard enzymatic methods. Betatrophin levels were measured using the Human Betatrophin (Total) using ELISA Kit (Aviscera Bioscience, Inc., CA, USA) based on the quantitative sandwich ELISA method. All laboratory analyses were conducted at the partner laboratory of Poltekkes Tanjung Karang.

Statistical analysis was performed using IBM SPSS Statistics version 25. Descriptive data were presented as mean ± standard deviation or median (interquartile range), depending on data distribution. Normality was assessed using the Shapiro–Wilk test. Pearson or Spearman correlation tests were used to analyse associations among variables. A p-value of <0.05 was considered statistically significant.

This study was approved by the Ethics Committee of Universitas Aisyah Pringsewu (No. 698/22/08/2025). Written informed consent was obtained from all participants prior to data collection.

RESULTS

Based on anthropometric measurements, the 36 respondents had an average Body Mass Index (BMI) of 30.2 ± 4.2 kg/m², which falls within the obesity category according to the Asia-Pacific classification (≥25 kg/m²). The median visceral fat was 9% (4.5–20.5) and total body fat was 35.5% (27.1–42), indicating a high proportion of body fat, particularly visceral fat, which is closely linked to metabolic risk. Meanwhile, the average muscle mass was 23.3 ± 1.2%, remaining within the normal range.

Energy and macronutrient intake, adiposity, and lipid profile in this study are descriptively presented in Table 1.

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Table 1. Descriptive Statistics of Energy and Macronutrient Intake, Antropometric, Lipid Profile and Betatrophin Level

Variables	Mean±SD	Median	Min-max
	Recall 2x24h		
Energy intake (kcal)	2139,1 ± 297,8*	2078,8	1498,5-2805,3
Protein intake (g)	83,6 ± 28,2*	73,4	36-151
Fat intake (g)	104,3 ± 35,3*	106,7	40,2-183,6
Carbohydrate intake (g)	220,8 ± 84,2*	201,1	80-378
	Anthropometric		
Body Mass Index (BMI) (kg/m2)	30,2 ± 4,2	29,3	24,3-39
Visceral fat (%)	10,4 ± 4,3	9	4,5-20,5
Total fat (%)	34,5 ±4,0*	35,5	27,1-42
Total muscle (%)	23,3 ± 1,2*	23,6	20,2-26,7
	Lipid Profile and Betatrophin Level		
HDL (mg/dl)	44,3 ± 9,1	43,5	32-63
Triglyceride (mg/dl)	168,5 ± 41,5*	174	70-250
Cholesterol (mg/dl)	198,1 ± 24,9	190	160-260
LDL (mg/dl)	129,1 ±20,8	128,5	100-198
Betatrophin (np/mL)	11,3 ± 5,5	10,3	4,0-33

*Normally distributed

The lipid profile showed an average HDL level of 44.3 ± 9.1 mg/dl, lower than the optimal threshold for women (≥50 mg/dl). The median triglyceride level was 174 mg/dl (70–250), exceeding the normal value of <150 mg/dl. The mean total cholesterol level was 198.1 ± 24.9 mg/dl, approaching the high-risk threshold (≥200 mg/dl), while LDL levels averaged 129.1 ± 20.8 mg/dl, nearly reaching the upper limit (≥130 mg/dl). Overall, these findings suggest a lipid profile pattern consistent with dyslipidaemia, a condition commonly associated with obesity. Furthermore, the median betatrophin level was 10,3 ng/mL (4,0-33).

Table 2. Energy and Fat Intake, Adiposity, and Their Associations with Betatrophin Levels and Lipid Profile

Variable	Betatrophin (r;p-value)	Cholesterol (r;p-value)	LDL (r;p-value)	Triglyceride (r;p-value)	HDL (r;p-value)
Energy Intake	0,566; 0,0001**	0,282; 0,096	0,315; 0,062	-0,258; 0,113	-0,019;0,935
Fat Intake	0,366; 0,028*	0,466; 0,005**	0,298; 0,082	-0,242; 0,146	-0,126; 0,466
Visceral fat	0,24; 0,158	0,333; 0,047*	0,231; 0,186	-0,001; 0,992	0,085; 0,622
Total fat	0,188; 0,273	0,465; 0,004**	0,350; 0,034*	0,124; 0,552	-0,147; 0,494
Total muscle	-0,172; 0,317	-0,434; 0,008**	-0,360; 0,031*	-0,298; 0,082	-0,126; 0,466
BMI	0,198; 0,246	0,353; 0,034*	0,149; 0,435	-0,149; 0,435	-0,126; 0,466

*p < 0,05; **p<0,01

The bivariate analysis revealed a significant positive correlation between betatrophin levels and both energy intake (r = 0.566; p = 0.0001) and fat intake (r = 0.366; p = 0.028). However, other variables such as carbohydrate intake, visceral fat, total body fat, muscle mass, body mass index, lipid profile (LDL, Cholesterol, Triglyceride and HDL) showed no significant associations with betatrophin. Although women with irregular menstrual cycles tended to have higher betatrophin levels

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compared to those with regular cycles (13.9 vs 18.57 vs 20.29 ng/L; p = 0.492), the difference was not statistically significant.

DISCUSSION

Participants in this study were characterized by excess adiposity, reflected by obesity-level BMI and elevated body fat percentage. Excess adipose tissue, particularly visceral fat, is recognized as a metabolically active organ that promotes chronic low-grade inflammation and insulin resistance. These metabolic alterations may influence hepatokines and adipokines involved in lipid and glucose regulation, including betatrophin. Similar findings have been reported in previous studies indicating that visceral adiposity is more strongly associated with metabolic dysfunction than BMI alone [6].

The lipid profile pattern observed in this study is consistent with metabolic alterations commonly reported among individuals with obesity. Excess adiposity contributes to increased free fatty acid flux to the liver, promoting hepatic lipid synthesis and unfavorable lipid metabolism. Previous evidence suggests that these metabolic disturbances may coexist with alterations in circulating betatrophin concentrations, although findings remain inconsistent across populations and metabolic conditions [6].

The median betatrophin level among respondents was 10.3 ng/mL (range 4.0–33), this value is within the range reported in previous studies on obese populations[18,19]. Betatrophin levels tend to increase in parallel with elevated triglycerides and increased visceral fat accumulation. A study by Liu et al. (2020) demonstrated a significant association between circulating betatrophin and lipid parameters, particularly triglycerides, among obese women[20]. Therefore, the findings of this study are consistent with earlier evidence suggesting that betatrophin may serve as a potential metabolic biomarker in individuals with obesity.

Additionally, fat intake was positively correlated with total cholesterol (r = 0.466; p = 0.005), while total body fat was positively correlated with both total cholesterol (r = 0.465; p = 0.004) and LDL (r = 0.350; p = 0.034). In contrast, total muscle mass showed a significant negative correlation with total cholesterol (r = −0.434; p = 0.008) and LDL (r = −0.360; p = 0.031).The bivariate analysis revealed that betatrophin levels were significantly and positively correlated with energy intake (r = 0.566; p = 0.0001) and fat intake (r = 0.366; p = 0.028). This finding suggests that higher energy and fat consumption are associated with elevated betatrophin concentrations. Physiologically, betatrophin plays a regulatory role in lipid metabolism and insulin sensitivity. Previous studies have similarly reported that individuals with obesity and high-energy dietary patterns tend to experience increased circulating betatrophin compared to those with normal nutritional status[20–22]. Moreover, the correlation may be explained by increased hepatic lipid flux and stimulation of ANGPTL8 expression in response

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to excess caloric intake. High fat consumption may increase triglyceride metabolism and contribute to insulin resistance pathways, thereby increasing circulating betatrophin levels[23,24].

In contrast, other variables such as carbohydrate intake, visceral fat, total body fat, muscle mass, and body mass index showed no significant correlation with betatrophin. Moreover, no association was observed between physical activity (p = 0.944) or menstrual cycle status and betatrophin levels. This finding may indicate that betatrophin reflects dynamic metabolic responses to recent nutritional intake rather than long-term body fat accumulation. Similar observations have been reported by Zheng et al., who suggested that circulating betatrophin is more closely associated with metabolic status and fat distribution than with overall obesity[25]. However, women with irregular menstrual cycles exhibited slightly higher median betatrophin levels (13.9 vs 18.57 vs 20.29 ng/mL; p = 0.492), which aligns with findings by Keikha et al. (2021) and Gong et al. (2023), indicating that elevated betatrophin might be linked to hormonal and metabolic disturbances among women with menstrual irregularities and polycystic ovary syndrome (PCOS)[26,27].

Significant relationships were also observed among other variables. Fat intake was positively correlated with total cholesterol (r = 0.466; p = 0.005), while total body fat showed a positive association with both total cholesterol (r = 0.465; p = 0.004) and LDL cholesterol (r = 0.350; p = 0.034). Conversely, total muscle mass was negatively correlated with total cholesterol (r = −0.434; p = 0.008) and LDL (r = −0.360; p = 0.031). Skeletal muscle plays a central role in glucose uptake and lipid utilization; therefore, preservation of muscle mass may contribute to improved cardiometabolic health among women with excess body weight [23,24].

This study has several limitations that should be considered. First, the cross-sectional design does not allow causal correlation between dietary intake and betatrophin levels. Second, dietary intake was assessed using a 2x24-hour recall, which may be subject to recall bias and may not fully represent habitual intake. Third, insulin resistance was not directly measured, such as HOMA-IR, limiting the ability to confirm the mechanistic role of betatrophin. Fourth, the sample size of this study is small (n=36), which may limit statistical power and the ability to detect modest associations. Future studies with larger sample sizes are required to strengthen the evidence. Participants were recruited using purposive sampling from a single university population, which may limit representativeness and introduce selection bias. Therefore, the findings should be interpreted cautiously and may not be generalizable to all Indonesian women of reproductive age. Despite these limitations, this study provides important preliminary evidence on the relationship between dietary intake and betatrophin levels among overweight young women in Indonesia.

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CONCLUSION

Overall, Energy and fat intake were positively associated with circulating betatrophin concentrations among overweight young women. Additionally, higher fat intake and body fat percentage were associated with less favorable lipid profiles, whereas muscle mass demonstrated protective associations. These findings suggest that dietary modification, particularly reducing excessive energy and fat intake, may contribute to improving metabolic health among overweight women. Future longitudinal studies involving larger populations and direct measures of insulin resistance are required to clarify the role of betatrophin as an early metabolic biomarker.

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