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Association between dietary inflammatory index (DII) and insulin resistance (HOMA-IR) in obese adults

Hubungan antara indeks inflamasi diet (dietary inflammatory index/DII) dan resistensi insulin (HOMA-IR) pada orang dewasa dengan obesitas

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Abstract

The prevalence of obesity is increasing in many regions, including Semarang, and is strongly associated with insulin resistance. The Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) is a widely used indicator of this condition. Dietary patterns contribute to systemic inflammation, and the Dietary Inflammatory Index (DII) is used to assess the inflammatory potential of individual diets. However, evidence linking the DII to insulin resistance in obese populations remains limited. This study aimed to analyze the association between DII scores and HOMA-IR in obese adults in Semarang. A cross-sectional study was conducted between September and November 2024, involving 66 obese adults aged 26–50 years (BMI ≥25 kg/m^2) recruited using purposive sampling. Dietary intake was assessed using a semi-quantitative food frequency questionnaire (SQ-FFQ) and subsequently converted to DII scores. Fasting glucose and insulin levels were measured to calculate HOMA-IR. Pearson's correlation and multiple linear regression analyses were performed, controlling for age and BMI. The DII scores were positively and significantly correlated with HOMA-IR (p=0.002). Multiple regression analysis confirmed that the DII remained an independent predictor of HOMA-IR (β=0.368; p=0.002), with the final model explaining 24.3% of the variance in HOMA-IR (R^2=0.243). In conclusion, higher DII scores were significantly associated with increased insulin resistance in obese adults, independent of age and BMI. These findings highlight the importance of adopting anti-inflammatory dietary patterns to prevent metabolic disorders.

Keywords: Dietary inflammatory index, HOMA-IR, insulin resistance, obesity

Abstrak

Obesitas semakin meningkat di berbagai wilayah, termasuk Kota Semarang, dan berisiko memicu resistensi insulin. Salah satu indikatornya adalah *Homeostatic Model Assessment for Insulin Resistance* (HOMA-IR). Pola makan berperan dalam proses inflamasi sistemik, dan *Dietary Inflammatory Index* (DII) digunakan untuk menilai sifat inflamasi dari konsumsi makanan. Namun, bukti hubungan DII dengan resistensi insulin pada populasi obesitas masih terbatas. Penelitian bertujuan untuk menganalisis keterkaitan antara skor DII dan HOMA-IR pada orang dewasa obesitas di Kota Semarang. Penelitian potong lintang dilakukan September–November 2024 pada 66 orang dewasa obesitas usia 26–50 tahun (IMT ≥25 kg/m^2) yang dipilih dengan purposive sampling. Asupan makanan dikumpulkan melalui kuesioner SQ-FFQ, kemudian dihitung skor DII. Kadar glukosa dan insulin puasa diperiksa untuk menghitung HOMA-IR. Analisis menggunakan uji korelasi Pearson dan regresi linear berganda dengan kontrol usia dan IMT. Hasil, skor DII berhubungan positif signifikan dengan HOMA-IR (p=0,002). Regresi linear berganda menunjukkan skor DII tetap berpengaruh signifikan terhadap HOMA-IR setelah dikontrol usia dan IMT (β=0,368; p=0,002), dengan model akhir menjelaskan 24,3% variasi HOMA-IR (R^2=0,243).

Kesimpulan, skor DII yang lebih tinggi berhubungan signifikan dengan peningkatan resistensi insulin pada orang dewasa obesitas, independen dari usia dan IMT. Temuan ini menekankan pentingnya pola makan anti-inflamasi dalam pencegahan gangguan metabolik.

Kata Kunci: Indeks inflamasi diet, HOMA-IR, obesitas, resistensi insulin

Introduction

Obesity is a complex and multifactorial condition characterized by the excessive accumulation of body fat, which poses significant health risks (Yuliadewi et al., 2021). It has become a global epidemic, with the World Health Organization (WHO) reporting that worldwide obesity rates have nearly tripled since 1975 (WHO 2022). The burden of obesity is not limited to high-income countries, but is also rising rapidly in low- and middle-income countries, including Indonesia(Ahmed et al., 2021)(Lemamsha et al., 2019). According to the Indonesian Health Survey (*Survei Kesehatan Indonesia* or SKI, 2023), the national prevalence of obesity among adults aged 18 years and above is 23,4%, with a notably higher prevalence in women (31,2%) than in men (15,7%).

The increasing prevalence of obesity is associated with numerous modifiable and non-modifiable risk factors, including unhealthy dietary patterns, reduced physical activity, genetic predisposition, socioeconomic conditions, and environmental influences (Purdy et al., 2022). Inappropriate dietary intake, especially excessive consumption of energy-dense and nutrient-poor foods, has consistently been identified as a key contributor to the obesity epidemic. Although genetic and hormonal factors play a role in weight regulation, recent evidence has emphasized the importance of chronic low-grade inflammation as a central mechanism linking obesity to metabolic dysfunction (Jurczewska et al., 2023; Al Rahmad et al., 2020).

Obesity-induced inflammation results from the abnormal expansion of adipose tissue and infiltration of immune cells, leading to the increased production of pro-inflammatory cytokines such as TNF-α and IL-6. These inflammatory mediators disrupt insulin signaling pathways, contributing to insulin resistance, a metabolic condition in which peripheral tissues fail to respond adequately to insulin stimulation (Faccioli et al., 2023; Mayoral et al., 2020). As insulin resistance progresses, compensatory hyperinsulinemia occurs, followed by impaired glucose homeostasis, which can lead to metabolic syndrome or type 2 diabetes (Lestari, 2019; Dewi, 2016).

Recent studies have highlighted the role of dietary patterns in the modulation of inflammation and insulin sensitivity. The Dietary Inflammatory Index (DII), developed by Shivappa et al. (2017), provides a standardized method to quantify the inflammatory potential of a person's diet based on the intake of nutrients and food components (Marx et al., 2021). Diets with high DII scores, often rich in saturated fats and refined carbohydrates, and low in antioxidants, are considered pro-inflammatory, whereas diets with low DII scores, rich in fiber, fruits, and vegetables, are anti-inflammatory. Several epidemiological studies have demonstrated that higher DII scores are associated with increased levels of inflammatory biomarkers and insulin resistance, as measured by the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) (Shu et al., 2022; Phillips et al., 2019).

HOMA-IR is a widely used clinical and research tool for assessing insulin resistance based on fasting glucose and insulin levels (Phillips et al., 2019). Elevated HOMA-IR values indicate a reduced ability of insulin to facilitate glucose uptake, and are often found in individuals with obesity, metabolic syndrome, or prediabetes. A previous study in Brazil reported a HOMA-IR cut-off of 2,80 associated with obesity in adolescents, with women and obese individuals showing a higher prevalence of insulin resistance (Chissini, 2020). Despite a growing body of international evidence, limited research has examined the association between DII and HOMA-IR in the Indonesian adult population, particularly in urban areas such as Semarang.

In Indonesia, where dietary transitions toward processed and high-fat foods are increasingly common, understanding the inflammatory potential of diets and their relationship to metabolic health is crucial. To our knowledge, no prior studies have specifically investigated the association between dietary inflammatory potential and insulin resistance among obese adults in Semarang. Filling this knowledge gap is essential to guide targeted nutritional interventions and public health

strategies aimed at reducing the burden of obesity-related metabolic diseases.

Therefore, this study aims to examine the association between the Dietary Inflammatory Index (DII) and insulin resistance, as measured by HOMA-IR, among obese adults in Semarang, Indonesia. This investigation is expected to contribute to a better understanding of the inflammatory role of diet in metabolic health and to offer evidence-based insights for preventive nutrition policies in urban Indonesian populations.

Methods

Study Design and Setting

This study employed an observational, cross-sectional design, and was conducted in Semarang City, Central Java, Indonesia, between September and November 2024. The study protocol was approved by the Ethics Committee of the Faculty of Medicine of Diponegoro University (No. 489/EC/KEPK/FK-UNDIP/IX/2024), prior to the initiation of data collection. All participants provided written informed consent after being fully informed of the objectives, procedures, and benefits of the study.

Study Population and Sampling

The study population consisted of adults aged 26–50 years with obesity (defined as Body Mass Index [BMI] ≥ 25 kg/m^2). A total of 66 participants were recruited using purposive sampling. While this non-random sampling method was appropriate for targeting specific inclusion criteria, we acknowledge that it introduced potential selection bias and limited the generalizability of the findings.

The sample size was calculated using a formula for correlation analysis based on a significance level of 5% ($Z\alpha$ = 1,96), power of 80% ($Z\beta$ = 0,84), and a correlation coefficient (r) of 0,67 derived from prior research (Moslehi et al., 2016). An initial sample size of 59 was obtained and an additional 10% was added to account for potential dropouts, yielding a final target of 66 participants.

Inclusion and Exclusion Criteria

Participants were included if they met the following criteria: aged 26–50 years, obese (BMI ≥ 25 kg/m^2), good general health (body temperature between 36,5°C and 37.2°C), not taking medications or supplements known to affect glucose metabolism, not following a medically prescribed restrictive diet, willingness to fast for 8–12 hours, and willingness to provide informed consent.

Individuals who were uncooperative, withdrew from the study, or were using diet pills or other interventions known to affect glucose or insulin levels were excluded. The term "not on a strict diet" was operationalized as not having been under the supervision of a registered dietitian or physician for caloric or macronutrient restriction in the prior two months.

Recruitment and Data Collection Procedures

Recruitment was conducted through social media and WhatsApp using digital flyers. Eligible individuals were screened through anthropometric assessments, including weight and height, which were measured using calibrated digital scales and a stadiometer, respectively. BMI was calculated as the weight in kilograms divided by the square of height in meters (kg/m^2).

Participants who met the inclusion criteria and signed the informed consent form were invited to provide fasting blood samples (3 mL venous blood) after an 8–12 hour overnight fast. Fasting blood glucose (FBG) levels were measured using a COBAS MIRA spectrophotometer, while fasting insulin levels were analyzed using an ELISA ELX 800 Universal Microplate Reader. Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) was calculated as follows:

$$\text{HOMA} - \text{IR} = \frac{\text{FI (μU/mL)} \times \text{FG (mg/dL)}}{22{,}5}$$

Where:
FI = Fasting Insulin
FG = Fasting Glucose

Participants with HOMA-IR ≥ 2,5 were classified as having insulin resistance (Moslehi et al., 2016). All laboratory equipment used in this study were calibrated prior to analysis, and standard procedures were followed to ensure data accuracy.

Dietary Assessment and DII Calculation

Dietary intake was assessed using a validated Semi-Quantitative Food Frequency Questionnaire (SQ-FFQ) adapted from previous

studies (Syauqy and Diana 2021). The SQ-FFQ was used to estimate the nutrient intake over the past month. Data were entered into NutriSurvey software to calculate the average daily intake of nutrients.

The Dietary Inflammatory Index (DII) was computed according to the standardized methodology of Shivappa et al. (2017), which involves subtracting the global mean intake for each nutrient from the individual intake, dividing by the global standard deviation, converting to a centered percentile score, and multiplying by the corresponding inflammatory effect score. The total DII score was obtained by summing all nutrients.

This study included 29 of the 45 food parameters commonly used in the DII computation: energy, protein, carbohydrates, total fat, saturated fat, trans fat, monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), omega-3, omega-6, cholesterol, fiber, magnesium, iron, selenium, zinc, vitamin A, vitamin C, vitamin D, vitamin E, thiamine, riboflavin, vitamin B6, vitamin B12, folate, niacin, beta-carotene, caffeine, and tea. Several components, such as turmeric, garlic, and polyphenols (Kim et al., 2021), were excluded because of data unavailability in the Indonesian food composition database.

Positive DII scores indicate a pro-inflammatory diet, whereas negative scores indicate an anti-inflammatory diet.

Statistical Analysis

All data were entered into Microsoft Excel and analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Prior to inferential analysis, the normality of continuous variables was assessed using the Shapiro–Wilk test. Univariate analysis was used to summarize the participant characteristics. Bivariate analysis was conducted using Pearson's correlation to assess the relationship between the DII and HOMA-IR.

Multivariate analysis was performed using multiple linear regression with backward elimination to identify the best-fitting model, while controlling for potential confounders. The selection of covariates was based on both theoretical justification and bivariate analysis results. Three models were tested: model 1, DII adjusted for age and BMI; Model 2, DII adjusted for age only; and model 3, DII without adjustment.

The significance level was set at $P < 0,05$. Effect sizes were reported using standardized beta coefficients and R^2 values to evaluate the strength of the associations.

Result and Discussion

Descriptive statistics of the study participants (n = 66) are presented in Table 1. The mean age was 33,55 ± 7,35 years, and the average Body Mass Index (BMI) was 29,38 ± 2,59 kg/m², indicating a population with class I obesity based on WHO classification. Fasting insulin levels displayed high variability (mean: 13,82 ± 12,98 µU/mL; range: 1,30–63,20 µU/mL), suggesting the presence of outliers or inter-individual metabolic differences. Similarly, HOMA-IR scores ranged from 0,31 to 14,35 (mean: 3,38 ± 3,37), with approximately 59% of participants having values ≥2,5, consistent with insulin resistance (Moslehi et al., 2016). The Dietary Inflammatory Index (DII) score ranged from –3,08 to +3,10, with a mean of 0,65 ± 1,19, indicating a slight predominance of pro-inflammatory diets in the sample.

Table 1. Characteristics of study participants (n = 66)

Variabels	Unit	Mean ± SD	Min-Max
Age	Year	33,55 ± 7,35	26-50
BMI	Kg/m²	29,38 ± 2,59	26,12-39,35
Fasting Insulin	µU/mL	13,82 ± 12,98	1,30-63,20
Glucose Fasting	mg/dL	96,57 ± 14,52	69,00-172,00
HOMA-IR Score	-	3,38 ± 3,37	0,31-14,35
DII Score	-	0,65 ± 1,19	-3,08-3,10

Pearson's correlation analysis revealed a statistically significant positive association between DII scores and HOMA-IR values (r = 0,368; p = 0,002) (Table 2). This suggests that individuals with higher dietary inflammatory potential tend to exhibit greater insulin resistance.

Table 2. Correlation Between DII Score and HOMA-IR

Variable	r	p-value
Dietary Inflammatory Index (DII) Score	0,368	0,002

This study identified a significant and positive association between Dietary Inflammatory Index (DII) scores and insulin resistance, as measured by HOMA-IR, among obese adults. Higher DII scores, reflecting a more pro-inflammatory dietary pattern, were consistently associated with elevated insulin resistance even after adjusting for age and BMI. These findings are consistent with the existing literature (Shu et al., 2022; Farhangi et al., 2019), supporting the role of diet-induced inflammation in metabolic dysfunction.

Although the current study did not directly measure inflammatory biomarkers (e.g., CRP and IL-6), the observed association is biologically plausible. Pro-inflammatory diets can activate the nuclear factor kappa B (NF-κB) and c-Jun N-terminal kinase (JNK) pathways in adipose tissue, liver, and muscle, impairing insulin signaling through serine phosphorylation of IRS-1 (Ouchi et al., 2016). This inflammatory cascade contributes to disruption of glucose homeostasis and elevated insulin levels, which is reflected in the increased HOMA-IR scores.

Conversely, anti-inflammatory diets rich in omega-3 fatty acids, fiber, polyphenols, and antioxidants are known to suppress pro-inflammatory cytokines and enhance insulin sensitivity via adiponectin-mediated activation of the PI3K/Akt pathway (Ouchi et al., 2016; Yi et al., 2021). Interventions targeting dietary inflammation have been shown to improve glycemic control and metabolic profiles (Phillips et al. 2018; Calder 2022).

Our findings are consistent with those of Shu et al. (2022), who reported that individuals in the highest tertile of the DII had a significantly greater risk of insulin resistance (OR = 1,79; 95% CI: 1,49–2,14), independent of other risk factors. Similarly, Farhangi et al. (2019) emphasized the detrimental role of pro-inflammatory diets in promoting metabolic disorders including insulin resistance. A recent study by Ramírez-Vélez et al. (2022) also confirmed that the DII was independently associated with HOMA-IR, reinforcing the applicability of the DII as a non-invasive marker for early metabolic risk.

Table 3. Multivariate linear regression: association between DII score and HOMA-IR

Model	Variable	β	95% CI	Adjusted R^2	p-value
1	DII Score	0,362	0,079 – 0,481	0,116	0,007
	Age	–0,144	–0,048 – 0,011		0,224
	BMI	0,045	–0,076 – 0,109		0,727
2	DII Score	0,381	0,115 – 0,475	0,128	0,002
	Age	–0,142	–0,047 – 0,011		0,228
3	DII Score	0,368	0,105 – 0,465	0,122	0,002

Although our regression models showed relatively modest R^2 values (11,6%–12,8%), the DII emerged as the most consistent predictor across all models, surpassing age and BMI. This finding highlights the importance of dietary quality beyond caloric intake in the modulation of insulin sensitivity.

Several unmeasured confounding variables such as physical activity, smoking status, stress levels, and sleep patterns may have influenced the results. These lifestyle factors are known to impact both inflammation and insulin resistance and should be addressed in future studies. Additionally, while the use of a validated SQ-FFQ is a strength, it is susceptible to recall and reporting bias, particularly in obese individuals (Galland, 2015; Shivappa et al., 2017)(Galland, 2015). The wide range of fasting insulin levels also suggests the presence of outliers, which are not excluded but reflect real-world variability (Calder, 2022; Ramírez-Vélez et al., 2022).

This study had several limitations. First, its cross-sectional design limits causal inference, as the findings reflect associations rather than directional effects. Second, the small sample size and purposive sampling reduced the generalizability of the results to a broader population. Third, dietary intake was assessed using the SQ-FFQ, which is subject to recall bias and misreporting, particularly in overweight or obese populations. Fourth, we did not assess physical activity, stress, sleep, or inflammatory biomarkers, which are potential confounders. Finally, the absence of longitudinal or interventional data prevents the determination of the temporal sequence between diet and insulin resistance.

Conclusion

This study confirmed a significant positive association between the Dietary Inflammatory Index (DII) score and Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) among obese adults. Participants with higher DII scores, indicating greater pro-inflammatory dietary potential, demonstrated increased insulin resistance independent of age and body mass index (BMI). These findings highlight the role of diet-induced inflammation as a modifiable factor in the development of metabolic dysfunction.

Given the growing burden of insulin resistance and related metabolic disorders, these results have important clinical and public health implications. Specifically, routine screening of DII scores in obesity management clinics could serve as an early risk stratification tool to identify individuals at a higher metabolic risk. Integrating DII assessments into dietary counseling protocols may facilitate personalized interventions that emphasize anti-inflammatory dietary components, such as increased intake of fruits, vegetables, whole grains, omega-3 fatty acids, and polyphenol-rich foods, while reducing the consumption of processed meat, refined carbohydrates, and added sugars.

Future clinical trials and longitudinal studies are warranted to evaluate the long-term effectiveness of anti-inflammatory dietary strategies for improving insulin sensitivity and preventing metabolic syndrome in high-risk populations.

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