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Body fat percentage, not dietary inflammatory index, is associated with type 2 diabetes in reproductive-aged women: a case-control study

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) in reproductive-aged women is a growing public health concern. While both adiposity and dietary factors are known to influence diabetes risk, their independent contributions remain unclear.
Objectives: This study aimed to assess the association between body fat percentage, Body Mass Index (BMI), and the Dietary Inflammatory Index (DII) with T2DM risk in reproductive-aged women.
Materials and Methods: A case-control study was conducted among 70 women aged 30–50 years. Cases were women with diagnosed T2DM, and controls were non-diabetic women matched by residential area. BMI, body fat percentage, and DII were assessed and categorized: BMI (≤27 vs >27 kg/m²) and body fat percentage (≤32% vs >32%) were measured using a digital scale with bioelectrical impedance function and height assessed with a microtoise, while DII (low, medium, high tertiles) was calculated based on dietary data collected using a semi-quantitative food frequency questionnaire (SQ-FFQ). Statistical analyses were performed using the Mann-Whitney test for group comparisons and logistic regression (bivariate and multivariate) to examine associations.
Results: Body fat percentage and BMI were significantly higher in the T2DM group (p = 0.006 and p = 0.014, respectively). In bivariate logistic regression, high body fat percentage was significantly associated with T2DM (p = 0.007), BMI and DII were not (p = 0.095 and p = 0.662). In the multivariate model, only body fat percentage remained significant (OR = 5.58; 95%CI = 1.39-22.39; p = 0.015), while BMI (OR = 1.04; 95%CI = 0.32-3.41; p = 0.951) and DII (OR = 0.66; 95%CI = 0.34-1.28; p = 0.219) were not.
Conclusion: Body fat percentage was an independent risk factor for T2DM in reproductive-aged women, whereas BMI and DII were not. These findings emphasize the need for precise body composition assessment and suggest adiposity may play a greater role than dietary inflammatory potential.

Keywords*: BMI; body fat percentage; dietary inflammatory index; reproductive-aged women; type 2 diabetes mellitus*

BACKGROUND

Type 2 diabetes mellitus (T2DM) continues to pose a significant global public health challenge, with its prevalence steadily increasing in both low- and high-income countries.[1–3] Among women of reproductive age, the incidence of T2DM is rising due to a combination of lifestyle changes, dietary transitions, and shifts in nutritional status.[4] In Africa, the pooled prevalence of type 2 diabetes among women of reproductive age was found to be 7.2%, with a higher prevalence observed in older age groups.[5] In Indonesia, according to the 2018 Basic Health Research (Riskesdas), the prevalence of T2DM among women has increased compared to 2013, and the number of female T2DM cases surpasses that of male patients. This demographic is especially important because early identification and intervention may prevent long-term complications and reduce the burden of chronic diseases in later life. We focused on women of reproductive age since cyclical hormonal fluctuations during the menstrual cycle influence appetite regulation, body composition, and insulin sensitivity, making this population particularly relevant for examining the link between metabolic factors and T2DM risk.

Body composition, particularly excess body fat, plays a critical role in the pathophysiology of T2DM. Excess body fat contributes to the development of insulin resistance in T2DM through adipocyte dysfunction, macrophage infiltration, and low-grade chronic inflammation.[6] While Body Mass Index (BMI) has long been used as a standard indicator to assess overweight and obesity, it does not differentiate between fat mass and lean mass and may not accurately reflect metabolic risk in all individuals, particularly among women. Therefore, measuring body fat percentage is increasingly recognized as a more specific and sensitive marker for evaluating obesity-related health outcomes, including T2DM risk.

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In addition to body composition, dietary patterns also contribute to the development of metabolic disorders. The Dietary Inflammatory Index (DII) was developed to assess the inflammatory potential of an individual's diet based on the intake of various nutrients and food components.[7] Diets with high DII scores are considered pro-inflammatory and have been associated with increased risk of chronic diseases, including T2DM.[8] However, findings have been inconsistent across populations, and the strength of this association may vary depending on other individual characteristics, such as adiposity and metabolic status. Several studies have shown that individuals with higher DII scores exhibit elevated levels of inflammatory biomarkers such as C-reactive protein (CRP) and interleukin-6 (IL-6), supporting the biological plausibility of this index.[9,10] Moreover, the impact of DII may be influenced by cultural dietary patterns and local food composition, which could explain variations observed across different studies.

Despite growing interest in the relationship between diet, inflammation, and metabolic diseases, few studies have examined the combined roles of DII and body fat percentage in predicting the risk of T2DM, particularly among reproductive-aged women. Most existing research still focuses on BMI as the primary measure of obesity, potentially underestimating the true metabolic risk carried by excess body fat. This study aimed to examine the associations between Dietary Inflammatory Index and body fat percentage with the risk of type 2 diabetes mellitus in reproductive-aged women using a case-control design. Specifically, it seeks to determine which of these factors more strongly predicts T2DM risk in this population.

MATERIALS AND METHODS

Study Design and Participants

This study employed an observational analytical design with a case-control approach, conducted from July to September 2024. Participants were selected using purposive sampling based on predefined inclusion and exclusion criteria. The study included women aged 30–50 years who were divided into case and control groups. The inclusion criteria for the case group were: (1) women aged 30–50 years, (2) diagnosed with type 2 diabetes mellitus (T2DM), (3) taking antidiabetic medication, and (4) not diagnosed with other degenerative diseases such as hypertension or dyslipidemia. The inclusion criteria for the control group were: (1) women aged 30–50 years, (2) not diagnosed with T2DM, and (3) free from other degenerative diseases such as hypertension or dyslipidemia. Matching was performed based on residential area, assuming that participants living in the same area shared similar sociodemographic characteristics. Exclusion criteria included women who were menopausal, pregnant, in the postpartum period, or exclusively breastfeeding. The study was carried out at four public health centers (*Puskesmas*) in Bekasi City, Indonesia, namely Bekasi Jaya, Karang Kitri, Mustika Jaya, and Jatiasih. The population in this study comprised all women of reproductive age who were patients at public health centers implementing the *Prolanis* program in Bekasi City. The sample size was determined using the Sample Size Calculator version 2.0 from the World Health Organization (WHO).[11] Based on the calculation, a minimum of 35 participants per group was required, resulting in a total of 70 participants, consisting of 35 cases and 35 controls.

Subjects were divided into two groups. The case group included women aged 30–50 years who had been diagnosed with type 2 diabetes mellitus (T2DM), were taking antidiabetic medications, and had no history of other degenerative diseases such as hypertension or cardiovascular disease. The control group consisted of women aged 30–50 years with no diagnosis of T2DM and no history of other degenerative diseases. Matching was performed based on residential area, under the assumption that geographical proximity reflects similar sociodemographic characteristics. Exclusion criteria included pregnant women, postpartum mothers, and those exclusively breastfeeding during the study period. All participants provided written informed consent prior to participation. This study received ethical approval from the Health Research Ethics Committee of BRIN (National Research and Innovation Agency) under reference number 131/KE.03/SK/07/2024.

Anthropometric and Body Composition Measurements

Body weight and body fat percentage were measured using a digital scale with bioelectrical impedance analysis (Omron HBF-375, Omron Healthcare, Kyoto, Japan), and height was measured with a microtoise (Stature meter, Indonesia). Body Mass Index (BMI) was calculated as weight (kg) divided by height squared (m^2) and categorized as non-obese (≤ 27 kg/m^2) and obese (>27 kg/m^2) according to the Indonesian classification.

Dietary Assessment and DII Calculation

The Dietary Inflammatory Index (DII) was calculated to evaluate the inflammatory potential of each participant's diet. Dietary intake was assessed using a Semi-Quantitative Food Frequency Questionnaire (SQ-FFQ), which captured habitual dietary patterns over the previous 6 month. Nutrient intake was analyzed based on the Indonesian Food Composition Table 2019. The DII was computed based on the method developed by Shivappa et al., which compares an individual's intake of specific dietary components to a global reference database and assigns an inflammatory score based on literature-derived values.[7] In this study, seventeen components were used for the DII calculation, including macronutrients (energy, carbohydrate, protein, fat, and fiber), specific fatty acids (omega-3 and omega-6), vitamins and minerals (vitamins A, C, and E, zinc, magnesium, and iron), bioactive compounds (beta-carotene), and caffeine. For each component, a Z-score was calculated and then converted into percentile scores and centered to minimize skewness. Each component's centered percentile was multiplied by its respective inflammatory effect score, derived from the global literature review. The final DII score was obtained by summing the weighted contributions of each component, with higher scores indicating a more pro-inflammatory dietary profile and lower scores indicating an anti-inflammatory profile. For statistical analysis, DII values were categorized into tertiles representing low, moderate, and high inflammatory potential.

Diagnosis of T2DM

The diagnosis of type 2 diabetes mellitus was confirmed through medical records and physician diagnosis using the PERKENI criteria.[12]

Statistical Analysis

Descriptive statistics were used to summarize the participants' characteristics. Continuous variables that were not normally distributed are presented as median and interquartile range (IQR), where the IQR represents the difference between the 75th percentile (Q3) and the 25th percentile (Q1). Differences between the case and control groups were analyzed using Mann–Whitney U tests. In addition, bivariate logistic regression was performed to calculate crude odds ratios (ORs) for the association between each independent variable and T2DM status. Variables with $p < 0.25$ in bivariate analysis were included in the multivariate logistic regression model to identify independent predictors of T2DM. A p-value < 0.05 was considered statistically significant. All statistical analyses were performed using Real Statistics for Excel.

RESULTS

This study involved 70 reproductive-aged women (30–50 years), divided equally into case (type 2 diabetes mellitus) and control groups. The comparison of mean values between the two groups showed significant differences in Body Mass Index (BMI) and Body Fat Percentage (PLT), while the Dietary Inflammatory Index (DII) was not significantly different. The group comparison results are presented in Table 1.

Table 1. Subjects' Characteristics

Characteristics	T2DM		p-value
	Yes median±IQR	No median±IQR	
BMI (kg/m^2)	27.3±4.8	25.2±4.8	0.014*
Body Fat (%)	36.3±5.15	32.2±7	0.006*
DII (score)	-0.78±0.5	-0.65±0.4	0.125

*Significant based on Mann Whitney test(p-value<0.05)
BMI – Body Mass Index; DII – Dietary Inflammatory Index; T2DM – Type 2 Diabetes Mellitus

Table 1 presents a comparison of characteristics between reproductive-aged women with and without T2DM. The median BMI was significantly higher in the diabetes group compared to the non-diabetes group (27.3 ± 4.8 vs 25.2 ± 4.8; $p = 0.014$). Similarly, body fat percentage was significantly higher among those with diabetes (36.3 ± 5.15 vs 32.2 ± 7; $p = 0.006$). Although the DII score appeared lower (more anti-inflammatory) in the diabetes group than in the non-diabetes group (-0.78 ± 0.5 vs -0.65 ± 0.4), the difference was not statistically significant ($p = 0.125$).

Bivariate logistic regression was performed to assess the crude association of each independent variable with the risk of type 2 diabetes mellitus. For this analysis, all predictor variables were categorized. Body Mass Index (BMI) was classified as normal (≤27 kg/m²) and obese (>27 kg/m²), body fat percentage was classified

as normal (≤32%) and high (>32%), and DII was categorized into tertiles[13] (low, moderate, and high inflammatory potential).

As presented in Table 2, women with obesity were more likely to have T2DM (54.2%) compared to their non-obese counterparts (36.0%). Similarly, women with high body fat percentage had a markedly higher prevalence of T2DM (61.5%) compared to those with normal body fat (16.7%). In contrast, the majority of women with normal body fat were in the non-diabetic group (83.3%). For DII, the proportion of T2DM cases was relatively comparable across the low (54.2%), moderate (47.8%), and high (47.8%) tertiles, indicating no clear trend across categories. Regression analysis further demonstrated that body fat percentage was significantly associated with T2DM (p = 0.007), while BMI showed a borderline association (p = 0.095). No significant association was observed for DII (p = 0.662). The crude odds ratios (ORs) and 95% confidence intervals (CIs) are shown in Table 2.

Table 2. Association of BMI, Body Fat, and DII with the risk of T2DM

Variable	T2DM		Crude OR (Exp(B))	95% CI	p-value
	Yes n (%)	No n (%)			
BMI			2.28	0.87-5.97	0.095
Obese (>27)	26 (54.2)	19 (45.8)			
Non-Obese (≤27)	9 (36.0)	16 (64.0)			
Body Fat			4.56	1.52-13.72	0.007*
High (>32)	32 (61.5)	20 (38.5)			
Normal (≤32)	3 (16.7)	15 (83.3)			
DII			0.88	0.49-1.56	0.662
Low (<-0.871)	13 (54.2)	11 (45.8)			
Moderate ([-0.871]─[-0.478])	11 (47.8)	12 (52.2)			
High (>-0,478)	11 (47.8)	12 (52.2)			

*Significant based on logistic regression test (p-value<0.05)
BMI – Body Mass Index; DII – Dietary Inflammatory Index; T2DM – Type 2 Diabetes Mellitus

The logistic regression analysis showed that body fat percentage was a significant predictor of type 2 diabetes mellitus among reproductive-aged women. Individuals with higher body fat had an odds ratio (OR) of 4.56 with a 95% confidence interval (CI) of 1.52 to 13.72, indicating a markedly increased risk of diabetes. Body mass index (BMI) demonstrated an elevated odds ratio of 2.28 (95% CI: 0.87–5.97), suggesting a potential association, though it did not reach statistical significance. In contrast, the dietary inflammatory index (DII) showed no meaningful association with diabetes risk, with an OR of 0.88 (95% CI: 0.49–1.56).

Multivariate logistic regression analysis was conducted to identify independent predictors of type 2 diabetes mellitus. Variables with a p-value < 0.25 in the bivariate analysis (BMI and body fat percentage) were included in the model. Additionally, the Dietary Inflammatory Index (DII) was included due to its theoretical relevance to diabetes risk, despite not being statistically significant in the bivariate model. For this analysis, all predictor variables were entered as categorical variables. The multivariate logistic regression model is presented in Table 3.

Table 3. Adjusted Odds Ratios of BMI, Body Fat, and DII for T2DM

Variable	Adjusted OR	95% CI	p-value
BMI	1.04	0.32-3.41	0.951
Body Fat	5.58	1.39-22.39	0.015*
DII	0.66	0.34-1.28	0.219

* Values are adjusted odds ratios (ORs) with 95% confidence intervals (CIs), obtained from multivariate logistic regression including BMI, body fat percentage, and DII in the model. Significant results were considered at p < 0.05.
BMI – Body Mass Index; DII – Dietary Inflammatory Index; T2DM – Type 2 Diabetes Mellitus

The multivariable logistic regression analysis identified body fat percentage as an independent risk factor for type 2 diabetes mellitus in reproductive-aged women. After adjusting for other variables, women with higher body fat had an adjusted OR of 5.58, with a 95% CI ranging from 1.39 to 22.39. This indicates that higher body fat remained strongly associated with increased diabetes risk, independent of BMI and dietary inflammatory index. In contrast, BMI showed no significant association, with an adjusted OR of 1.04 (95% CI: 0.32–3.41), and the DII also did not demonstrate a meaningful relationship, with an adjusted OR of 0.66 (95% CI: 0.34–1.28).

DISCUSSION

This study aimed to examine the association between body composition and dietary inflammatory potential with the risk of type 2 diabetes mellitus (T2DM) among reproductive-aged women using a case-control design. The findings indicate that body fat percentage is a significant and independent risk factor for T2DM, whereas Body Mass Index (BMI) and Dietary Inflammatory Index (DII) were not significantly associated in the adjusted model.

Although obesity is widely recognized as a risk factor for T2DM, our findings indicate that BMI did not remain significant after adjustment, suggesting that body composition may be more relevant than overall body size. Previous studies have shown that women of reproductive age with higher BMI are at increased risk of developing T2DM, particularly those who experience rapid changes in BMI across their lifespan.[14] Moreover, each 5% increase in BMI has been associated with a 1.33-fold higher risk of T2DM in this population.[15]

The significant association between high body fat percentage and T2DM supports previous research highlighting the role of adiposity as a major contributor to insulin resistance and metabolic dysfunction.[16] Unlike BMI, which does not distinguish between fat and lean mass, body fat percentage provides a more precise estimate of actual fat accumulation, particularly in women.[17] This finding underscores the importance of assessing adiposity through body composition measurements rather than BMI alone, especially in female populations where fat distribution and hormonal influences may vary significantly. Although BMI showed a borderline association in the bivariate analysis, its effect was no longer significant after adjusting for body fat percentage. This suggests that the impact of BMI on diabetes risk may be mediated or confounded by actual fat mass, reinforcing the superiority of direct adiposity measures in predicting metabolic outcomes.

The relationship between adiposity and metabolic dysfunction significantly impacts the development of T2DM by influencing insulin resistance and other metabolic pathways. Adiposity, particularly when associated with metabolic dysfunction, exacerbates the risk of T2DM through various mechanisms, including the secretion of inflammatory mediators and adipokines, which impair insulin sensitivity. This relationship is complex and involves multiple physiological processes that contribute to the pathogenesis of T2DM.

Although our study did not directly measure visceral fat, previous evidence indicates that adipose tissue distribution, particularly visceral fat, plays a central role in insulin resistance. Visceral fat is metabolically active, prone to lipolysis, and releases free fatty acids and inflammatory mediators that impair insulin action in peripheral tissues.[18] This mechanism may partly explain the observed association between higher body fat percentage and increased risk of T2DM in our study. The accumulation of adipose tissue leads to the secretion of adipokines, such as leptin and adiponectin, which play crucial roles in energy metabolism and insulin sensitivity. Dysregulation of these adipokines in obesity contributes to metabolic dysfunction and T2DM.[19] White adipose tissue (WAT) and brown adipose tissue (BAT) are involved in regulating systemic energy homeostasis. Dysfunctional WAT, characterized by hypertrophy and inflammation, contributes to chronic low-grade inflammation and insulin resistance.[19]

Interestingly, the Dietary Inflammatory Index (DII), which theoretically reflects the pro- or anti-inflammatory potential of dietary intake, was not significantly associated with T2DM in this study, even after being retained in the multivariate model for its theoretical importance. In our subjects, the median DII scores were similar between T2DM and non-T2DM groups, and the distribution across tertiles (low, moderate, and high inflammatory potential) showed no clear differences. This lack of variation may partly explain the absence of a significant association in our analysis. Several explanations may account for this finding. First, the variation in DII scores among participants may not have been substantial enough to detect a meaningful effect. Second, the components in the DII calculation are derived from a global database, and do not necessarily reflect the local Indonesian dietary context, which may differ from the Western population studies from which the index was developed. Third, it is also possible that the relationship between dietary inflammation and diabetes is less direct, or may be mediated through other mechanisms such as adiposity or oxidative stress, which were not assessed in this study.

Moreover, our findings reflect the complex interplay between diet, body composition, and chronic disease risk. While diet remains a modifiable factor of interest, this study suggests that in this population, body fat accumulation is a more robust and consistent predictor of T2DM risk than dietary inflammatory scores. Previous studies have shown that dietary inflammatory potential contributes to T2DM primarily through its effects on adiposity and systemic inflammation rather than as a direct independent factor.[20,21] This indicates that interventions targeting both dietary quality and body composition may offer synergistic benefits for diabetes prevention.

This study has several limitations. First, its case–control design precludes establishing causal relationships between the identified risk factors and T2DM. Second, body fat percentage was assessed using BIA, which, while practical and non-invasive, may be less precise than gold-standard methods such as DXA or MRI, and visceral fat was not directly measured. Third, dietary intake was assessed using an SQ-FFQ, which is subject to recall bias and may not fully capture habitual diet. Despite these limitations, the study provides novel insights into the role of body fat percentage in T2DM among women of reproductive age.

CONCLUSIONS

In conclusion, body fat percentage was found to be a significant and independent risk factor for type 2 diabetes mellitus in reproductive-aged women, whereas BMI and Dietary Inflammatory Index were not significantly associated. These findings highlight the importance of assessing body fat directly and suggest that adiposity may be a more critical factor than dietary inflammation in the development of diabetes in this population. These results emphasize the need for routine screening of body fat percentage, not just BMI, in early identification of metabolic risk among reproductive-aged women. Public health interventions should also consider targeted strategies to reduce adiposity in addition to promoting dietary quality.

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CONFLICT OF INTEREST

Authors declare no conflict of interest to disclose.

DECLARATION USE AI

During the preparation of this manuscript, the authors used ChatGPT (OpenAI) to assist with language refinement, improving clarity and readability, restructuring selected paragraphs, and enhancing the overall flow of the manuscript. All scientific interpretations, data analyses, conclusions, and final editorial decisions were made by the authors. The authors reviewed and edited all AI-assisted content and take full responsibility for the content of the published article.

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