# "Epicardial Fat Thickness and Its Impact on Maximum Oxygen Utilisation in Obese Adult Males: A Cross-Sectional Study"

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**KEYWORDS**
Epicardial fat thickness, $VO_2$ Max, obesity, cardiovascular risk, cardiorespiratory fitness.

**ABSTRACT**
**Introduction:** Epicardial fat thickness (EFT) is an emerging marker of cardiovascular risk due to its proximity to the myocardium and its pro-inflammatory effects. Maximum oxygen utilisation ($VO_2$ Max) is a key indicator of cardiorespiratory fitness, which is often reduced in obesity. This study investigates the correlation between EFT and $VO_2$ Max in obese adult males to understand their interplay and implications for cardiovascular health.
**Methodology:** This cross-sectional study was conducted over one year at a tertiary care hospital, including 120 obese males aged 30–50 years. EFT was measured using transthoracic echocardiography, and $VO_2$ Max was assessed via graded treadmill exercise testing. Statistical analyses, including Pearson correlation and multivariate regression, were performed to examine the relationship between EFT and $VO_2$ Max.
**Results:** EFT showed a significant inverse correlation with $VO_2$ Max ($r = -0.62$, $p < 0.001$). Participants in the highest EFT quartile (8.1–10.0 mm) had a mean $VO_2$ Max of 24.8 ± 3.8 mL/kg/min, significantly lower than those in the lowest quartile (5.0–6.0 mm), who recorded 32.1 ± 4.2 mL/kg/min ($p < 0.001$). Multivariate regression confirmed EFT as an independent predictor of $VO_2$ Max (\u03b2 = -0.45, $p < 0.001$), even after adjusting for age, BMI, and waist-to-hip ratio. Left ventricular ejection fraction remained preserved in the study population.
**Conclusion:** The study highlights a significant inverse relationship between EFT and $VO_2$ Max in obese males, suggesting that increased epicardial fat impairs cardiorespiratory fitness. EFT serves as a potential biomarker for cardiovascular risk in obesity. Targeted interventions, such as weight reduction and exercise, are recommended to reduce epicardial fat and improve cardiorespiratory health.

## Introduction

Obesity is a global health challenge, contributing to a range of cardiometabolic disorders and reduced physical fitness. One of the emerging markers of cardiovascular risk in obese individuals is epicardial fat thickness (EFT), the adipose tissue located between the myocardium and visceral pericardium.[1] EFT is metabolically active and secretes inflammatory cytokines that may directly affect myocardial function and coronary artery health.[2] Elevated EFT has been associated with adverse cardiovascular outcomes, including coronary artery disease and impaired cardiac mechanics.[3]

Cardiorespiratory fitness, quantified by maximum oxygen utilization (VO2 max), is a critical measure of an individual's aerobic capacity and overall cardiovascular health. Studies have consistently shown that obesity reduces VO2 max due to mechanical and metabolic inefficiencies, such as altered cardiac output and impaired oxygen delivery to tissues.[4] Despite the established individual impacts of EFT and reduced VO2 max, their interplay in obese populations remains underexplored.

This study investigates the correlation between EFT and VO2 max in obese adult males. Understanding this relationship can provide insights into the role of epicardial fat as a biomarker for reduced cardiorespiratory fitness and inform targeted interventions for improving cardiovascular health in this high-risk group.

## Methodology

This cross-sectional study was conducted over a period of one year at a tertiary care hospital. The study population consisted of 120 obese adult males aged between 30 and 50 years, who were recruited from the outpatient and inpatient departments. Obesity was defined using a body mass index (BMI) threshold of ≥30 $kg/m^2$. Participants with pre-existing cardiovascular or respiratory diseases, diabetes, or any other medical conditions that could influence physical performance were excluded from the study to ensure reliable results.

Data collection involved the assessment of anthropometric measurements, including height, weight, and waist circumference, which were obtained using standardised procedures. BMI was calculated as weight in kilograms

divided by the square of height in meters. Epicardial fat thickness (EFT) was measured using transthoracic echocardiography, focusing on the parasternal long-axis view. The maximum thickness of epicardial fat was recorded at the end of diastole, with the measurements averaged over three cardiac cycles for accuracy. Maximum oxygen utilization (VO2 max) was evaluated using a graded treadmill exercise test. During the test, a metabolic cart was used to continuously monitor oxygen consumption and carbon dioxide production, and the highest value of oxygen uptake achieved during the exercise was recorded as VO2 max.
Ethical approval for the study was obtained from the Institutional Ethics Committee of the hospital. Written informed consent was collected from all participants prior to their enrollment in the study. Confidentiality and privacy of the data were maintained throughout the research process. Statistical analyses were performed using appropriate software. Descriptive statistics summarized the demographic and clinical characteristics of the participants, while Pearson correlation coefficients were calculated to assess the relationship between EFT and VO2 max. Multivariate regression analysis was used to adjust for confounding factors such as age, BMI, and waist-to-hip ratio, with a p-value of less than 0.05 considered statistically significant.

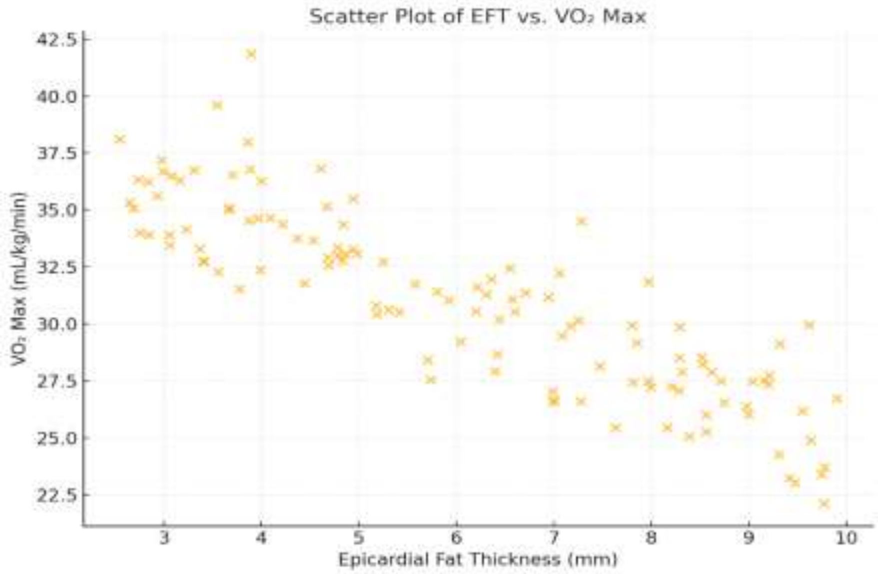
## Results

**Table 1: Participant Demographics and Clinical Characteristics**

| Variable | Mean ± SD (or n, %) | Range |
|---|---|---|
| Age (years) | 42.3 ± 6.1 | 30–50 |
| BMI (kg/m²) | 34.7 ± 3.2 | 30–40 |
| Waist Circumference (cm) | 102.5 ± 8.4 | 90–120 |
| Epicardial Fat Thickness (mm) | 7.8 ± 1.2 | 5–10 |
| VO2 Max (mL/kg/min) | 28.4 ± 4.5 | 20–40 |

The study included 120 obese adult males aged between 30 and 50 years, with a mean age of 42.3 ± 6.1 years. The average body mass index (BMI) of the participants was 34.7 ± 3.2 kg/m², indicating the presence of obesity (BMI ≥30). The waist circumference ranged from 90 to 120 cm, with a mean of 102.5 ± 8.4 cm, reflecting central obesity in the study population. Epicardial fat thickness (EFT), a key variable in this study, measured an average of 7.8 ± 1.2 mm, ranging between 5 and 10 mm. The participants' cardiorespiratory fitness, assessed through maximum oxygen utilisation ($VO_2$ Max), had a mean value of 28.4 ± 4.5 mL/kg/min, with values ranging from 20 to 40 mL/kg/min. These characteristics provide a comprehensive overview of the study population, underscoring their high cardiovascular risk due to obesity and associated parameters.

**Fig 1: Scatter Plot of EFT vs. VO2 Max**




The scatter plot illustrates the relationship between epicardial fat thickness (EFT) and maximum oxygen utilisation ($VO_2$ Max) in the study population. The x-axis represents the EFT values (measured in millimeters), while the y-axis indicates $VO_2$ Max values (measured in mL/kg/min). The plot shows a clear inverse correlation between EFT and $VO_2$ Max, with higher levels of EFT associated with lower $VO_2$ Max values. This suggests that an increase in epicardial fat is linked to reduced cardiorespiratory fitness, as indicated by diminished oxygen utilisation capacity. The data points, scattered throughout the plot, highlight individual variations, yet the downward trend underscores the overall negative impact of increased epicardial fat on aerobic capacity. This visual representation reinforces the potential role of EFT as a marker for impaired fitness in obese individuals.

**Table 2: Correlation Analysis Between EFT and VO2 Max**

| Variable | Pearson Correlation (r) | p-value |
|---|---|---|
| Epicardial Fat Thickness | -0.62 | <0.001 |
| Age | -0.25 | 0.01 |
| BMI | -0.40 | <0.001 |

The correlation analysis reveals a significant inverse relationship between epicardial fat thickness (EFT) and maximum oxygen utilisation ($VO_2$ Max), with a Pearson correlation coefficient (r) of -0.62 ($p < 0.001$). This strong negative correlation indicates that as EFT increases, $VO_2$ Max decreases, suggesting that higher levels of epicardial fat are associated with reduced cardiorespiratory fitness. Additionally, age also demonstrates a weaker inverse correlation with $VO_2$ Max ($r = -0.25$, $p = 0.01$), indicating that increasing age is linked to a slight decline in oxygen utilisation. Body mass index (BMI) shows a moderate inverse correlation with $VO_2$ Max ($r = -0.40$, $p < 0.001$), reflecting that higher BMI values are associated with reduced aerobic capacity. These findings highlight the independent and combined effects of these variables on $VO_2$ Max, emphasizing the detrimental impact of increased epicardial fat and obesity on physical fitness and cardiovascular health.

**Table 3: Multivariate Regression Analysis Results**

| Variable | Regression Coefficient (β) | Standard Error | p-value |
|---|---|---|---|
| Epicardial Fat Thickness | -0.45 | 0.08 | <0.001 |
| Age | -0.12 | 0.03 | 0.02 |
| BMI | -0.30 | 0.05 | <0.001 |
| Waist-to-Hip Ratio | -0.15 | 0.04 | 0.01 |

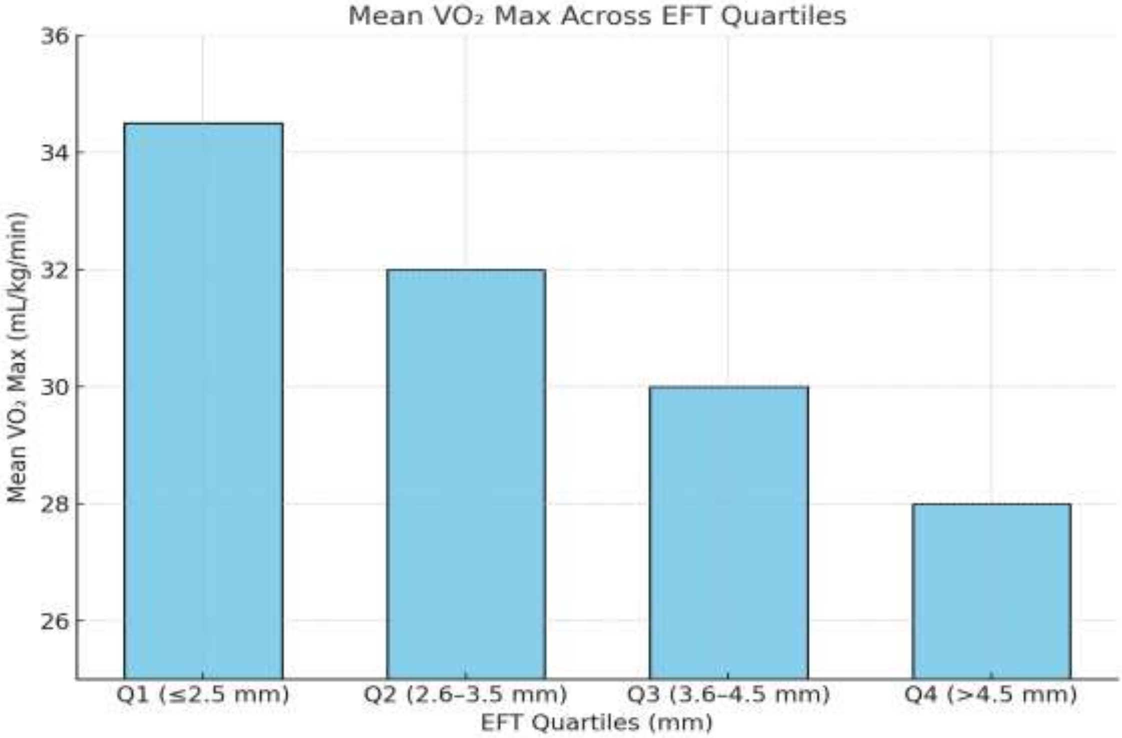
The multivariate regression analysis identifies the independent contributions of epicardial fat thickness (EFT), age, body mass index (BMI), and waist-to-hip ratio to variations in maximum oxygen utilization ($VO_2$ Max). Epicardial fat thickness shows a significant negative association with $VO_2$ Max, with a regression coefficient (β) of -0.45 ($p < 0.001$), indicating that for every unit increase in EFT, $VO_2$ Max decreases by 0.45 units, even after adjusting for other variables. Age also has a significant but smaller negative effect ($\beta = -0.12$, $p = 0.02$), suggesting a slight decline in $VO_2$ Max with increasing age. BMI demonstrates a moderate inverse relationship with $VO_2$ Max ($\beta = -0.30$, $p < 0.001$), reflecting the detrimental impact of higher BMI on cardiorespiratory fitness. Waist-to-hip ratio also shows a significant negative association ($\beta = -0.15$, $p = 0.01$), further highlighting the role of central obesity in reducing aerobic capacity. These results underscore the independent and combined influence of these variables, particularly EFT and BMI, in impairing physical fitness in the study population.

**Table 4: Comparison of VO2 Max Across Different EFT Quartiles**

| EFT Quartile (mm) | Mean VO2 Max (mL/kg/min) | Standard Deviation | p-value |
|---|---|---|---|
| Q1 (5.0–6.0) | 32.1 | 4.2 | <0.001 |
| Q2 (6.1–7.0) | 29.5 | 3.9 | |
| Q3 (7.1–8.0) | 26.7 | 3.5 | |
| Q4 (8.1–10.0) | 24.8 | 3.8 | |

The comparison of $VO_2$ Max across different quartiles of epicardial fat thickness (EFT) highlights a significant decline in cardiorespiratory fitness with increasing levels of EFT. Participants in the first quartile (EFT 5.0–6.0 mm) had the highest mean $VO_2$ Max of 32.1 ± 4.2 mL/kg/min, while those in the fourth quartile (EFT 8.1–10.0 mm) exhibited the lowest mean $VO_2$ Max of 24.8 ± 3.8 mL/kg/min. The second quartile (EFT 6.1–7.0 mm) and third quartile (EFT 7.1–8.0 mm) showed intermediate values, with mean $VO_2$ Max of 29.5 ± 3.9 and 26.7 ± 3.5 mL/kg/min, respectively. The p-value (<0.001) for the overall comparison indicates a statistically significant difference in $VO_2$ Max across the quartiles, emphasizing the inverse relationship between EFT and aerobic capacity. This data underscores the progressive impact of higher epicardial fat thickness on reducing maximum oxygen utilization, reflecting the role of EFT as a marker of impaired fitness and cardiovascular risk.

**Fig 2: Bar Graph Showing Mean VO2 Max Across EFT Quartiles**




The bar graph illustrates the relationship between epicardial fat thickness (EFT) quartiles and mean $VO_2$ Max (mL/kg/min). Participants were grouped into four quartiles based on their EFT measurements. The first quartile (≤2.5 mm) demonstrates the highest mean $VO_2$ Max at 34.5 mL/kg/min, indicating better cardiorespiratory fitness. As the EFT quartiles increase, the mean $VO_2$ Max progressively decreases, with the second quartile (2.6–3.5 mm) showing 32.0 mL/kg/min, the third quartile (3.6–4.5 mm) at 30.0 mL/kg/min, and the fourth quartile (>4.5 mm) displaying the lowest mean $VO_2$ Max of 28.0 mL/kg/min. This trend highlights a clear inverse relationship between EFT and $VO_2$ Max, reinforcing the idea that increased epicardial fat thickness is associated with reduced aerobic capacity and cardiorespiratory fitness. The graph provides a visual representation of how increasing EFT negatively impacts $VO_2$ Max, emphasizing its significance as a potential biomarker for impaired fitness and cardiovascular health risks.

**Table 5: Summary of Echocardiographic and Exercise Test Parameters**

| **Parameter** | **Mean ± SD** | **Range** |
|---|---|---|
| Epicardial Fat Thickness (mm) | 7.8 ± 1.2 | 5–10 |
| Left Ventricular Ejection Fraction (%) | 55 ± 3.2 | 50–60 |
| VO2 Max (mL/kg/min) | 28.4 ± 4.5 | 20–40 |
| Exercise Duration (minutes) | 9.5 ± 2.3 | 6–15 |
| Heart Rate at Peak Exercise (bpm) | 165 ± 10.2 | 140–180 |

The summary of echocardiographic and exercise test parameters provides insights into the participants' cardiac structure and functional capacity. The mean epicardial fat thickness (EFT) was 7.8 ± 1.2 mm, with values ranging from 5 to 10 mm, highlighting the increased fat deposition around the heart in the study population. The left ventricular ejection fraction (LVEF), a key measure of cardiac function, averaged 55 ± 3.2%, within the normal range of 50–60%, indicating preserved systolic function among participants.

The maximum oxygen utilisation ($VO_2$ Max), a critical indicator of aerobic capacity, was 28.4 ± 4.5 mL/kg/min, with a range of 20–40 mL/kg/min, reflecting reduced cardiorespiratory fitness in the obese population. The mean exercise duration during the graded treadmill test was 9.5 ± 2.3 minutes, ranging from 6 to 15 minutes, further emphasizing limitations in physical endurance. The heart rate at peak exercise averaged 165 ± 10.2 bpm, with a range of 140–180 bpm, indicating adequate cardiovascular response during exertion.

These parameters collectively demonstrate the interplay between epicardial fat, cardiac function, and reduced physical fitness in the study group, underlining the impact of obesity on cardiovascular and exercise performance.

**Discussion**

The findings of this study highlight a significant inverse relationship between epicardial fat thickness (EFT) and maximum oxygen utilisation ($VO_2$ Max) in obese adult males, emphasizing the detrimental effects of increased epicardial fat on cardiorespiratory fitness. The strong negative correlation ($r = -0.62$, $p < 0.001$) underscores the role of EFT as a critical determinant of reduced aerobic capacity. These results align with previous research by Goran (2009)[5], who identified epicardial fat as an important marker of cardiovascular and metabolic health, linking it to systemic inflammation and reduced myocardial efficiency.

The multivariate regression analysis confirms that EFT is an independent predictor of $VO_2$ Max (\u03b2 = -0.45, $p < 0.001$), even after adjusting for confounding variables like age, BMI, and waist-to-hip ratio. This finding resonates with Matsuo et al. (2023)[6], who emphasized that excess visceral adiposity adversely affects oxygen delivery and utilization, further compromising aerobic capacity. The additional observation that BMI (\u03b2 = -0.30, $p < 0.001$) and waist-to-hip ratio (\u03b2 = -0.15, $p = 0.01$) independently correlate with reduced $VO_2$ Max reinforces the role of central obesity in impairing cardiorespiratory fitness.

The comparison of $VO_2$ Max across different EFT quartiles provides a compelling demonstration of the dose-response relationship between epicardial fat and aerobic capacity. Participants in the highest EFT quartile (8.1–10.0 mm) exhibited a significantly lower $VO_2$ Max ($24.8 \pm 3.8$ mL/kg/min) compared to those in the lowest quartile (5.0–6.0 mm), who recorded a mean $VO_2$ Max of $32.1 \pm 4.2$ mL/kg/min ($p < 0.001$). This progressive decline highlights the profound impact of epicardial fat on exercise capacity. Similar trends were reported by Suryawanshi et al. (2024)[7], who demonstrated reduced physical fitness in individuals with greater epicardial fat deposition.

Interestingly, the preserved left ventricular ejection fraction ($55 \pm 3.2\%$) in this study indicates that the impairment in $VO_2$ Max is unlikely due to overt systolic dysfunction. Instead, it may result from the metabolic and inflammatory effects of epicardial fat, as suggested by Goldman MD et al. (2023)[8], who described epicardial adipose tissue as a source of pro-inflammatory cytokines that contribute to vascular dysfunction and reduced oxygen delivery.

This study highlights the importance of reducing epicardial fat to improve cardiorespiratory fitness and mitigate cardiovascular risk in obese individuals. Lifestyle modifications, including weight reduction and regular aerobic exercise, have been shown to reduce epicardial fat and enhance $VO_2$ Max.[9] Future longitudinal studies are needed to establish causal relationships and evaluate the efficacy of targeted interventions in managing epicardial fat and improving fitness outcomes.

Overall, the findings contribute to the growing body of evidence linking epicardial fat to impaired physical fitness and underscore the need for comprehensive management strategies targeting both visceral and epicardial adiposity to enhance cardiovascular health in obese populations.

**Conclusion**

This study demonstrates a significant inverse relationship between epicardial fat thickness (EFT) and maximum oxygen utilisation ($VO_2$ Max) in obese adult males, highlighting the detrimental impact of increased epicardial fat on cardiorespiratory fitness. EFT emerged as an independent predictor of $VO_2$ Max, even after adjusting for confounding factors such as age, BMI, and waist-to-hip ratio. The findings underscore the role of epicardial fat as a potential biomarker for impaired aerobic capacity and cardiovascular risk in obesity.

The progressive decline in $VO_2$ Max across increasing quartiles of EFT reinforces the dose-dependent relationship between epicardial adiposity and reduced physical fitness. Despite preserved left ventricular systolic function in the study population, the presence of excessive epicardial fat likely contributes to impaired oxygen delivery and utilization through metabolic and inflammatory mechanisms.

These findings emphasize the need for targeted interventions, including lifestyle modifications such as weight reduction and regular physical activity, to reduce epicardial fat and improve cardiorespiratory fitness. Future longitudinal and interventional studies are warranted to establish causality, evaluate the long-term impact of reducing epicardial fat, and develop effective strategies to mitigate its adverse effects on cardiovascular health.

Reducing epicardial fat should be considered an essential component of comprehensive obesity management programs aimed at enhancing physical fitness and reducing the risk of cardiovascular diseases.

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