

## Correlation of the *cd36* rs1761667 Polymorphism and Lifestyle Factors with Fatty Acid Perception Threshold

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### ABSTRACT

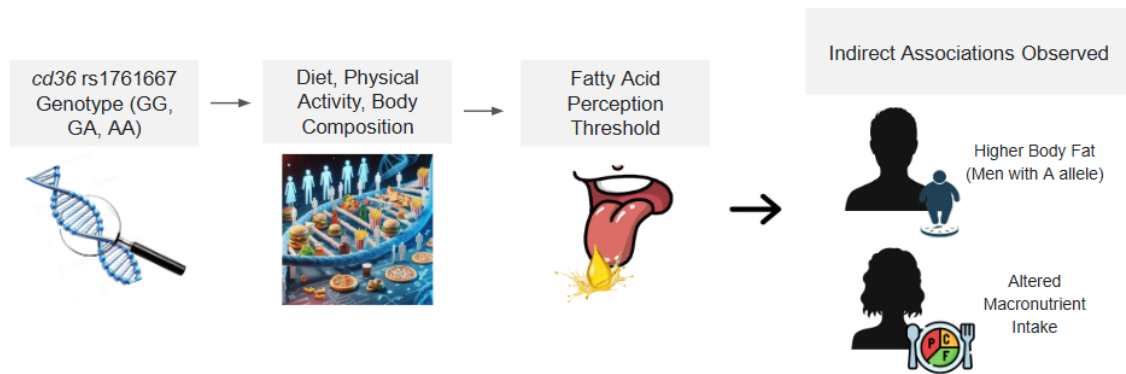
Genetic variations, such as in the *CD36* gene (rs1761667), may influence fat perception and dietary patterns, contributing to obesity. This cross-sectional study involved 87 office workers who underwent genotyping for *cd36* rs1761667, sensory testing for oleic acid perception threshold, and assessments of body composition, physical activity, and dietary intake. No direct association was found between the *cd36* rs1761667 genotype and the fatty acid perception threshold ( $p > 0.05$ ). However, stratified analyses revealed that men carrying the A allele (GA/AA,  $n=23$ ) had a higher body fat percentage compared to those with the GG genotype ( $n=11$ ). In women, GA/AA carriers consumed more protein, while GG carriers derived a higher proportion of energy from carbohydrates. Among overweight individuals, GG carriers reported higher carbohydrate intake than A allele carriers ( $p=0.026$ ). These findings suggest that while *cd36* rs1761667 may not be a primary determinant of fat taste sensitivity in this cohort, it may contribute modestly to body fat composition and dietary behaviors through interactions with gender and BMI status.

#### Key Messages:

- The *cd36* rs1761667 polymorphism is not a primary determinant of fatty acid perception threshold.
- Male carriers of the A allele have significantly higher body fat percentages than GG carriers.
- Genotype-related differences exist in macronutrient intake, with distinct carbohydrate and protein patterns in women and overweight individuals.
- These results underscore that body composition and diet are shaped by complex gene-environment interactions, extending beyond the *CD36* gene alone.

## GRAPHICAL ABSTRACT

### Correlation of the *cd36* rs1761667 Polymorphism and Lifestyle Factors with Fatty Acid Perception Threshold



*cd36* rs1761667 may indirectly influence body composition and eating behavior through its association with body fat and macronutrient intake, supporting personalized approaches in obesity research.

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## INTRODUCTION

Sexuality is an Obesity is a health condition that has been considered a global epidemic by the World Health Organization [1]. Obesity increases the risk of metabolic diseases, such as cardiovascular disease and type 2 diabetes mellitus. Sex-specific differences in body composition may further modulate this risk [2]. The global prevalence of obesity was 23% in 2022, with a continuing upward trend each year. In Indonesia, the Indonesian Health Survey (SKI) reported that 23.4% of individuals aged 18 and older were classified as obese, with a BMI  $\geq 27$  kg/m<sup>2</sup> [3]. According to Asia-Pacific standards, obesity is defined by a BMI  $> 25$  kg/m<sup>2</sup> [4]. Obesity is influenced by excessive energy intake from fat and low physical activity. Furthermore, individuals with reduced fat taste sensitivity may have poorer diet quality, contributing to increased risk of obesity [5]. Wang et al. also found that a high-fat diet increases the likelihood of obesity [6].

One of the factors influencing fat consumption is the fatty acid perception threshold, which affects preference and satiety [7,8]. Humans can detect free fatty acids independently of food aroma, texture, or visual cues, suggesting a specific sensory mechanism [9,10]. These mechanisms involve fatty acid receptors such as CD36 and GPR120. CD36 is a multifunctional membrane glycoprotein expressed in taste bud cells and in metabolic tissues, where it facilitates fatty acid uptake and signaling [11]. In the oral cavity, CD36 contributes to the detection of long-chain fatty acids, linking molecular receptor activity to behavioral dietary choices [12]. The CD36 protein is encoded by the CD36 gene, with several known variants, among which SNP rs1761667 has been most widely studied because the A allele is associated with reduced expression through altered transcription factor binding affinity [11]. This reduced expression has been linked to higher fatty acid perception thresholds and altered fat intake in several populations, including those in Southeast Asia [13]. Although other polymorphisms such as rs1527483 have been investigated, rs1761667 is considered more relevant due to its functional role in oral fat sensitivity and its relatively higher frequency across populations [14].

Despite increasing evidence on the role of *cd36* genetic variation in fat taste sensitivity, findings remain inconsistent across populations, and limited evidence is available from Indonesian cohorts. In addition, gene–environment interactions may play a significant role in shaping dietary behavior and obesity risk. Office workers represent a relevant study population, as this demographic is characterized by sedentary behavior and distinct dietary patterns, making them particularly prone to weight gain and metabolic risk. To address these gaps, the present study was designed to examine the association between

the *cd36* rs1761667 genotype, fatty acid perception threshold, body composition, physical activity, and dietary intake among Indonesian office workers. We hypothesized that (1) the A allele of *cd36* rs1761667 would be associated with a higher fatty acid perception threshold, and (2) this reduced sensitivity would correlate with higher fat intake and adiposity.

## **METHODS**

### **Research Subjects**

This study involved 87 adult office workers (53 females and 34 males) from Jakarta and Bogor, recruited from corporate office environments. Inclusion criteria required participants to be generally healthy, free from lactose intolerance, not undergoing dietary interventions, and not taking any medications. Individuals who smoked or were pregnant were excluded. All participants were fully informed of the research procedures and provided written informed consent before participation. As this was a pilot study in the Indonesian population, the sample size was not determined using a priori power analysis.

### **Sample Collection**

Saliva was collected for genomic DNA extraction. Participants were instructed to stimulate saliva production by rubbing the inner cheek with the tongue. A minimum of 2 microtubes containing at least 1 mL of saliva each (total ~2 mL) were collected per subject using sterile 1.5 mL microtubes.

### **Genomic DNA Isolation**

DNA extraction was performed using a modified Alkaline Lysis Method [15], adapted to saliva samples and standardized laboratory protocols. A 500  $\mu$ L volume of saliva was mixed with 12.5  $\mu$ L of SDS and 40  $\mu$ L of proteinase K in a 1.5 mL microtube, vortexed briefly, and incubated at 56°C for 30 minutes. DNA was extracted by adding 80  $\mu$ L of phenol:chloroform:isoamyl alcohol (25:24:1), then centrifuging at 12,000 rpm for 5 minutes. The aqueous phase was transferred to a new microtube, combined with 550  $\mu$ L of cold absolute ethanol, and incubated at 4°C for 5 minutes. Samples were centrifuged again at 12,000 rpm for 3 minutes to precipitate DNA. The resulting pellet was washed with 750  $\mu$ L of 70% ethanol, centrifuged, air-dried, and then resuspended in 40  $\mu$ L of distilled water before being stored at -20°C. DNA concentration and purity were assessed by spectrophotometry, and samples with a 260/280 ratio greater than 1.8 were considered of sufficient quality for genotyping.

### **Identification of CD36 rs1761667 Genotype**

Genotyping of *cd36* SNP rs1761667 was performed using the Tetraprimer Amplification Refractory Mutation System PCR (T-ARMS-PCR) method. This technique employed four primers: two outer primers and two allele-specific inner primers designed based on established sequences [16]. Amplification conditions were standardized for 35 cycles using a G-Storm GS00482 thermal cycler. PCR products were resolved on agarose gel electrophoresis in Tris-acetate-EDTA (TAE) buffer, stained with ethidium bromide, and visualized under UV light. The resulting band patterns indicated genotypes: 496 bp and 341 bp for GG, 496 bp and 155 bp for AA, and all three bands for GA. Genotyping was successfully conducted for all 87 participants, yielding a 100% success rate with no dropout cases. Genotypes were grouped into two categories for analysis: GG (More Sensitive) and GA/AA (Less Sensitive). This grouping was based on prior functional evidence showing that the presence of the A allele reduces *cd36* expression through altered transcription factor binding affinity, which has been associated with decreased sensitivity to fatty acid perception [12].

### **Fatty Acid Perception Threshold Testing**

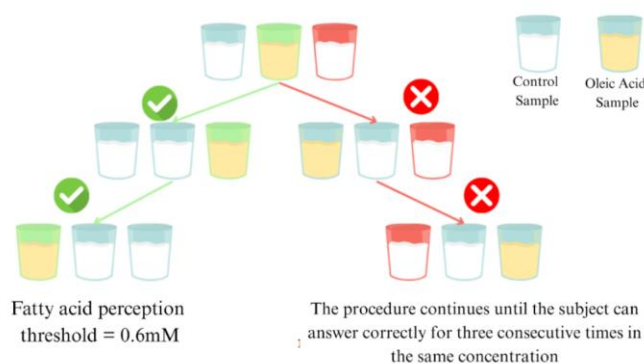
Sensory assessment of fatty acid perception threshold was conducted following an adapted method [17]. Skim milk was used as a base matrix and combined with liquid paraffin, gum arabic, and EDTA to ensure consistent viscosity and prevent oxidation. Oleic acid was added at increasing concentrations (0.6 mM, 2 mM, 3.8 mM, and 5 mM) to create test samples. The test followed a triangle test format using an

ascending forced-choice (AFC) procedure, with participants asked to identify the different sample from sets of three. The lowest concentration at which participants consistently identified the fatty sample was recorded as their threshold.

**Table 1. Composition of Fatty Acid Perception Threshold Test Sample.**

Material	Control	0.6 mM	2 mM	3.8 mM	5 mM
Skim Milk	100 mL	100 mL	100 mL	100 mL	100 mL
Liquid paraffin	0.5 gram	0.5 gram	0.5 gram	0.5 gram	0.5 gram
Gum Arabic	0.5 gram	0.5 gram	0.5 gram	0.5 gram	0.5 gram
EDTA	0.01 gram	0.01 gram	0.01 gram	0.01 gram	0.01 gram
Oleic Acid	-	17 $\mu$ L	63.1 $\mu$ L	119.9 $\mu$ L	157.8 $\mu$ L

Fatty acid perception threshold was determined by conducting a sensory test using the Ascending Forced Choice (AFC) method. This method was done by providing samples to taste from the lowest concentration and gradually increase the concentration until the subject correctly identifies the treatment sample or the highest concentration (5 mM) is reached. The sensory panel was asked 1 different sample among the 3 presented samples, also known as the Triangle Test (TAT). In each set, 3 of 10 mL samples were presented consisting of 2 control and 1 treatment sample with a random 3-number code. The sensory test ends when the correct sample was selected in three consecutive trials at the same concentration level, which is then determined as the threshold value for fatty acid perception. If unable to determine a different sample, then a sample with an increased concentration will be given until panels were able to correctly answer. The position of the sample placement is randomized to avoid bias. During the test, panels were asked to cover the nose to avoid being affected by the aroma factor (Figure 1). All sensory panelists were trained and calibrated annually using the five basic taste threshold method and difference tests, including the triangle test, to ensure inter-rater reliability.



**Figure 1. Fatty acid perception threshold test sample presentation procedure.**

### Bioelectrical Impedance Analysis (BIA)

The body mass composition was measured by the Bioelectrical Impedance Analysis (BIA) using the InBody 230®. The body mass composition consisted of Body Mass Index (BMI), body fat percentage, and Waist-to-Hip Ratio (WHR). Respondents were asked not to consume foods for 2 hours and not consume caffeine within 24 hours prior to the measurement.

### Dietary Pattern: 24-Hour Food Recall

The dietary pattern was examined using the online questionnaire "Dietary Pattern: 24-Hour Food Recall" given to the respondent. Respondents were asked to describe the food consumed by respondents, starting from breakfast, lunch, dinner, morning snack and afternoon snack along with the quantity, composition, cooking method, and the brand. It is recommended to enter the quantity in grams, but for the convenience of respondents, it is allowed to use other units, such as plates, spoons, or other units. The questionnaire was filled in for 3 days, consisting of 2 weekdays and 1 weekend day. Dietary intake was

recorded using a three-day 24-hour food recall (two weekdays and one weekend), considered sufficient to describe habitual intake patterns [18]. Data from the questionnaire were analyzed using both NutriBase and FatSecret applications. NutriBase provided detailed nutrient breakdown based on international food databases, while FatSecret offered broader coverage of Indonesian food items. Results from the two platforms were cross-checked to improve accuracy and minimize bias from database limitations.

### Physical Activity Level Measurement

Physical activity level was assessed to describe the activity patterns of the respondents. Participants recorded their physical activities for 7 consecutive days using a modified version of the International Physical Activity Questionnaire (IPAQ, 2002). This questionnaire captured the type and duration of physical activities performed, excluding sedentary sitting time.

### Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 25. Group comparisons between independent genotype groups (GG vs GA/AA) were performed using the Mann–Whitney U test for non-normally distributed data. The correlation between the *cd36* SNP rs1761667 genetic profile and lifestyle factors with fatty acid perception threshold was assessed using Spearman’s rho test. A two-tailed significance level of  $p < 0.05$  was applied for all analyses. For descriptive statistics, mean  $\pm$  SD and 95% confidence intervals (CI) were reported. CI for group means were calculated using the Student’s *t*-distribution. All tests were two-tailed and  $p < 0.05$  was considered significant.

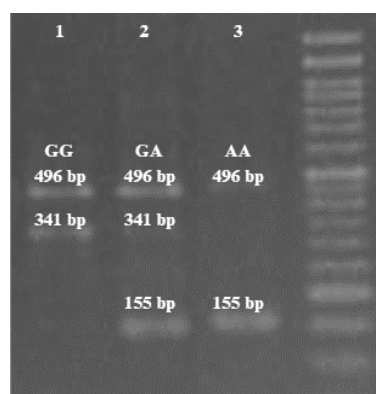
### CODE OF HEALTH ETHICS

Ethical approval for this research was granted by the Ethics Development Center of Atma Jaya Catholic University under license letter number 0001R/III/PPPE.PM.10.05/3/2024.

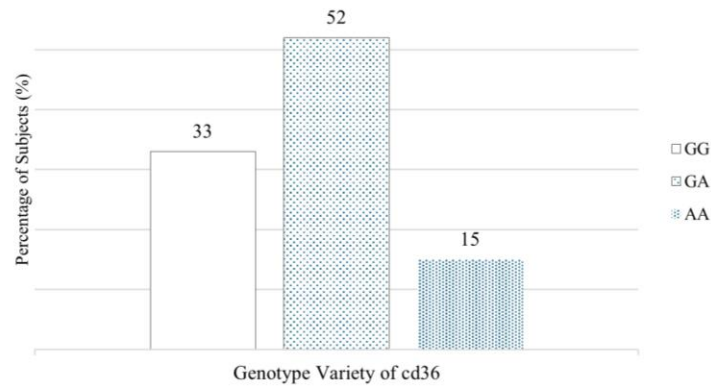
## RESULTS

### Genetic Profile of *CD36* Gene SNP rs1761667

The genotyping of *CD36* SNP rs1761667 was conducted using the T-ARMS-PCR method with allele-specific primers designed according to validated sequences [16]. PCR amplification products were visualized using agarose gel electrophoresis and documented under UV illumination (Figure 3). The GG genotype was identified by the presence of 496 bp and 341 bp DNA fragments (Figure 2), while the AA genotype showed bands at 496 bp and 155 bp. Heterozygous GA genotypes displayed all three bands: 496 bp, 341 bp, and 155 bp. Out of the 87 participants, 33% exhibited the GG genotype, 52% had the GA genotype, and 15% carried the AA genotype, indicating that the GA genotype was the most prevalent within the study population.



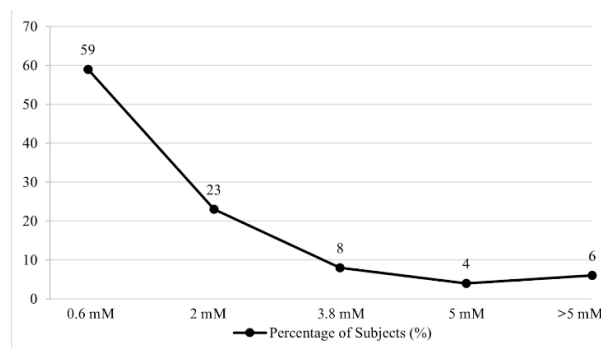
**Figure 2. T-ARMS-PCR visualization of *cd36* rs1761667 allele detection.**



**Figure 3. Distribution of CD36 gene genetic profile SNPs rs1761667.**

### Distribution of Fatty Acid Perception Thresholds

The fatty acid perception threshold was determined based on the lowest oleic acid concentration correctly identified three consecutive times in the sensory test. The higher the threshold concentration detected, the lower the taste sensitivity to fatty acid perception. About 59% of the population had a fatty acid perception threshold of 0.6 mM. While, 23% of the respondents have a fat fatty acid perception threshold at 2 mM. Only 8% of respondents had a threshold at 3.8 mM, while another 4% could detect oleic acid only at a concentration of 5 mM. The remaining 6% could not answer correctly at all four concentrations. The proportion of respondents able to detect fatty acids decreased progressively with increasing threshold concentrations, indicating that a greater number of individuals exhibited high sensitivity to fat perception (Figure 4). A floor effect was observed, as most participants detected fatty acids at the lowest concentration. This may differ from previous studies and could reflect the habitual consumption of oily foods in the Indonesian population, leading to greater familiarity with fatty taste sensations.



**Figure 4. Distribution of fatty acid perception thresholds (N=87)**

### Dietary Pattern Based on 24-Hour Food Recall

The dietary pattern analysis revealed the average daily consumption patterns of the respondents, summarized in Table 2. The data include total calorie intake, macronutrient distribution, and key nutrient components such as sugar, salt, protein, fat, and fiber. To provide additional context, sex-specific dietary distribution is presented in Table 3, allowing comparison of macronutrient intake between men and women.

**Table 2. Dietary Pattern per Day.**

	Nutrient Intake (N=87)	95% CI
Calorie Intake		
Total Calories (kcal)	1,584 ± 340	[1,512 – 1,657]
Percentage of Calories from Carbohydrates (%)	50 ± 6	[48.7 – 51.3]
Percentage of Calories from Fat (%)	31 ± 6	[29.7 – 32.3]
Nutrient Intake (gram)		
Sugar	30.7 ± 14.8	[27.5 – 33.9]
Salt	2.6 ± 1.2	[2.34 – 2.86]

	Nutrient Intake (N=87)	95% CI
Fat	53.5 ± 14.3	[50.5 - 56.6]
Protein	69.2 ± 19.6	[65.0 - 73.4]
Fiber	10.0 ± 4.7	[9.00 - 11.00]

All data are presented as Mean ± Standard Deviation (95% CI).

Participants' average daily caloric intake was below the recommended requirement for Indonesian adults, at 1,584 kcal/day. Carbohydrates contributed the largest share of energy, followed by fat and protein. Fat intake accounted for 31% of energy, exceeding the national guideline of 20–25%, while carbohydrate and fiber intake were below recommendations, suggesting an imbalance in dietary composition (Table 2).

**Table 3. Dietary Distribution of Women and Men.**

	Nutrient Intake (N=87)		Male (N=34)	95% CI	<i>p</i> -value (Sig.)
	Women (N=53)	95% CI			
Calorie Intake					
Total Calories (kcal)	1,501 ± 308	[1,416 - 1,586]	1,713 ± 352	[1,590 - 1,836]	0.009*
Percentage of Calories from Carbohydrates (%)	49 ± 6	[47.4 - 50.6]	52 ± 6	[49.9 - 54.1]	0.064
Percentage of Calories from Fat (%)	32 ± 5	[30.6 - 33.4]	29 ± 6	[26.9 - 31.1]	0.004*
Nutrient Intake (gram)					
Sugar	31.7 ± 15.1	[27.5 - 35.9]	29.0 ± 14.4	[24.0 - 34.0]	0.451
Salt	2.3 ± 0.8	[2.08 - 2.52]	3.0 ± 1.4	[2.51 - 3.49]	0.032*
Fat	52.5 ± 12.8	[49.0 - 56.0]	55.0 ± 16.4	[49.3 - 60.7]	0.542
Protein	64.8 ± 16.6	[60.2 - 69.4]	76.2 ± 22.1	[68.5 - 83.9]	0.016*
Fiber	10.1 ± 5.0	[8.7 - 11.5]	9.9 ± 4.3	[8.4 - 11.4]	0.969

All data are presented as Mean ± Standard Deviation (95% CI). Group comparisons were analyzed using the Mann-Whitney U test. \*Significant at 0.05 standard.

Gender-based differences were observed in dietary distribution: women derived a higher proportion of energy from protein, whereas men consumed a greater proportion from carbohydrates. Fat contributed a comparable proportion of total energy in both groups, indicating distinct macronutrient preferences that may relate to differences in body composition (Table 3).

### Comparison of Fatty Acid Perception Threshold and Lifestyle Characteristics by CD36 Genetic Profile

The presence of allele A decreases the expression of the *cd36* SNPs rs1761667 gene [12]. The subjects were divided into 2 groups, sensitive and less sensitive. The sensitive group is a group that has "GG" allele and not allele A. The less sensitive group is the one with allele A (alleles "GA" and "AA"). A comparison of average values between CD36 genetic profile groups was conducted to evaluate differences in fatty acid perception threshold and lifestyle-related variables, as presented in Table 4.

**Table 4. Comparison of Fatty Acid Perception Threshold and Lifestyle Variables by CD36 Genetic Profile.**

	Genetic Profile (N=87)		Less Sensitive (N=58)	95% CI	<i>p</i> -value (Sig.)
	Sensitive (N=29)	95% CI			
Fatty acid perception threshold (mM)	2.0 ± 1.76	[1.33 - 2.67]	1.54 ± 1.56	[1.13 - 1.95]	0.167
Physical Activity Score (METs)	1,728.45 ± 1,524.35	[1,148 - 2,309]	1,378.79 ± 1,086.86	[1,093 - 1,664]	0.74
Body Composition					
Body Mass Index (BMI) (kg/m <sup>2</sup> )	23.11 ± 23.91	[14.02 - 32.20]	23.91 ± 4.17	[22.81 - 25.01]	0.482
Body Fat Percentage (%)	27.88 ± 10.12	[24.03 - 31.73]	30.49 ± 9.35	[28.03 - 32.95]	0.292
Waist-to-Hip Ratio (WHR)	0.85 ± 0.05	[0.831 - 0.869]	0.86 ± 0.05	[0.847 - 0.873]	0.419
Calorie Intake					
Total Calories (kcal)	1,580 ± 357	[1,444 - 1,716]	1,586 ± 335	[1,498 - 1,674]	0.893

Percentage of Calories from Carbohydrates (%)	52 ± 6	[49.7 – 54.3]	50 ± 6	[48.4 – 51.6]	0.059
Percentage of Calories from Fat (%)	31 ± 5	[29.1 – 32.9]	31 ± 6	[29.4 – 32.6]	0.446
Nutrient Intake (gram)					
Sugar	31.8 ± 11.6	[27.39 – 36.22]	30.1 ± 16.2	[25.84 – 34.36]	0.438
Salt	2.5 ± 0.8	[2.20 – 2.80]	2.6 ± 1.3	[2.26 – 2.94]	0.854
Fat	52.1 ± 15.8	[46.1 – 58.1]	54.1 ± 13.6	[50.5 – 57.7]	0.36
Protein	66.1 ± 20.5	[58.3 – 73.9]	70.8 ± 19.2	[65.8 – 75.9]	0.163
Fiber	10.1 ± 4.6	[8.35 – 11.85]	10.0 ± 4.8	[8.74 – 11.26]	0.55

All data are presented as Mean ± Standard Deviation (95% CI). Group comparisons were analyzed using the Mann–Whitney U test. \*Significant at 0.05 standard.

No significant differences were observed in fatty acid perception thresholds or in lifestyle-related variables across the *cd36* genotype groups. For example, the mean perception threshold was 2.4 mM in GG carriers compared to 2.5 mM in GA/AA carriers ( $p > 0.05$ ), and average physical activity scores were similar between groups. These results indicate that, within this cohort, the presence of the A allele did not correspond to measurable differences in taste sensitivity or lifestyle patterns (Table 4).

### Comparison of Fatty Acid Perception Threshold and Related Variables by *cd36* Genetic Profile within Gender Groups

Women and men have different body composition standards, women tend to have higher fat mass than men. Therefore, a comparison of body composition between genetic profiles within the same sex was conducted. Comparative analysis of *CD36 rs1761667* genetic profile groups in women subjects is presented in Table 5, while results for male subjects are shown in Table 6.

**Table 5. Comparative analysis of *cd36 rs1761667* genetic profile groups in women.**

	Genetic Profile				<i>p</i> -value ( <i>Sig.</i> )
	Women (N=53)				
	<i>Sensitive</i> (N=18)	95% CI	<i>Less Sensitive</i> (N=35)	95% CI	
Fatty acid perception threshold (mM)	1.89 ± 1.70	[1.11 – 2.67]	1.56 ± 1.70	[1.02 – 2.10]	0.314
Body Composition					
Body Fat Percentage (%)	34.42 ± 5.94	[31.47 – 37.37]	36.19 ± 6.15	[34.08 – 38.30]	0.329
Waist-to-Hip Ratio (WHR)	0.85 ± 0.05	[0.83 – 0.87]	0.86 ± 0.05	[0.84 – 0.88]	0.51
Body Mass Index (BMI) (kg/m <sup>2</sup> )	23.49 ± 3.19	[22.08 – 24.90]	24.16 ± 4.44	[22.78 – 25.54]	0.771
Physical Activity Score (METs)	1,334.17 ± 1,191.94	[743 – 1,925]	1,470.43 ± 1,287.73	[1,023 – 1,918]	0.714
Calorie Intake					
Total Calories (kcal)	1,502 ± 314	[1,346 – 1,658]	1,500 ± 310	[1,394 – 1,606]	0.925
Percentage of Calories from Carbohydrates (%)	52 ± 6	[49.0 – 55.0]	48 ± 6	[46.0 – 50.1]	0.013*
Percentage of Calories from Fat (%)	32 ± 4	[30.0 – 34.0]	33 ± 5	[31.1 – 34.9]	0.236
Nutrient Intake (gram)					
Sugar	32.1 ± 11.7	[52.1 – 64.1]	31.6 ± 16.7	[62.1 – 74.3]	0.844
Salt	2.4 ± 0.7	[2.06 – 2.74]	2.3 ± 0.9	[2.40 – 3.00]	0.666
Fat	50.7 ± 12.0	[44.6 – 56.8]	53.4 ± 13.3	[48.8 – 58.0]	0.493
Protein	58.1 ± 12.0	[52.0 – 64.2]	68.2 ± 17.7	[62.1 – 74.3]	0.039*
Fiber	10.8 ± 4.5	[8.56 – 13.04]	9.7 ± 5.3	[7.89 – 11.51]	0.072

All data are presented as Mean ± Standard Deviation (95% CI). Group comparisons were analyzed using the Mann–Whitney U test. \*Significant at 0.05 standard.

Among male participants, a key difference was observed in body fat percentage, where A allele carriers (GA/AA) showed higher adiposity than their GG counterparts (Table 6).

**Table 6. Comparative analysis of *cd36* rs1761667 genetic profile groups in men.**

	Genetic Profile				
	Male (N=34)				
	<i>Sensitive</i> (N=11)	95% CI	<i>Less Sensitive</i> (N=23)	95% CI	<i>p-value</i> ( <i>Sig.</i> )
Fatty acid perception threshold (mM)	2.16 ± 1.94	[1.28 – 3.04]	1.51 ± 1.36	[1.01 – 2.01]	0.327
Body Composition					
Body Fat Percentage (%)	17.18 ± 4.91	[13.88 – 20.48]	21.81 ± 6.16	[19.15 – 24.47]	0.034*
Waist-to-Hip Ratio (WHR)	0.85 ± 0.04	[0.82 – 0.88]	0.86 ± 0.05	[0.84 – 0.88]	0.698
Body Mass Index (BMI) (kg/m <sup>2</sup> )	22.48 ± 2.68	[21.02 – 23.94]	23.53 ± 3.78	[22.01 – 25.05]	0.397
Physical Activity Score (METs)	2,373.64 ± 1,831.18	[1,437 – 1,977]	1,239.35 ± 681.25	[1,571 – 1,861]	0.118
Calorie Intake					
Total Calories (kcal)	1,707 ± 401	[47.0 – 55.0]	1,716 ± 336	[49.4 – 54.6]	0.84
Percentage of Calories from Carbohydrates (%)	51 ± 6	[46.9 – 55.1]	52 ± 6	[49.5 – 54.5]	0.971
Percentage of Calories from Fat (%)	29 ± 5	[25.7 – 32.3]	29 ± 8	[25.5 – 32.5]	0.912
Nutrient Intake (gram)					
Sugar	31.3 ± 11.9	[23.6 – 39.0]	28.0 ± 15.6	[21.4 – 34.6]	0.461
Salt	2.8 ± 1.0	[2.1 – 3.5]	3.1 ± 1.6	[2.4 – 3.8]	0.811
Fat	54.5 ± 21.1	[41.4 – 67.6]	55.2 ± 14.2	[49.2 – 61.2]	0.519
Protein	79.2 ± 25.0	[53.5 – 86.9]	74.7 ± 21.0	[65.6 – 82.4]	0.645
Fiber	8.8 ± 4.6	[5.8 – 11.8]	10.4 ± 4.2	[8.6 – 12.2]	0.292

All data are presented as Mean ± Standard Deviation (95% CI). Group comparisons were analyzed using the Mann–Whitney U test. \*Significant at 0.05 standard.

Based on the comparative analysis, significant differences were observed in certain variables between women and men subjects, as shown in (Table 5)(Table 6). In female subjects (Table 5), significant differences were found in the percentage of calorie intake from carbohydrates and protein intake. Women with the GG allele consumed a higher percentage of calories from carbohydrates ( $p = 0.013$ ), while those with the A allele (GA or AA) had higher protein intake ( $p = 0.039$ ). Other variables such as BMI, body fat percentage, and physical activity showed no significant differences between genetic profiles in female subjects.

In male subjects (Table 6), a significant difference was observed in body fat percentage between genetic profiles. Individuals carrying the A allele (GA or AA) had a higher body fat percentage compared to those with the GG allele ( $p = 0.034$ ). No other significant differences were found in BMI, physical activity, or dietary intake between genetic profiles in men. However, this finding should be interpreted with caution given the relatively small number of men in the GA/AA group ( $n = 23$ ), which may limit the robustness and generalizability of the result.

### Comparison of Fatty Acid Perception Threshold and Body Composition by *cd36* Genetic Profile across BMI Categories

Subjects were grouped into normal weight, overweight, and obese categories based on their BMI, as previously defined in the Methods section. The standards used are normal weight:  $< 23 \text{ kg/m}^2$ , overweight:  $23 - 24.9 \text{ kg/m}^2$ , and obesity:  $> 25 \text{ kg/m}^2$ . In normal weight subjects (Table 7), no significant differences were found between *cd36* genetic profiles in fatty acid perception threshold, body composition, physical activity, or dietary intake.

In overweight subjects (Table 8), a significant difference was observed in the percentage of calorie intake from carbohydrates. Respondents with the GG allele had a higher carbohydrate intake compared to those with the A allele ( $p = 0.026$ ). Other variables showed no significant differences between genetic profiles in this group. In obese subjects (Table 9), no significant differences were found between *cd36* genetic profiles in fatty acid perception threshold, body composition, physical activity, or dietary intake. This loss of significance may be partly explained by a dietary shift in obese participants, whose intake patterns showed relatively higher fat and protein proportions, thereby diluting the contribution of carbohydrates to total energy intake and masking the genotype-related difference observed in the

overweight group.

**Table 7. Comparison of Fatty Acid Perception Threshold & Body Composition by CD36 Genetic Profile in Normal Weight Subjects.**

	Genetic Profile				
	<i>Normal Weight (N=41)</i>				
	<i>Sensitive (N=18)</i>	95% CI	<i>Less Sensitive (N=35)</i>	95% CI	<i>p-value (Sig.)</i>
Fatty acid perception threshold (mM)	2.05 ± 1.73	[1.19 – 2.91]	2.01 ± 2.01	[1.32 – 2.70]	0.702
Body Composition					
Body Fat Percentage (%)	25.56 ± 8.64	[21.26 – 29.86]	25.60 ± 8.33	[22.74 – 28.46]	0.843
Waist-to-Hip Ratio (WHR)	0.81 ± 0.03	[0.80 – 0.82]	0.82 ± 0.03	[0.81 – 0.83]	0.147
Physical Activity Score (METs)	1,941.86 ± 1,774.34	[1059.50 – 2824.22]	1,237.40 ± 853.77	[944.12 – 1530.68]	0.517
Calorie Intake					
Total Calories (kcal)	1,594 ± 358	[1415.97 – 1772.03]	1,617 ± 312	[1509.82 – 1724.18]	0.682
Percentage of Calories from Carbohydrates	51 ± 7	[47.59 – 54.41]	49 ± 7	[46.57 – 51.43]	0.207
Percentage of Calories from Fat (%)	31 ± 5	[28.55 – 33.45]	63 ± 5	[61.28 – 64.72]	0.125
Nutrient Intake (gram)					
Sugar	32.1 ± 14.5	[25.09 – 39.11]	31.7 ± 17.9	[25.59 – 37.81]	0.781
Salt	2.5 ± 0.8	[2.11 – 2.89]	2.9 ± 1.6	[2.36 – 3.44]	0.843
Fat	53.0 ± 17.1	[44.50 – 61.50]	56.6 ± 13.2	[52.07 – 61.13]	0.207
Protein	61.5 ± 13.5	[54.97 – 68.03]	69.5 ± 26.4	[60.27 – 78.73]	0.947
Fiber	10.0 ± 2.2	[9.10 – 11.30]	11.3 ± 4.4	[9.72 – 12.88]	0.682

All data are presented as Mean ± Standard Deviation (95% CI). Statistical comparison was performed using the Mann-Whitney U test. \*Significant at 0.05 standard.

**Table 8. Comparison of Fatty Acid Perception Threshold & Body Composition by CD36 Genetic Profile in Overweight Subjects.**

	Genetic Profile				
	<i>Overweight (N=22)</i>				
	<i>Sensitive (N=18)</i>	95% CI	<i>Less Sensitive (N=35)</i>	95% CI	<i>p-value (Sig.)</i>
Fatty acid perception threshold (mM)	1.45 ± 1.23	[0.84 – 2.06]	1.00 ± 0.92	[0.68 – 1.32]	0.407
Body Composition					
Body Fat Percentage (%)	25.56 ± 10.39	[20.39 – 30.73]	31.23 ± 8.42	[28.34 – 34.12]	0.237
Waist-to-Hip Ratio (WHR)	0.86 ± 0.02	[0.85 – 0.87]	0.87 ± 0.01	[0.87 – 0.87]	0.837
Physical Activity Score (METs)	1,667.86 ± 1,592.59	[875.88 – 2459.84]	1,273.33 ± 770.00	[1008.83 – 1537.83]	0.891
Calorie Intake					
Total Calories (kcal)	1,641 ± 402	[1441.09 – 1840.91]	1,558 ± 340	[1441.21 – 1674.79]	0.447
Percentage of Calories from Carbohydrates	54 ± 7	[50.54 – 57.46]	48 ± 4	[46.64 – 49.36]	0.026*
Percentage of Calories from Fat (%)	30 ± 5	[27.57 – 32.43]	30 ± 9	[26.95 – 33.05]	0.783
Nutrient Intake (gram)					
Sugar	34.8 ± 9.3	[30.06 – 39.54]	27.9 ± 14.0	[23.11 – 32.69]	0.237
Salt	2.2 ± 0.69	[1.86 – 2.54]	2.6 ± 1.1	[2.22 – 2.98]	0.407
Fat	49.3 ± 11.3	[43.70 – 54.90]	51.4 ± 14.3	[46.49 – 56.31]	0.680
Protein	64.0 ± 22.6	[52.47 – 75.53]	73.4 ± 16.7	[67.73 – 79.07]	0.162
Fiber	10.3 ± 7.0	[6.88 – 13.72]	9.8 ± 6.9	[7.41 – 12.19]	0.731

All data are presented as Mean ± Standard Deviation (95% CI). Statistical comparison was performed using the Mann-Whitney U test. \*Significant at 0.05 standard.

**Table 9. Comparison of Fatty Acid Perception Threshold and Body Composition by CD36 Genetic Profile in Obese Subjects**

	Genetic Profile				
	Obese (N=24)				
	<i>Sensitive</i> (N=6)	95% CI	<i>Less Sensitive</i> (N=18)	95% CI	<i>p-value</i> ( <i>Sig.</i> )
Fatty acid perception threshold (mM)	2.00 ± 2.23	[0.34 – 4.34]	1.43 ± 1.13	[0.87 – 1.99]	0.890
Body Composition					
Body Fat Percentage (%)	35.31 ± 8.89	[25.98 – 44.64]	37.37 ± 8.02	[33.38 – 41.36]	0.646
Waist-to-Hip Ratio (WHR)	0.86 ± 0.02	[0.84 – 0.88]	0.87 ± 0.01	[0.87 – 0.87]	0.132
Physical Activity Score (METs)	1,116.25 ± 368.66	[729.37 – 1503.13]	1,774.33 ± 1,578.51	[989.36 – 2559.30]	0.500
Calorie Intake					
Total Calories (kcal)	1,641 ± 402	[1219.13 – 2062.87]	1,577 ± 399	[1378.58 – 1775.42]	0.903
Percentage of Calories from Carbohydrates	51 ± 4	[46.80 – 55.20]	51 ± 6	[48.02 – 53.98]	0.559
Percentage of Calories from Fat	32 ± 5	[26.75 – 37.25]	31 ± 9	[26.52 – 35.48]	0.518
Nutrient Intake (gram)					
Sugar	34.75 ± 9.3	[24.99 – 44.51]	26.6 ± 14.0	[19.64 – 33.56]	0.177
Salt	2.8 ± 1.1	[1.65 – 3.95]	2.3 ± 0.7	[1.95 – 2.65]	0.270
Fat	52.1 ± 16.9	[34.36 – 69.84]	53.8 ± 14.2	[52.23 – 64.57]	0.710
Protein	64.0 ± 22.6	[40.28 – 87.72]	73.4 ± 16.7	[65.10 – 81.70]	0.602
Fiber	10.3 ± 7.0	[2.95 – 17.65]	9.8 ± 6.9	[6.37 – 13.23]	0.781

All data are presented as Mean ± Standard Deviation (95% CI). Statistical comparison was performed using the Mann-Whitney U test. \*Significant at 0.05 standard.

**Correlation between *cd36* Genetic Profile, Fatty Acid Perception Threshold, Body Fat Composition, Physical Activity, and Dietary Intake**

The Spearman rho correlation analysis showed no significant correlations between *cd36* genetic profile, fatty acid perception threshold, body composition, physical activity, and dietary intake, as presented in (Table 10)(Table 11). The correlation coefficients were consistently low ( $r < 0.2$ ), indicating very weak and non-meaningful relationships. There was no correlation between fatty acid perception threshold with physical activity, BMI, body fat composition, and WHR. Only a small but not significant positive trend of fatty acid perception and physical activity level in all respondents.

**Table 10. Correlation of Fatty Acid Perception Threshold, Physical Activity Level, and Body Composition.**

		Correlation			
		Physical Activity Level	Body Mass Index (BMI) Value	Fat Composition	Waist-to-Hip Ratio (WHR)
Fatty acid perception threshold	Correlation Coefficient	0.071	-0.125	-0.178	-0.202
	<i>p-value</i> ( <i>Sig.</i> )	0.516	0.247	0.099	0.06

**Table 11. Correlation between Fatty Acid Perception Threshold and Dietary Pattern.**

		Correlation							
		Calories	Carbohydrate Calories	Fat Calories	Sugar Intake	Salt Intake	Fat Intake	Protein	Fiber
Fatty acid Perception Threshold	Correlation Coefficient	0.127	0.066	0.015	-0.032	0.007	0.081	0.08	-0.113
	<i>p-value</i> ( <i>Sig.</i> )	0.24	0.542	0.892	0.771	0.949	0.458	0.461	0.297

The correlation analysis between fatty acid perception threshold and dietary pattern showed no statistically significant associations. Although a weak positive correlation was observed between fatty acid perception threshold and protein intake ( $r = 0.24$ ), this relationship did not reach significance. Other dietary components, including total calorie intake, carbohydrate, fat, sugar, salt, and fiber, also

demonstrated very weak correlations ( $r < 0.2$ ) with the perception threshold. These findings suggest that dietary pattern, particularly macronutrient distribution, may not be directly associated with fatty acid perception threshold in this sample population.

## DISCUSSION

This study explored the association between the *cd36* rs1761667 genetic profile—a polymorphism known to encode a fatty acid receptor and transporter—and fatty acid perception threshold, as well as its relationship with body composition, physical activity, and dietary intake among adult office workers. The genotype distribution (GG: 33%, GA: 52%, AA: 15%) resembled that of African American populations [19] rather than previous findings in Indonesia [20]. This difference may reflect the highly mixed population structure in Jakarta and Bogor, where migration and intermarriage contribute to genetic heterogeneity, as well as potential environmental influences that shape allele frequencies. This observation aligns with studies in Malaysia reporting regional polymorphism differences across Southeast Asia [3].

Although the A allele has been associated with lower CD36 protein expression and reduced sensitivity to fatty acid taste, this study found no significant differences in fatty acid perception thresholds between the GG genotype and the GA or AA genotypes. These findings contrast with previous evidence indicating higher sensitivity in GG individuals [21], but are consistent with other studies reporting no significant difference in fatty acid sensitivity between obese and non-obese individuals [22]. The absence of a significant association between genotype and fatty acid perception threshold in this cohort may be explained by several factors: (1) the relatively modest sample size ( $N=87$ ) may have been underpowered to detect subtle effects; (2) other genetic variants (e.g., rs1527483) or unmeasured environmental influences such as early-life nutrition may override the effect of a single SNP; and (3) the sensory test employed only oleic acid, whereas sensitivity to other fatty acids may differ.

Gender-specific patterns were observed in dietary intake. Among women, those with the GA or AA genotypes consumed significantly more protein, while GG carriers had a higher proportion of energy derived from carbohydrates. These patterns are in line with prior reports suggesting that individuals with lower fat sensitivity may prefer high-protein and high-fat foods, particularly from animal sources [23,17]. In male participants, individuals with the A allele exhibited significantly higher body fat percentages compared to GG carriers, despite having similar caloric intake and physical activity levels. One possible explanation is that CD36, beyond its role in oral sensation, also functions as a fatty acid translocase in muscle and adipose tissue. Variants such as rs1761667 may alter fatty acid partitioning and storage efficiency, thereby influencing adiposity independently of dietary intake [21]. This suggests a possible metabolic mechanism underlying fat accumulation that may be independent of energy intake, potentially linked to CD36's role in fatty acid uptake and partitioning in adipose tissue and muscle as reported in metabolomics and gene expression studies [21]. Gender-specific differences in hormonal regulation and fat storage could further contribute to these results [2].

When analyzed by body mass index (BMI), a significant difference in carbohydrate intake was detected in the overweight group. Individuals with the GG genotype consumed more calories from carbohydrates than those with the A allele. This trend was not present in the normal-weight or obese groups, which may reflect BMI-specific gene–diet interactions or metabolic differences [24]. Supporting this, previous studies found that the metabolic effects of *cd36* rs1761667 can vary between obese and non-obese individuals [25]. According to national dietary guidelines [26], the recommended fat contribution to total daily energy intake for Indonesian adults is 20–25%. While the average fat intake in this study slightly exceeded this range (31%), total caloric intake (1,584 kcal/day) remained below standard adult requirements. We hypothesize that the higher fat percentage may reflect a redistribution of macronutrients rather than overconsumption, although this requires further validation.

Despite the lack of strong correlations between fatty acid perception threshold and lifestyle factors, prior research has indicated possible links between fat taste sensitivity and dietary quality [5]. The weak trends observed, particularly with protein intake and physical activity, may reflect subtle or complex effects, potentially moderated by genetic or behavioral variability. Such nuances may not be fully captured

in a cross-sectional design. Recent advances in nutrigenetics further emphasize the role of gene–metabolism interactions in long-term phenotypic outcomes [14]. The *cd36* rs1761667 polymorphism alone does not appear to fully explain individual differences in fat taste sensitivity or its physiological and behavioral consequences. Other factors, including additional genetic variants (e.g., rs1527483), tongue papillae density [21], and environmental influences, may play more prominent roles. In line with this, the rs1527483 variant has been shown to significantly impact oral fat perception [27], reinforcing the need to consider multiple genetic markers in future research.

This study has several limitations. First, the cross-sectional design precludes causal inference. Second, the modest sample size may have limited statistical power. Third, the dietary analysis relied on self-reported recall, which is subject to reporting bias. Finally, only oleic acid was tested as the stimulus for fatty acid perception; other fatty acids may yield different sensitivity patterns. Although the observed differences were not statistically significant, the reported confidence intervals provide a range of plausible values and suggest potential trends. These exploratory findings should be interpreted cautiously given the sample size and absence of multiple-testing correction. These limitations should be addressed in future studies with larger, more diverse cohorts and comprehensive sensory protocols.

## CONCLUSION

This study did not find significant overall associations between the *cd36* rs1761667 genetic profile and fatty acid perception threshold, physical activity, dietary intake, or body composition in a population of adult office workers. However, stratified analyses revealed that men with the GA or AA genotypes had a significantly higher body fat percentage compared to those with the GG genotype. Additionally, women and overweight individuals showed differences in macronutrient intake patterns that may be linked to variations in fat taste sensitivity. Taken together, these findings suggest that the *cd36* rs1761667 polymorphism may contribute modestly to obesity-related traits through gene–sex and gene–BMI interactions, rather than serving as a primary determinant of fat taste perception or body composition. Further research involving larger and more diverse populations is needed to examine other genetic variants, metabolic markers, and lifestyle factors to better understand the complex interactions between genetics, taste perception, and health outcomes.

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## CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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