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Association of MC4R Gene Variants with Body Fat, Diet, and Stress in Obese Young Adults: A Cross-Sectional Study

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KEYWORDS

Body composition, Dietary patterns, MC4R gene polymorphism, Obesity, Perceived stress

ABSTRACT:

Introduction: Melanocortin-4 receptor (MC4R) gene plays a central role in appetite regulation and energy homeostasis. However, limited evidence exists on interactions between MC4R gene-polymorphisms (rs12970134) and lifestyle factors in obese individuals.

Objectives: Analytical cross-sectional study investigated association of MC4R gene-polymorphism with body composition, dietary patterns, and perceived stress levels among obese young adults.

Methods: Total 60 obese individuals of both genders aged 18–25 years were enrolled. Participants were screened and categorized based on Asia-Pacific BMI classification. Anthropometric measurements and body composition were assessed using bioelectrical impedance analysis, dietary intake was evaluated using food frequency questionnaire, stress was measured using Perceived Stress Scale and MC4R genotyping was performed using Polymerase Chain Reaction - Restriction Fragment Length Polymorphism (PCR-RFLP).

Results: BMI was 34.37±5.88 kg/m², with mean body fat mass (BFM) of 42.08±12.88 kg and percent body fat (BF%) of 48.58±7.87%. Genotype allele distribution GG (46.7%), GA (41.7%) and AA (11.7%) was in Hardy-Weinberg equilibrium. AA genotype individuals showed higher BMI, BFM, and BF% compared to GG and GA genotypes. A significant negative correlation was observed between MC4R genotype and total fat intake (p=0.018), while no associations were found with BMI or BFM. About 70% participants reported moderate stress which correlated insignificantly with BMI and BFM.

Conclusions: MC4R gene-polymorphism in young obese individuals do not show association with anthropometric indicators. However, significant associations with dietary fat intake suggests the role of gene-polymorphism in appetite-regulation pathways thereby influencing food preferences. Large sample studies are needed to elucidate gene–environment interactions in obesity.

1. Introduction

Obesity has emerged as one of the most pressing global public health challenges of the twenty-first century, characterized by excessive accumulation of body fat that significantly increases the risk of metabolic and cardiovascular diseases. According to recent estimates by the World Health Organization, more than 650 million adults worldwide are classified as obese and the global prevalence has nearly tripled since 1975 [1]. This rapid increase has been attributed to shifts in dietary patterns, reduced physical activity and increasingly sedentary lifestyles [2,3]. In India, the prevalence of obesity among young adults has increased significantly due to rapid urbanization, dietary transitions, and sedentary lifestyles. The rising prevalence of obesity among young adults is particularly concerning, as early-onset adiposity is

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associated with an increased lifetime risk of chronic conditions such as type 2 diabetes mellitus, hypertension and cardiovascular diseases [3,4].

Although environmental and lifestyle factors are key drivers of obesity, substantial evidence suggests that genetic predisposition plays a critical role in determining individual susceptibility to weight gain and adiposity. Obesity is now recognized as a complex multifactorial disorder resulting from the interaction of genetic, behavioral and environmental influences [5]. Heritability studies have estimated that genetic factors account for approximately 40–70% of the inter-individual variation in body mass index (BMI) [6,7]. Advances in genome-wide association studies have identified several genes involved in appetite regulation and energy homeostasis, including FTO, LEP, LEPR, POMC and the melanocortin-4 receptor (MC4R) gene [8,9].

Among these, the MC4R gene has emerged as one of the most significant genetic determinants of body weight regulation. The MC4R gene encodes a G-protein-coupled receptor expressed in the hypothalamus and plays a central role in appetite regulation and energy balance via the leptin–melanocortin signaling pathway [10,11]. Activation of MC4R promotes satiety and increases energy expenditure, whereas reduced receptor function is associated with hyperphagia and increased adiposity [12]. Both rare mutations and common polymorphisms in the MC4R gene have been linked to obesity. In particular, the rs12970134 polymorphism has been widely studied and is associated with modest increased BMI, higher caloric intake, and greater susceptibility to obesity across diverse populations [13,14]. However, the magnitude of these associations is often modest and may vary across ethnic groups, suggesting that genetic predisposition alone does not fully explain obesity risk [14,15].

Emerging evidence highlights the importance of environmental and behavioral factors such as dietary patterns, physical activity and psychological stress in modulating genetic susceptibility to obesity. Diets rich in energy-dense, nutrient-poor foods contribute to positive energy balance and increased fat accumulation [16]. In contrast, physical activity plays a crucial role in maintaining energy balance and mitigating obesity risk [2]. Psychological stress has also been implicated in obesity through neuroendocrine mechanisms involving activation of the hypothalamic–pituitary–adrenal axis, leading to altered eating behaviors and increased fat deposition [17]. Importantly, these factors may interact with genetic variants such as MC4R, giving rise to gene–environment interactions that influence phenotypic outcomes [18]. However, such interactions remain underexplored in Indian young adult populations, particularly in relation to dietary patterns and stress.

Furthermore, reliance on BMI alone may not adequately capture differences in adiposity, as individuals with similar BMI may exhibit significant variation in body composition, including fat mass and lean mass [19]. Assessment of body composition provides a more comprehensive understanding of obesity-related risk and may help to better elucidate the role of genetic and environmental determinants.

Despite extensive research on MC4R polymorphisms in Western populations, evidence from the Indian population, particularly among young adults, remains limited. Rapid lifestyle transitions, changing dietary habits and increasing stress levels in this population may further modify genetic susceptibility to obesity [20]. Understanding the interplay between genetic and modifiable lifestyle factors is therefore critical for identifying high-risk individuals and developing targeted prevention strategies.

2. Objectives

Therefore, the present study aims to investigate the association between MC4R rs12970134 polymorphism and body composition among young obese adults, while examining the influence of dietary patterns, perceived stress, and physical activity. It is hypothesized that MC4R gene polymorphism is associated with variations in body composition and dietary behavior in this population.

3. Methods

Study Design: This study was designed as an analytical cross-sectional study to investigate the association between MC4R gene polymorphism (rs12970134) and obesity-related traits among young obese adults. The study further aimed to examine the relationship between genetic variation, body composition parameters, dietary intake, and perceived stress levels in young obese adults.

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Study Site and Duration: The study was conducted at MGM School of Biomedical Sciences and MGM Medical College, Kamothe, Navi Mumbai, India from August 2025 to March 2026. Laboratory analyses, including DNA isolation, polymerase chain reaction (PCR), and genotyping, were carried out at the Central Research Laboratory, MGM Institute of Health Sciences, Navi Mumbai.

Population Sampling and Ethical Considerations: The study population comprised college-going 60 obese individuals of both genders, aged 18–25 years and enrolled at MGM Institutions, Navi Mumbai. Participants were recruited using a convenience sampling technique through campus-based announcements. The inclusion of obese participants was based on the Asia-Pacific BMI classification (≥25 kg/m²) [23], and willingness to participate in the study. Individuals diagnosed with metabolic disorders, undergoing weight-loss treatment, chronic systemic illnesses, or acute infections at the time of the study were excluded. The sample size was determined based on standard recommendations for exploratory genetic association studies, where 50–100 participants are considered adequate to detect moderate effect sizes ($r \approx 0.30–0.35$) with 80% power at $\alpha = 0.05$, while also ensuring sufficient minor allele frequency representation for MC4R rs12970134 polymorphism.

The study was approved by the Institutional Ethics Committee of MGM Institute of Health Sciences (MGMIHS), Kamothe, Navi Mumbai (Ethical Approval No. MGM/DCII/IEC/60/01/2025, dated April 24, 2025). All procedures adhered to the principles of the Declaration of Helsinki, and written informed consent was obtained from all participants prior to enrollment [21].

Socio-demographic Data Collection: Baseline demographic data were collected using a structured questionnaire. Socioeconomic status was assessed using the Modified Kuppuswamy Socioeconomic Status Scale (2024) [22].

Anthropometric Measurements and Body Composition Assessment: Anthropometric measurements were obtained using standardized procedures. Body weight was measured using a calibrated digital weighing scale, and height was measured using a stadiometer. BMI was calculated using the standard formula: BMI = Weight (kg) / Height (m)². Participants were categorized according to Asia-Pacific BMI classification guidelines [23]. Body composition was assessed using Bioelectrical Impedance Analysis (BIA), which estimates body fat percentage, lean mass, and total body water based on electrical conductivity of body tissues [24].

Dietary Assessment: Nutrient intake was estimated using the 24-hour dietary recall method. Habitual dietary patterns were assessed using a semi-quantitative Food Frequency Questionnaire (FFQ).

24-Hour Dietary Recall: Dietary intake was assessed using the 24-hour dietary recall method, a widely used approach in nutritional epidemiology for estimating short-term dietary intake [25]. Nutrient values were calculated using standard Indian food composition tables.

Food Frequency Questionnaire (FFQ): Habitual dietary intake of major food groups was assessed using a semi-quantitative Food Frequency Questionnaire (FFQ), a validated tool used to assess long-term dietary patterns [26]. The FFQ estimate was considered for the last three months food intake of the participants.

Perceived Stress Assessment: Perceived stress was measured using the Perceived Stress Scale (PSS-14), a validated psychological instrument developed to assess perceived stress levels [27].

Genotype analysis: Approximately 2 mL of venous blood was collected in EDTA vacutainers under aseptic conditions and stored at −80°C until analysis. Genomic DNA was extracted using the QIAamp DNA Mini Kit (QIAGEN) following manufacturer protocols, and DNA quality and concentration were assessed using spectrophotometry. Polymerase chain reaction (PCR) was performed to amplify the target region of the MC4R gene using gene-specific primers. PCR products were subjected to restriction enzyme digestion by DDE-I endonuclease enzyme to identify polymorphic variants. The digested product was run on agarose gel electrophoresis for separation and visualization under ultraviolet light. Participants were classified into three genotype groups based on MC4R rs12970134 polymorphism: GG (wild type), GA (heterozygous), and AA (homozygous risk genotype) [10].

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Statistical Analysis: Statistical analyses were performed using SPSS statistical software (v.25) after data entry in Microsoft Excel. Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Genotype distribution of the melanocortin-4 receptor (MC4R) gene was assessed, and Hardy–Weinberg equilibrium was evaluated using the chi-square (χ^2) test with 1 degree of freedom. Descriptive comparisons of body composition parameters across genotype groups (GG, GA, AA) were presented as mean ± standard deviation; inferential testing was not performed for genotype group comparisons due to the small sample size of the AA genotype group (n = 7). Pearson's correlation coefficient (r) was used to examine bivariate associations between MC4R genotype, body composition parameters, dietary intake variables, and perceived stress scores [28]. A p-value < 0.05 was considered statistically significant for all analyses.

4. Results

Table 1. Socio-demographic and socio-economic characteristics of participants (n=60)

Variable	Category	Frequency (n)	Percentage (%)	Mean ± SD
Gender	Male	14	23.3	-
	Female	46	76.7	-
Education	Undergraduate	48	80.0	-
	Postgraduate	12	20.0	-
Smoking status	Smoker	10	16.7	-
	Non-smoker	50	83.3	-
Sleep pattern	<4 hours	2	3.3	-
	5–8 hours	54	90.0	-
	>8 hours	4	6.7	-
Sedentary lifestyle	Mild	24	40.0	-
	Moderate	32	53.3	-
	Strenuous	4	6.7	-
Socioeconomic status	Upper (I)	8	13.3	28.0 ± 1.5
	Upper middle (II)	30	50.0	20.0 ± 3.0
	Lower middle (III)	12	20.0	13.0 ± 1.5
	Upper lower (IV)	6	10.0	8.0 ± 1.2
	Lower (V)	4	6.7	3.0 ± 1.0

The socio-demographic profile of the participants is presented in Table 1. The majority participants were female (76.7%, n=46), undergraduate students (80.0%, n=48), and non-smokers (83.3%, n=50). Most participants reported a sleep duration of 5–8 hours (90.0%, n=54). Regarding lifestyle patterns, 53.3% (n=32) exhibited moderate sedentary behaviour. Socio-economic status assessment using the Modified Kuppuswamy scale indicated that 50.0% (n=30) of participants belonged to the upper middle class, with an overall mean socio-economic score of 19.6 ± 6.0.

Table 2 summarizes the anthropometric and clinical characteristics of the obese young adults. The mean age was 21.1 ± 4.65 years. The mean BMI was

Table 2: Anthropometric and Clinical Profile (n=60)

Parameter	Mean ± SD
Age (years)	21.1 ± 4.65
Systolic BP (mmHg)	122.5 ± 22.57
Diastolic BP (mmHg)	79.8 ± 17.4
Height (cm)	160.25 ± 11.25
Weight (kg)	87.45 ± 20.30
BMI (kg/m²)	34.37 ± 5.88
Body fat mass (kg)	42.08 ± 12.88
Soft lean mass (kg)	42.40 ± 9.44
Fat-free mass (kg)	44.72 ± 9.66
Skeletal muscle mass (kg)	24.58 ± 5.86
Percent body fat (%)	48.58 ± 7.87
Waist-hip ratio	0.99 ± 0.10

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34.37 ± 5.88 kg/m², with a mean percent body fat of 48.58 ± 7.87% and body fat mass of 42.08 ± 12.88 kg. The mean waist-hip ratio was 0.99 ± 0.10. Blood pressure values were normal to pre-hypertensive with systolic BP of 122.5 ± 22.57 mmHg and diastolic BP of 79.8 ± 17.4 mmHg. Table 3 displays the food group consumption frequencies assessed by food frequency questionnaire for the last three months. Cereals were the most frequently consumed food group (36.90 ± 20.15), followed by junk foods (22.24 ± 14.71) and nuts (21.52 ± 10.38). Vegetables (10.38 ± 10.10) and pulses (12.78 ± 9.37) were the least frequently consumed items

Table 3: Food group consumption patterns (FFQ n=60)

Food group	Mean ± SD
Cereals	36.90 ± 20.15
Pulses	12.78 ± 9.37
Dairy	16.84 ± 10.01
Vegetables	10.38 ± 10.10
Fruits	14.40 ± 14.88
Nuts	21.52 ± 10.38
Non-vegetarian foods	11.98 ± 10.38
Junk foods	22.24 ± 14.71
Fats & oils	8.31 ± 6.20
Sugar	5.77 ± 4.32

Table 4. Mean daily nutrient intake and % RDA consumption by gender (n=60)

Nutrient	Male			Female		
	RDA	Intake (mean ± SD)	RDA consumed (%)	RDA	Intake (mean ± SD)	RDA consumed (%)
Energy (kcal)	2110	1550.35 ± 186.04	73.48	1660	1659.12 ± 199.09	99.95
Protein (g)	54	58.73 ± 7.05	108.75	46	53.84 ± 6.46	117.03
Carbohydrate (g)	300	190.17 ± 22.82	63.39	250	196.28 ± 23.55	78.51
Total fat (g)*	27.5	68.46 ± 8.21	249.85	22.5	72.05 ± 8.65	320.22
Dietary fibre (g)	30	19.72 ± 2.37	65.73	25	21.93 ± 2.63	87.71
Saturated fat (g)	23	22.42 ± 2.69	97.48	18	24.98 ± 3.00	138.76
MUFA (g)*	30	17.16 ± 2.06	57.20	25	15.35 ± 1.84	61.40
PUFA(g)*	20	13.12 ± 1.57	65.59	16	13.49 ± 1.62	84.29
Calcium (mg)	1000	336.96 ± 40.43	33.70	1000	298.82 ± 35.86	29.88
Iron (mg)	19	10.68 ± 1.28	56.23	29	9.50 ± 1.14	32.75
Vitamin A (µg)	1000	521.89 ± 625.43	52.19	840	561.28 ± 673.71	66.82

*Midpoint of RDA range used.

Table 4 summarizes the mean daily nutrient intake and percentage adequacy relative to RDA among male and female participants. Energy intake was inadequate in males (73.48% of RDA) but nearly adequate in females (99.95%). Protein intake exceeded recommendations in both groups. Carbohydrate and dietary fibre intakes were below RDA in both sexes. Total fat intake was markedly elevated, exceeding recommended levels in both males (249.85%) and females (320.22%). Saturated fat intake approached recommended levels in males but exceeded them in females. Intake of unsaturated fats (MUFA and PUFA) was below recommended levels in both groups. Micronutrient intake was notably inadequate, with low calcium and iron intake observed in both sexes, particularly among females. Vitamin A intake was also below RDA, with considerable inter-individual variability.

Table 5 shows the perceived stress levels measured using the Perceived Stress Scale (PSS-14). The majority of participants (70.0%, n=42) experienced moderate stress, followed by low stress (16.7%,

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n=10) and high stress (13.3%, n=8). The overall mean PSS score was 20.50 ± 6.45.

Table 5. Perceived stress levels (PSS, n=60)

Stress level	Score range	Frequency (n)	Percentage (%)	PSS score (Mean ± SD)
Low	0–13	10	16.7	13.3 ± 1.52
Moderate	14–26	42	70.0	19.8 ± 3.5
High	27–40	8	13.3	33.2 ± 3.80
Overall	0–40	60	100	20.50 ± 6.45

Table 6. Hardy–Weinberg equilibrium analysis

Genotype	Observed (O) (n)	Expected (E) (n)	Chi-square test		
			$\chi^2 = \Sigma(O-E)^2/E$	df	p-value
GG	28	27.336	0.151	2	0.927
GA	25	26.325			
AA	7	6.337			
Total	60	60			

PCR-RFLP analysis successfully amplified a 124 bp fragment of the MC4R gene. Following restriction digestion, three genotype patterns were identified: GG (single band at 124 bp), GA (bands at 124 bp and 104 bp), and AA (single band at 104 bp). Among the 60 participants, the genotype distribution was as follows: GG in 28 participants (46.7%), GA in 25 participants (41.7%), and AA in 7 participants (11.7%). Allele frequency analysis revealed that the G allele frequency (p) was 0.675 and the A allele frequency (q) was 0.325. The observed counts for GG, GA, and AA genotypes were 28, 25, and 7, respectively, while the expected counts were 27.336, 26.325, and 6.337, respectively. Chi-sqaure analysis was performed to assess whether the observed genotype frequencies deviated from expected frequencies under Hardy–Weinberg equilibrium ($p^2 + 2pq + q^2 = 1$). The calculated chi-square value was 0.151 with 2 degree of freedom, yielding a p-value > 0.05. This indicates that the genotype distribution did not deviate significantly from Hardy–Weinberg equilibrium, confirming that the study sample was genetically stable and suitable for association analysis.

Table 7. Body composition parameters (mean ± SD) by MC4R genotype

Genotype	BMI (kg/m²)	Body fat mass (kg)	Percent body fat (%)	Waist-hip ratio
GG (n=28)	33.51 ± 5.16	40.93 ± 12.40	48.20 ± 6.75	0.99 ± 0.10
GA (n=25)	34.71 ± 6.02	41.98 ± 12.04	48.36 ± 10.00	0.99 ± 0.10
AA (n=7)	36.59 ± 8.45	47.10 ± 18.79	50.91 ± 2.84	1.01 ± 0.10

Table 7 presents body composition parameters stratified by MC4R rs12970134 genotype. Participants with the AA genotype (n=7) showed numerically higher mean BMI (36.59 ± 8.45 kg/m²), body fat mass (47.10 ± 18.79 kg), percent body fat (50.91 ± 2.84%), and waist-hip ratio (1.01 ± 0.10) compared to GG (n=28) and GA (n=25) genotypes. Statistical testing was not performed due to the small AA genotype sample size.

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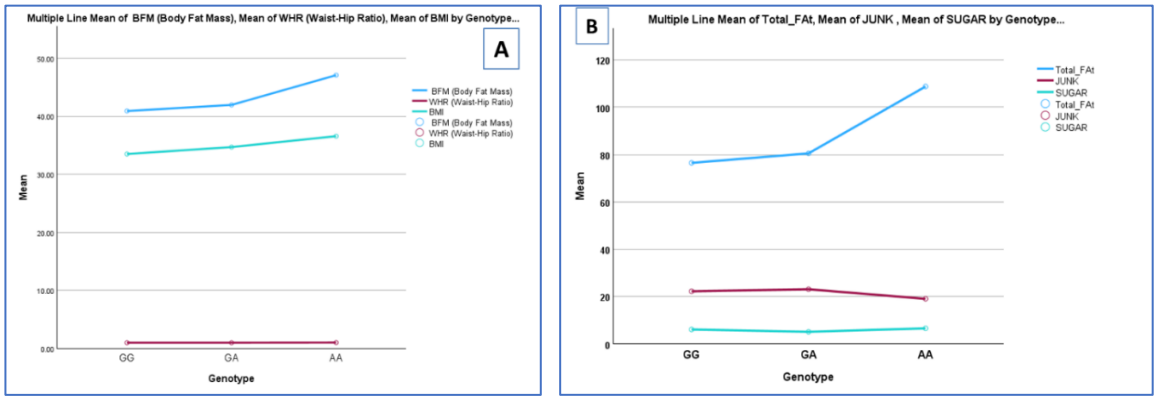

Figure 1: Multiple Line graphs showing genotype related variations in (A) Body fat mass, Waist to Hip ratio and BMI (B) Total intake of Fats, Junk and Sugar.

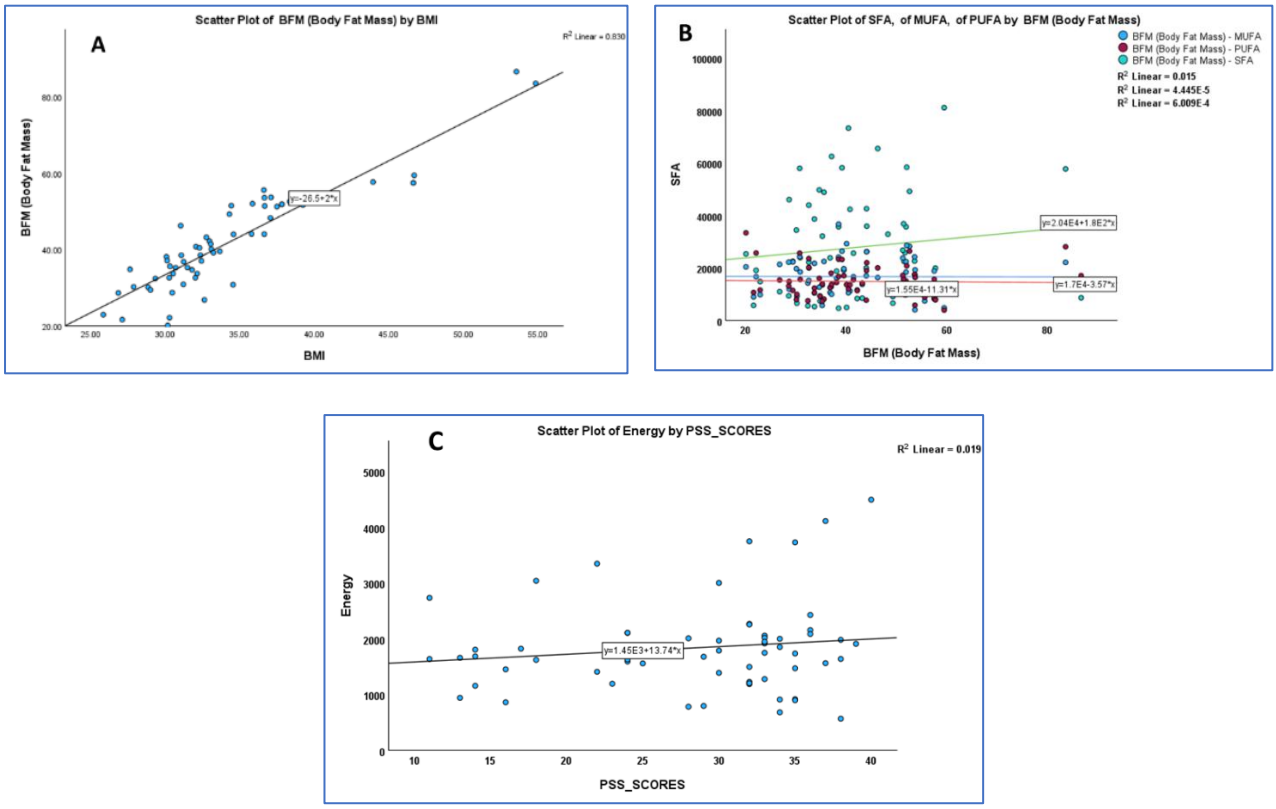

Figure 2: Correlation analysis of (A) BMI vs body fat mass; (B) fatty acid intake (SFA, MUFA, PUFA) vs body fat mass;(C) energy intake vs perceived stress score

Pearson correlation analysis showed associations between study variables. A very strong, statistically significant positive correlation was observed between BMI and body fat mass ($r = 0.911$, $p < 0.001$), as illustrated in **Figure 2A.** A moderate positive correlation was also observed between BMI and percent body fat ($r = 0.512$, $p < 0.001$). No statistically significant associations were found between individual fatty acids (saturated, monounsaturated, and polyunsaturated fats) and body fat mass (r ranging from −0.025 to 0.121; $p > 0.05$), as shown in **Figure 2B**. Similarly, energy intake showed a weak and non-significant correlation with perceived stress scores ($r = 0.137$, $p = 0.295$), as depicted in **Figure 2C.**

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5. Discussion

The present study investigated the association between MC4R gene polymorphism (rs12970134) and body composition, dietary patterns, and PSS among obese young adults. Obesity is a complex multifactorial disorder arising from the interaction of genetic, environmental, and behavioral factors, with genetic predisposition contributing substantially to inter-individual variability in adiposity [29,30]. Among the genetic determinants, the MC4R gene plays a pivotal role in appetite regulation and energy homeostasis through its involvement in the hypothalamic melanocortin pathway [31,32].

The sociodemographic profile of the study population demonstrated a predominance of female participants and undergraduate students, consistent with trends observed in university-based health research [33]. Although most participants reported adequate sleep duration and were non-smokers, a high prevalence of sedentary behavior was observed. This finding is noteworthy, as sedentary lifestyle patterns are independently associated with increased adiposity and metabolic risk in young adults [34,35]. The coexistence of adequate sleep and low physical activity suggests that lifestyle-related risk factors for obesity in this population may be driven more by inactivity than by sleep deprivation or substance abuse.

Anthropometric and body composition assessments revealed markedly elevated levels of adiposity, with mean BMI, body fat mass, and percent body fat all falling within the obese range. Given the relatively young age of participants, these findings are of particular concern, as early-onset obesity is strongly associated with increased long-term risk of cardio-metabolic disorders, including type 2 diabetes and cardiovascular disease [36]. Furthermore, the observed waist–hip ratio values indicate central fat accumulation, which is considered a more reliable predictor of cardio-metabolic risk than BMI alone [37].

Correlation analysis demonstrated a very strong positive association between BMI and body fat mass, along with a moderate positive correlation between BMI and percent body fat. These findings reinforce the utility of BMI as a practical surrogate indicator of adiposity at the population level, despite its recognized limitations in distinguishing between fat and lean mass [38,39]. The strong correlation observed in this study supports its continued use in epidemiological assessments, particularly when more advanced body composition tools are not feasible.

In contrast, no statistically significant associations were observed between individual fatty acid intake (SFA, MUFA, and PUFA) and body fat mass. This suggests that isolated nutrient components may not independently influence adiposity, and that overall dietary patterns and energy balance are likely more critical determinants. Additionally, methodological limitations inherent to dietary assessment techniques, including recall bias and day-to-day intake variability, may have contributed to the lack of observed associations [40,41].

The relationship between energy intake and perceived stress was found to be weak and non-significant. This aligns with existing literature suggesting that stress-related eating behaviors are highly heterogeneous, with individuals exhibiting both hyperphagic and hypophagic responses under stress [42]. The absence of a clear association in the present study may reflect variability in coping mechanisms, psychological resilience, and behavioral adaptations among participants.

Gene-polymorphism analysis revealed that MC4R genotype distribution in the study population was consistent with Hardy–Weinberg equilibrium, indicating a stable and representative study population. However, no statistically significant associations were observed between MC4R genotype and anthropometric or body composition parameters. Although individuals carrying the AA genotype exhibited higher mean BMI and body fat values, these differences were not statistically significant. This finding is consistent with previous studies demonstrating that common MC4R variants typically exert modest effects on obesity-related traits, often requiring large sample sizes to detect significant associations [43,44].

Interestingly, a statistically significant negative correlation was observed between MC4R genotype and total fat intake suggesting the influence of melanocortin pathway genetics on dietary behavior, particularly fat consumption patterns. The biological plausibility of this association is supported by evidence indicating the key role of melanocortin system in regulating appetite, satiety, and macronutrient preference [45,46]. However, no significant associations were identified between

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MC4R genotype and other dietary variables such as sugar intake or junk food consumption, suggesting a more specific rather than generalized influence on dietary behavior.

With respect to psychological factors, perceived stress did not demonstrate significant associations with BMI or body fat mass. A moderate negative correlation observed between perceived stress and waist–hip ratio was in contrasts with existing literature, which generally reports a positive association between chronic stress and central adiposity mediated through activation of the hypothalamic–pituitary–adrenal axis [42,47]. The unexpected direction of this relationship in the present study may be attributed to population-specific characteristics, reporting bias, or limitations associated with the cross-sectional design.

Overall, the findings of this study suggest that while the MC4R rs12970134 polymorphism is present among the Indian obese young adults, its direct association with obesity-related anthropometric parameters is limited. However, a significant negative correlation between MC4R genotype and total fat intake suggests that genetic variation in appetite-regulating pathways may influence dietary fat preferences. These findings underscore the importance of considering both genetic and environmental determinants in the development of obesity [49]. The unexpected moderate negative correlation between perceived stress and waist-hip ratio warrants further investigation. Overall, environmental and behavioral factors remain critical targets for obesity prevention and intervention strategies in young obese adult populations.

Thus, future research focusing on larger, longitudinal studies incorporating more precise dietary assessment tools and advanced gene–environment interaction models are warrantied to better elucidate the complex mechanisms underlying obesity. Such approaches would provide deeper insights into how genetic susceptibility interacts with modifiable lifestyle factors, thereby informing more targeted and personalized prevention strategies [50].

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