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Assessing the Role of *MC4R* Gene Variants and Dietary Habits in the Development of Obesity among Adolescents

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Abstract

Introduction: Obesity among adolescents is a growing public health concern, influenced by both genetic predispositions and modifiable lifestyle factors such as diet.

Objective: To assess the association between *MC4R* gene variants and dietary habits in the development of obesity among adolescents in Pakistan.

Hypothesis: This study hypothesizes that the C allele of *MC4R* rs17782313 and unhealthy dietary habits may interact to increase the risk of obesity among adolescents.

Methodology: A descriptive cross-sectional study was conducted from November 2023 to October 2024 at Abdul Wali Khan University Mardan, including 260 adolescents aged 12–18 years. Convenience sampling was employed. Anthropometric data were collected, and body mass index (BMI) was calculated using WHO growth reference standards. Dietary patterns were assessed using a validated food frequency questionnaire (FFQ). The FFQ was pilot tested and adapted to the local population. Trained personnel collected anthropometric measurements using standardized protocols to minimize inter-observer variability. Saliva samples were genotyped for *MC4R* rs17782313 polymorphism using PCR-based methods at the Centre of Excellence in Molecular Biology, Lahore. Laboratory staff were blinded to participants' obesity status. Genotyping call rates were 98.8%, and the genotype distribution was tested for Hardy-Weinberg equilibrium ($p = 0.47$). Statistical analysis was performed using SPSS version 26.0.

Results: Of the 260 participants, 18.46% (n = 48) were obese and 21.54% (n = 56) were overweight. Genotypic distribution revealed 56.92% (n = 148) with TT, 30.00% (n = 78) with TC, and 13.08% (n = 34) with CC genotype. Obesity was present in 12.16% of TT, 25.64% of TC, and 29.41% of CC genotypes ($p = 0.004$). High fast food consumption (≥3 times/week) and daily intake of sugar-sweetened beverages were significantly associated with obesity ($p < 0.01$). Multivariate logistic regression adjusted for socioeconomic status, sleep duration, and screen time showed CC genotype (OR = 3.08), high fast food intake (OR = 2.97), and low physical activity (OR = 1.79) as independent predictors. The model fit was acceptable (Hosmer–Lemeshow $p = 0.65$; Nagelkerke $R^2 = 0.38$). Gene-diet interaction was tested using an interaction term in the logistic model.

Conclusion: *MC4R* gene variants were associated with increased obesity prevalence in adolescents, especially in the presence of unhealthy dietary habits. Due to the cross-sectional nature of this study, causality cannot be inferred. Future prospective cohort studies are recommended to establish temporal relationships and validate these associations. These findings support the integration of genetic and lifestyle risk assessments for targeted obesity prevention strategies in youth.

Keywords: *MC4R* gene, obesity, adolescents, dietary habits, gene-diet interaction, Pakistan.

Introduction

The incidence of obesity in children and adolescents is rising quickly, making it a serious worldwide health problem [1]. The World Health Organization reports that the number of teenagers who are overweight or obese has increased significantly in recent decades, especially in low- and middle-income nations where dietary habits and levels of physical activity have been impacted by urbanisation and lifestyle changes [2,3]. Examining the variables that contribute to obesity is crucial throughout adolescence because it is a crucial developmental stage characterised by behavioural and physiological changes that may make people more likely to accumulate excessive amounts of weight [4].

A growing body of research indicates that genetic variables might modulate individual vulnerability to obesity, even while environmental and behavioural factors including sedentary lifestyle, excessive calorie

consumption, and poor food choices play a significant role in its beginning [5]. The melanocortin 4 receptor (*MC4R*) gene, which is essential for energy balance, appetite control, and body weight homeostasis, is one of the most researched genetic causes of obesity [6]. In a number of populations, variations in the *MC4R* gene, specifically single nucleotide polymorphisms (SNPs), have been connected to hyperphagia, increased hunger, and early-onset obesity [7].

The interplay between dietary practices and *MC4R* gene variations in teenagers may provide important new information about the complex nature of obesity [8]. Certain dietary habits, such as consuming a lot of processed foods, sugar-sweetened drinks, and saturated fats, may make the genetic susceptibility caused by *MC4R* polymorphisms worse [9]. On the other hand, well-balanced meals high in fruits, vegetables, and fibre may help reduce hereditary risk. Developing focused preventive and intervention methods requires an understanding of this gene-environment interaction, particularly in areas where obesity rates are rising [10]. This study hypothesizes that the C allele of *MC4R* rs17782313 and unhealthy dietary habits may interact to increase obesity risk in adolescents.

The function of *MC4R* variations in the teenage population of South Asia, whose cultural, nutritional, and genetic contexts vary greatly from Western settings, is little understood despite increased interest in gene-diet interactions. Therefore, in order to inform area-specific public health initiatives, it is vital to investigate both genetic predispositions and modifiable lifestyle variables in this region.

Research Objective
To assess the association between *MC4R* gene variants and dietary habits in the development of obesity among adolescents.

Materials and methods
Ethical Considerations
The study protocol was reviewed and approved by the Institutional Review Board (IRB) of AWKUM. All procedures adhered to the Declaration of Helsinki and national ethical standards. Informed written consent was obtained from all participants, with additional parental consent for minors. Confidentiality and the right to withdraw at any time were assured.

Study Design and Setting
This descriptive cross-sectional study was conducted at Abdul Wali Khan University Mardan (AWKUM) over one year, from November 2023 to October 2024. Participant recruitment and data collection were performed at AWKUM, while laboratory procedures, including DNA extraction and *MC4R* gene testing, were carried out at the Centre of Excellence in Molecular Biology (CEMB), Lahore.

Participant Selection Criteria
Participants in the research were teenagers of either sex, between the ages of 12 and 18, who lived permanently in the Mardan area and did not have any known endocrine or metabolic conditions. Every participant provided written, informed consent, and in the case of children, parental or guardian permission. Teens taking drugs that might affect hunger, metabolism, or body weight were not included, nor were adolescents with recent or ongoing infections. Furthermore, individuals who refused to give biological samples or who answered questionnaires incompletely were excluded from the final analysis.

Sampling Technique
Convenience sampling was employed, based on voluntary participation from students within the university catchment area. This may limit the generalizability of findings to the wider adolescent population of Pakistan and introduces the possibility of selection bias.

Sample Size
The sample size was calculated using the World Health Organization (WHO) formula for cross-sectional studies. Assuming an expected prevalence of adolescent obesity at 20%, a confidence level of 95%, and a margin of error of 5%, the required sample size was calculated to be 246. To account for potential dropouts or non-response, the sample size was increased to 260 participants. Although the sample size is slightly smaller than previous similar study, such as a recent study with 282 participants, it remains within a comparable range for observational research investigating gene-diet interactions in obesity [11].

Data Collection Procedures
The study team's standardized questionnaires and anthropometric measures were used in the data gathering process. Standardized procedures and calibrated equipment were used to measure each participant's height and weight. Weight in kilograms divided by height in meters squared (kg/m2) yielded the body mass index (BMI). In order to categorize obese status, BMI percentiles were then calculated using the age- and sex-specific growth reference criteria established by the World Health Organization. Anthropometric measurements were conducted by trained personnel using standardized protocols to ensure accuracy and minimize inter-observer variability. The food frequency questionnaire (FFQ) was adapted and pilot-tested for cultural relevance and reliability in the local population prior to full deployment. Dietary habits were evaluated using a validated food frequency questionnaire (FFQ), with particular attention paid to the frequency of intake of fruits, vegetables, whole grains, dairy products, sugar-sweetened drinks, and fast meals. Additional data on sleep habits, screen time, physical activity levels, family history of obesity, and socioeconomic background were gathered. When evaluating the gene-diet relationship, these factors were regarded as possible confounders.

Laboratory Analysis and Genotyping
Sterile collection kits were used to obtain saliva samples from each participant. Prior to being sent under cold chain conditions to the CEMB, one of Pakistan's top

centres for genetic and molecular research, the samples were kept at -20°C. Using QIAamp DNA Mini Kits (Qiagen), genomic DNA was extracted from the saliva samples at CEMB in accordance with the manufacturer's instructions. Laboratory personnel conducting genetic analyses were blinded to participants' obesity status to prevent observer bias.

Using a NanoDrop spectrophotometer, the amount and quality of the extracted DNA were evaluated by calculating absorbance ratios at 260 and 280 nm to guarantee purity. The rs17782313 polymorphism, which is the target area of the *MC4R* gene, was amplified using the polymerase chain reaction (PCR) for genotyping. The polymorphic site was flanked by specific primers. To verify effective amplification, 2% agarose gel electrophoresis was used to analyse the amplified PCR products. Depending on reagent availability and laboratory circumstances, either restriction fragment length polymorphism (RFLP) analysis or allele-specific PCR was used to genotype the rs17782313 variation. Every process was completed in compliance with quality assurance guidelines. Both positive and negative controls were included in every experimental run, and 10% of the samples were chosen at random for repeat analysis to guarantee accuracy and dependability. The genotyping call rate was 98.8%, and genotype frequencies conformed to Hardy–Weinberg equilibrium ($p = 0.47$), supporting data quality and validity.

Statistical Analysis

Version 26.0 of IBM SPSS Statistics was used to input and analyze all data. Dietary habits, genotype frequencies, and demographic traits were compiled using descriptive statistics. Using independent sample t-tests for continuous variables and chi-square tests for categorical data, the relationship between *MC4R* gene variations and obesity was investigated.

Multivariate logistic regression analysis was conducted to assess the independent and combined effects of dietary practices and *MC4R* polymorphisms on obesity, adjusting for confounders including socioeconomic status, sleep duration, screen time, family history, and physical activity. Model fit was assessed using the Hosmer–Lemeshow test ($p = 0.65$) and Nagelkerke's R^2 (0.38). To test for gene–diet interaction, an interaction term between genotype and dietary variables was included in the regression model. P-values below 0.05 were regarded as statistically significant.

Results

The demographic and lifestyle details of the 260 teenage participants are shown in table 1. The plurality (41.54%) were between the ages of 15 and 16, followed by those between the ages of 12 and 14 (35.38%) and 17 and 18 (20.08%). Females made up 46.92% (n = 122) and men 53.08% (n = 138). Forty percent (n = 104) reported having a positive family history of obesity, and forty-three percent (n = 112) reported being physically inactive. Furthermore, 45.38% (n = 118) slept for fewer than seven hours per night, and 56.54% (n = 147) reported using screens for more than two hours every day.

Table 1: Baseline Characteristics of Study Participants.

Variable	Category	Frequency (n)	Percentage (%)
Age Group	12–14 years	92	35.38
	15–16 years	108	41.54
	17–18 years	60	23.08
Sex	Male	138	53.08
	Female	122	46.92
Family History of Obesity	Yes	104	40.00
	No	156	60.00
Physical Activity Level	Low	112	43.08
	Moderate	96	36.92
	High	52	20.00
Screen Time (>2 hours/day)	Yes	147	56.54
	No	113	43.46
Sleep Duration (<7 hours/night)	Yes	118	45.38
	No	142	54.62

According to WHO guidelines, figure 1 groups individuals into BMI percentiles. Of the 260 teenagers, 11.15% (n = 29) were underweight, 48.85% (n = 127) were between normal weight ranges, 21.54% (n = 56) were overweight, and 18.46% (n = 48) were obese. This distribution shows that approximately 40% of individuals were overweight or obese, indicating a considerable prevalence of unhealthy weight.

Among participants, 56.92% (n = 148) had the TT genotype, 30.00% (n = 78) had the TC genotype, and 13.08% (n = 34) had the CC genotype (table 2). Obesity prevalence was 12.16% in TT, 25.64% in TC, and 29.41% in CC individuals, respectively ($p = 0.004$). The genotype distribution did not deviate from Hardy–Weinberg equilibrium ($p = 0.47$).

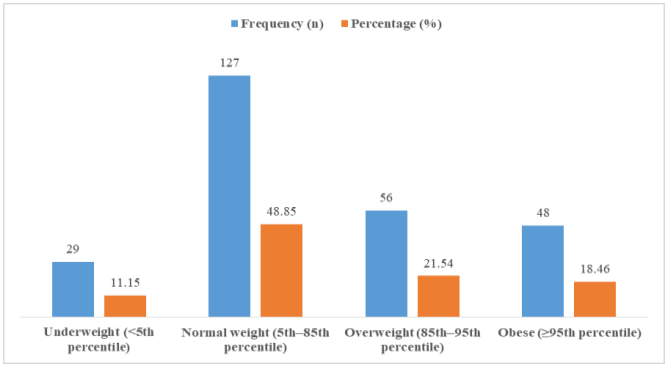

Figure 1: BMI Classification According to WHO Standards.

Table 2: *MC4R* rs17782313 Genotype Distribution and Association with Obesity

Genotype	Total (n)	% of Total	Obese (n)	% Obese within Genotype
TT (wild-type)	148	56.92%	18	12.16%
TC (heterozygous)	78	30.00%	20	25.64%
CC (homozygous mutant)	34	13.08%	10	29.41%

*Chi-square p-value = 0.004**

Obesity was significantly more prevalent among adolescents who consumed fast food ≥3 times/week (31.91%) and those who consumed sugar-sweetened beverages daily (30.77%). Low fruit and vegetable intake (<5 servings/day) was also associated with higher obesity prevalence (23.60%), shown in table 3.

Table 3: Dietary Habits and Obesity Status among Adolescents

Dietary Habit	Category	Obese (n)	% Within Category
Fast Food (≥3x/week)	Yes	30	31.91
	No	18	10.29
Sugar-Sweetened Beverages (daily)	Yes	28	30.77
	No	20	11.24
Fruit & Vegetable Intake (≥5 servings/day)	Yes	6	6.98
	No	42	23.60

Adjusted regression analysis included socioeconomic status, sleep duration, screen time, family history, and physical activity (table 4). Significant predictors of obesity were CC genotype (OR = 3.08), TC genotype (OR = 2.41), fast food intake ≥3 times/week (OR = 2.97), sugar-sweetened beverage intake (OR = 2.84), low physical activity (OR = 1.79), and family history of obesity (OR = 2.16). Sleep duration was not statistically significant (p = 0.138). Model fit was acceptable with Hosmer–Lemeshow p = 0.65 and Nagelkerke R^2 = 0.38.

Table 4: Multivariate Logistic Regression – Predictors of Obesity

Variable	Adjusted OR	95% CI	p-value
MC4R Variant (TC vs. TT)	2.41	1.18 – 4.94	0.016*
MC4R Variant (CC vs. TT)	3.08	1.21 – 7.83	0.018*
Fast Food ≥3x/week	2.97	1.51 – 5.83	0.002*
Sugar-Sweetened Beverages (daily)	2.84	1.37 – 5.89	0.004*
Low Physical Activity	1.79	1.02 – 3.12	0.042*
Sleep <7 hours	1.54	0.87 – 2.72	0.138
Family History of Obesity	2.16	1.20 – 3.90	0.010*

Independent sample t-tests compared obese (n = 48) and non-obese (n = 212) participants. Obese adolescents had a significantly higher mean BMI (28.42 ± 2.95 vs. 20.73 ± 3.12 kg/m^2), longer daily screen time (3.41 ± 1.08 vs. 2.21 ± 0.89 hours), shorter sleep duration (6.23 ± 1.02 vs. 7.11 ± 0.97 hours), and lower physical activity (2.15 ± 0.84 vs. 3.09 ± 1.12 hours/week), all with p < 0.001 (table 5).

Table 5: Independent Sample t-Tests for Obese vs. Non-Obese

Variable	Obese (Mean ± SD)	Non-Obese (Mean ± SD)	t-value	p-value
BMI (kg/m^2)	28.42 ± 2.95	20.73 ± 3.12	18.62	<0.001*
Daily Screen Time (hours)	3.41 ± 1.08	2.21 ± 0.89	7.88	<0.001*
Sleep Duration (hours/night)	6.23 ± 1.02	7.11 ± 0.97	–5.46	<0.001*
Physical Activity Duration (hours/week)	2.15 ± 0.84	3.09 ± 1.12	–6.08	<0.001*

Discussion

The current study investigated the relationship between *MC4R* rs17782313 gene variations and dietary practices in relation to adolescent obesity in a Pakistani population. It was shown that 18.46% of the 260 individuals were obese, while 21.54% were overweight. These results highlight the growing trend of adolescent obesity in South Asia, showing that over 40% of teenagers were overweight. This finding reflects the rising burden of adolescent obesity in South Asia and aligns with regional trends reported in earlier studies. For instance, a study in New Delhi observed an increase in obesity prevalence among urban adolescents from 9.8% in 2006 to 11.7% in 2009, while a Nigerian study found 11.4% overweight and 2.8% obesity among school-aged children [13].

According to genotypic analysis, 13.08% of participants carried the homozygous mutant CC genotype, 30% were heterozygous (TC), and 56.92% had the wild-type TT genotype. Obesity prevalence was notably higher among carriers of the C allele (TC: 25.64%, CC: 29.41%) compared to TT carriers (12.16%), highlighting a statistically significant association ($p = 0.004$) between *MC4R* polymorphism and obesity susceptibility. These results align with previous literature indicating that the C allele of rs17782313 is associated with increased obesity risk, particularly in adolescents and young adults [14].

The logistic regression model further confirmed that both daily consumption of sugar-sweetened beverages (OR = 2.84) and fast food intake ≥3 times/week (OR = 2.97) remained significant independent predictors of obesity after adjusting for genotype and confounders. The prevalence of obesity among adolescents who ate fast food three or more times per week was 31.91%, while the prevalence among adolescents who regularly drank sugar-sweetened drinks was 30.77%. On the other hand, only 6.98% of teenagers who consumed a lot of fruits and vegetables (≥5 servings per day) were obese. These dietary effects were robust even after adjustment and suggest a strong modifiable risk factor contributing to adolescent obesity. Global data supports these findings, indicating that processed foods and sugary beverages, together with high energy density diets, aggravate genetic predispositions to obesity [15,16].

Adolescents with the TC or CC genotype who frequently consumed fast food had a substantially higher likelihood of obesity than TT genotype individuals with healthier diets. This interaction emphasizes how behavioral and genetic risk factors can combine to elevate obesity risk, reinforcing the biological plausibility of *MC4R*'s role in energy homeostasis and appetite regulation. Previous research has shown similar gene-diet interactions involving *MC4R* variations, supporting the idea that nutrition modulates genetic vulnerability [17,18].

However, caution is warranted when interpreting these associations. Given the cross-sectional nature of the study, causal relationships cannot be inferred. It is unclear whether unhealthy dietary habits led to obesity or whether pre-existing obesity influenced dietary choices. Future longitudinal and prospective cohort studies are needed to clarify temporal relationships.

The observed associations are biologically plausible. *MC4R* encodes a receptor involved in the leptin–melanocortin pathway that regulates satiety and energy expenditure. Genetic variants at rs17782313 may disrupt this signaling, resulting in hyperphagia and decreased energy regulation. This biological rationale supports the observed relationship between *MC4R* variants and elevated obesity risk, particularly when compounded by high-calorie dietary exposure.

Interestingly, although variables such as sleep duration and screen time showed significant differences in univariate analysis between obese and non-obese groups, they did not remain significant in the multivariate model, possibly due to measurement limitations, residual confounding, or intercorrelations with dietary and physical activity behaviors. Similar patterns have been reported in other adolescent obesity studies, indicating that these lifestyle factors may play a mediating or contextual role.

Strengths and Limitations

This study has several strengths. It is among the first to explore *MC4R*-related gene–diet interactions in Pakistani adolescents using validated dietary tools and robust genotyping protocols. Adjustment for multiple behavioral and socioeconomic confounders, inclusion of model fit statistics, and high genotyping call rates enhance the credibility of the findings. The inclusion of interaction analysis between genotype and diet represents a key contribution, offering a more nuanced understanding of obesity etiology in adolescents.

Nonetheless, several limitations should be acknowledged. First, the cross-sectional design limits causal inference. Second, dietary data were self-reported, introducing the possibility of recall and reporting bias. Third, the study used convenience sampling within a single geographic region, limiting generalizability. Fourth, potential population stratification in genetic analysis cannot be excluded. Finally, the FFQ used assessed intake frequency but not portion size, which may reduce dietary assessment precision.

Conclusion

In conclusion, this study provides evidence that *MC4R* gene variants are associated with increased obesity prevalence in Pakistani adolescents, and that this association is exacerbated by unhealthy dietary habits. The identification of a significant gene–diet interaction reinforces the importance of considering both genetic and environmental contributions to adolescent obesity. While the findings support the importance of gene–environment interactions in obesity, further prospective and interventional studies are required to confirm these

associations and guide effective prevention strategies. Public health interventions that combine genetic screening with culturally tailored lifestyle interventions may offer a promising approach to curbing adolescent obesity in Pakistan and similar settings.

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Conflict of interest
The authors state no conflict of interest.

Author Contributions
Ahmad Faraz: Conceptualization, data collection, laboratory analysis, and drafting of the manuscript. Muhammad Saqib Khan: Methodology design, literature review, data interpretation, and critical revision of the manuscript. Tipu Sultan Haider: Statistical analysis, data visualization, and contributed to manuscript editing. Hafsa Khalil: Supervision, project administration, final review, and corresponding author responsibilities.

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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