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EXPLORING THE RELATIONSHIP BETWEEN DIETARY PATTERNS AND THE RS2241766 POLYMORPHISM IN THE ADIPONECTIN GENE IN CORONARY ARTERY DISEASE: A GENE-DIET INTERACTION STUDY

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ABSTRACT:

Introduction The coronary artery disease remains a major health issue worldwide, especially in low- and middle-income countries where genetic predispositions significantly contribute to its prevalence.

Materials and methods This study, conducted at Chettinad Hospital and Research Institute, Kelambakkam, India, from January to April 2024, investigates the relationship between dietary patterns, genetic variations in Adiponectin, and coronary artery disease risk. Genetic analysis of the adiponectin (45T/G) polymorphism was performed using the Tetra-primer Amplification Refractory Mutation System Polymerase Chain Reaction method.

Rezults The results revealed notable links between genetic variations, dietary behaviours, and health indicators coronary artery disease patients. Anthropometric and biochemical measurements showed that narrowed coronary arteries were associated with elevated BMI and waist circumference. Lifestyle and sleep patterns also differed significantly between the groups. Among coronary artery disease participants, 64% followed non-vegetarian diets, with higher consumption of red meat and fast food, negatively impacting lipid profiles. The rs2241766 GG genotype of the adiponectin gene was significantly associated with these dietary habits ($p<0.01$) and obesity (OR=5.1429, p=0.0084). Similarly, the GT genotype demonstrated a strong correlation with obesity (OR=22.15, $p < 0.050$).

Conclusions This study highlights the intricate connections between genetic predispositions, dietary choices, and coronary artery disease risk. The rs2241766 GG genotype emerged as a strong predictor of coronary artery disease susceptibility compared to the rs2231142 TT genotype, emphasizing the complex interplay of diet, genetics and obesity in coronary artery disease development.

Keywords Gene-Diet interaction, adiponectin Rs2241766, coronary artery disease, genotype, dietary pattern.

EXPLORAREA RELAȚIEI DINTRE MODELE DE DIETE ȘI POLIMORFISMUL RS2241766 ÎN GENA ADIPONECTINEI ÎN BOALA CORONARIANĂ: UN STUDIU DE INTERACȚIUNE GENĂ-DIETĂ

Introducere Boala coronariană rămâne a fi o problemă majoră de sănătate la nivel global, în special în țările cu venituri mici și medii, unde predispozițiile genetice contribuie în mod semnificativ la prevalența acesteia.

Materiale și metode Studiul a fost realizat la Chettinad Hospital and Research Institute, Kelambakkam, India, din ianuarie până în aprilie 2024, și a investigat relația dintre regimurile alimentare, variațiile genetice ale adiponectinei și riscul de boală coronariană. Analiza genetică a polimorfismului adiponectinei (45T/G) a fost efectuată utilizând metoda Tetra-primer Amplification Refractory Mutation System Polymerase Chain Reaction.

Rezultate Au fost evidențiate legături notabile între variațiile genetice, comportamentele alimentare și indicatorii de sănătate la pacienții cu boală coronariană. Măsurătorile antropometrice și biochimice au demonstrat că arterele coronare îngustate au fost asociate cu indicele de masă corporală și circumferința taliei crescute. Printre participanții cu boli coronariene, 64% au urmat diete non-vegetariene, cu un consum mai mare de carne roșie și fast-food, influențând negativ profilurile lipidice. Genotipul rs 2241766 GG al genei adiponectinei a fost asociat semnificativ cu aceste obiceiuri alimentare ($p<0,01$) și cu obezitatea (OR=5,1429, p=0,0084). În mod similar, genotipul GT a demonstrat o corelație puternică cu obezitatea (OR=22,15, $p <0,050$).

Concluzii Acest studiu relevă conexiunile polivalente dintre predispozițiile genetice, selectarea dietei și riscul de afecțiune coronariană. Genotipul rs2241766 GG s-a manifestat ca un predictor puternic al susceptibilității la boala coronariană în comparație cu genotipul rs2231142 TT, subliniind interacțiunea complexă a dietei, geneticii și a obezității în dezvoltarea bolii coronariene.

Cuvinte cheie Interacțiunea genă-dietă, adiponectina Rs 2241766, boala coronariană, genotip, tipar alimentar.

INTRODUCTION

Coronary artery disease (CAD) results from plaque build-up in coronary arteries, restricting blood flow to the heart. Although CAD mortality has declined since the 1960s, it remains a leading cause of death. Atherosclerosis, driven by preventable factors like hypertension and smoking, as well as non-modifiable risks like age and genetics, leads to endothelial dysfunction and plaque formation (1-5). Diet plays a crucial role in CAD risk, with plant-based diets (PBDs) reducing the risk by up to 29% by preventing endothelial damage, LDL oxidation, and macrophage activation (6). CAD has a heritability of 40-60%, with genetic variations (SNPs) linked to disease susceptibility. Genome-wide association studies (GWAS) have identified SNPs associated with CAD. Adiponectin, which regulates metabolism, correlates with CAD, obesity, and diabetes. Genetic factors, including polymorphisms in the adiponectin gene, interact with diet to influence health outcomes (7-15). This cross-sectional study in South India explored the relationship between dietary patterns and the rs2241766 SNP in the adiponectin gene, aiming to understand its association with CAD and improve health outcomes.

MATERIALS AND METHODS

STUDY DESIGN AND POPULATION

This cross-sectional study was conducted at Chettinad Hospital and Research Institute (Chennai) from January to March 2024. It included 50 participants: 25 with Coronary Artery Disease (CAD) and 25 healthy controls, aged 30-70 (mean age: 45.4 ±12.81). Participants were excluded if they had chronic conditions, were pregnant or lactating, or used alcohol or tobacco. The study was approved by the Institutional Ethics Committee.

QUESTIONNAIRE

A semi-structured Food Frequency Questionnaire (FFQ) with 12 food groups was used to gather demographic, lifestyle, and dietary data. It captured food consumption frequency ranging from “never” to “2 or more times a day”, ensuring cultural relevance and aligning with study objectives.

ANTHROPOMETRIC DETAILS

Height, weight, BMI (calculated as weight in kg/height in m^2), and waist-to-hip ratio (WHR) were measured using standard procedures.

BIOCHEMICAL ANALYSIS

Blood samples were collected to measure cholesterol levels (TC, TG, HDL-C, LDL, VLDL), fasting blood glucose (hexokinase method), HbA1c (HPLC method), and cardiac markers (Trop-I, BNP, CK-MB) using Siemens and Beckman Coulter analyzers.

DNA EXTRACTION AND GENOTYPING

DNA was extracted from peripheral blood leukocytes and analyzed for the ADIPOQ-RS2241766 SNP using Tetra-primer ARMS PCR. The PCR products were analyzed by agarose gel electrophoresis.

DIETARY CLASSIFICATION

Dietary patterns were classified into 10 food groups based on nutrient profiles, including categories such as red meat, seafood, fast food, and high-salt diets.

STATISTICAL ANALYSIS

Descriptive statistics summarized categorical variables, and continuous variables were expressed as mean ± standard deviation. An independent t-test was used to compare CAD and non-CAD groups. Pearson's correlation analyzed biochemical and food group relationships. Genotype distribution was assessed using Hardy-Weinberg equilibrium and Fisher's exact test. Odds ratios were calculated for genotype and dietary pattern associations. Statistical significance was set at $p < 0.05$, with analysis conducted using IBM SPSS version 29.0.

RESULTS

The table presents the mean and standard deviation (SD) values for various clinical and biochemical parameters, comparing overall (N=50), CAD group shows significantly higher glycaemic levels, lipid abnormalities, and elevated cardiac markers, indicating higher cardiovascular risk compared to non-CAD group.

Table 1. Characteristics of the Participants.

Parameters	N=50 Mean± SD	CAD group Mean ± SD	Non - CAD group Mean ± SD
AGE (yrs)	45.4 ±12.81	45.63 ±14.18	42.64 ±10.67
BMI kg/m2	23.3 ± 3.99	20.63 ± 2.322	26.10 ±3.99
WHR cm	0.82 ± 0.072	0.766 ± 0.058	0.87 ± 0.04
FBG mg/Dl	136.92 ± 55.50	88.28±13.36	190.84 ±29.53
Post Prandial mg/Dl	198.28 ± 46.99	160.92 ±24.23	238.72 ±30.19
HBA1C %	6.43 ± 1.61	5.20 ± 0.35	7.83 ±1.45
HDLC mg/Dl	40.72 ± 14.78	49.92 ±7.63	31.04 ±14.69
LDL mg/Dl	129.50 ± 27.67	107.96 ±18.50	153 ±11.53
VLDL mg/Dl	33.54 ± 15.15	20.68 ±6.52	47.36 ±8.19
TGL mg/Dl	179 ± 134.07	97.76±27.99	284.16 ±167.17
TC mg/Dl	293.42 ± 169.97	181.40 ± 3.59	426.28±176.42
NON- HDLC mg/Dl	139.90 ± 36.76	113.08 ±16.17	168.52 ±29.83
TROP I (pg/ml)	1091.43± 3483.8	2175.2 ± 4703.7	7.66± 4.56
BNP (peg /ml)	637.29 ± 1311.55	1217.5 ±1644.6	34 ±21.2
CKMB (mg/ml)	8.644 ± 10.828	14.94± 12.4	2.14 ± 1.426
Systolic BP (mm Hg)	140.45 ± 18	153.58 ± 18.46	128.2± 6.86
Diastolic BP (mm Hg)	84.38±12.78	95 ± 7.63	73.76 ± 6.46

*__BMI__: Body Mass Index; *__WHR__: Waist Hip Ratio; *__FBG__: Fasting Blood Glucose; *__HBA1C__: Glycosylated; Haemoglobin; *__TGL__: Triglycerides; *__TC__: Total Cholesterol; *__LDL__: Low-Density Lipoprotein; *__HDL__: High-Density Lipoprotein; *__BP__: Blood Pressure; *__CAD__: Coronary Artery Disease; *__SNP__: Single Nucleotide Polymorphism; *__TROP I__: Troponin I; __CK-MB__: Creatine kinase-MB; *__BNP__: Brain Natriuretic Peptide; *__OR__: Odds Ratio; *__SD__: Standard Deviation. *(Abbreviations used here also correspond to other tables where applicable.)*

The table shows dietary patterns of 50 individuals: 34% are vegetarians, 54% non-vegetarians, and 16% eggetarians. In the CAD group, 20% are vegetarians, 64% non-vegetarians, and 20% eggetarians, while the non-CAD group has 48% vegetarians, 44% non-vegetarians, and 13.6% eggetarians.

Table 2. Dietary Patterns of the participants.

Dietary pattern	N=50	%	Cad N=25	%	Non-cad N=25	%
Vegetarian	17	34%	5	20%	12	48%
Non- vegetarian	27	54%	16	64%	11	44%
Eggetarian	8	16%	5	20%	3	13.6%

The bar chart shows the frequency of different dietary habits among CAD subjects (N=25), with high salt diet and fast food being the most prevalent, followed by red meat and seafood. The dietary habits are further categorized into small, medium, and high frequency, with high salt diet being the most common in the high frequency group.

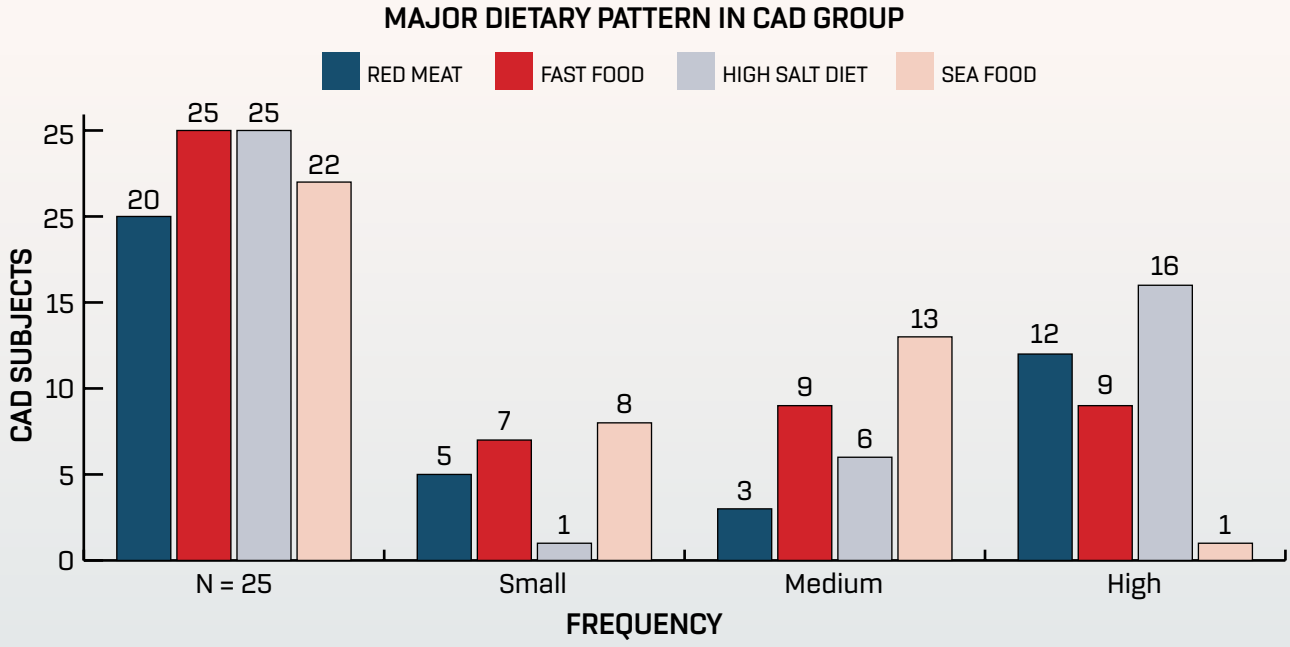

Figure 1. Major dietary pattern in CAD group.

The table shows Pearson correlation coefficients and p-values for cardiac markers with dietary factors such as red meat, fast food, seafood, and high salt intake. Significant correlations include HDL, LDL, VLDL, TGL, TC, and NON-HDLC with dietary components, while others like TROP-I, BNP, and CK-MB show non-significant associations.

Table 3. Correlation Analysis of biochemical parameters with dietary pattern among study articipants.

Cardiac marker	Pearson correlation	Red meat	Fast food	Sea food	High salt
TROP-I	r-value	0.077	-0.126	0.033	-0.014
	p-value	0.716	0.550	0.877	0.949
BNP	r-value	0.248	0.040	0.078	0.065
	p-value	0.232	0.848	0.709	0.757
CK-MB	r-value	0.091	0.236	-0.71	0.018
	p-value	0.665	0.256	0.736	0.931
HDL	r-value	-0.330	-0.327	-0.382	-0.112
	p-value	**0.02***	**0.003***	**0.05***	0.593
LDL	r-value	0.782	0.678	0.111	-0.078
	p-value	**0.02***	**0.01***	0.597	0.710
VLDL	r-value	0.868	0.764	-0.474	0.143
	p-value	**0.031***	**0.02***	**0.017***	0.495
TGL	r-value	0.891	0.678	-0.441	0.039
	p-value	**0.037***	**0.012***	**0.014***	0.853
TC	r-value	0.834	0.781	-0.228	-0.273
	p-value	**0.031***	**0.021***	0.273	0.186
NON-HDLC	r-value	0.718	0.151	-0.419	0.045
	p-value	**0.02***	0.472	**0.01***	0.833
Systolic BP	R-Value	0.141	0.025	0.035	0.618
	P-Value	0.452	0.913	0.733	**0.01***
Diastolic BP	R-Value	0.150	0.012	0.035	0.418
	p-value	0.521	0.812	0.733	**0.00***

Significant values are in bold*

The table shows the results of independent t-tests comparing clinical and biochemical parameters between CAD and non-CAD groups, with significant differences observed across all parameters ($p < 0.05$). Notably, parameters such as BMI, WHR, Troponin I, BNP, and various cholesterol levels exhibit substantial differences, highlighting the impact of CAD on these measures.

Table 4. Independent t-test of biochemical parameters between two groups.

Parameter	CAD group		Non-CAD group	
	T value	P value	T value	P value
BMI	7.607	**0.002**	7.506	**0.002**
WHR	6.370	**0.001**	6.216	**0.001**
TROP I	8.171	**<0.001**	8.171	**<0.001**
BNP	4.069	**<0.001**	4.069	**<0.001**
CKMB	4.853	**<0.001**	4.853	**<0.001**
Systolic BP	6.143	**<0.001**	6.143	**<0.001**
Diastolic BP	11.694	**0.000**	11.694	**0.000**
HDLC	5.701	**0.001**	5.701	**0.001**
LDLC	5.601	**<0.001**	5.601	**<0.001**
VLDLC	12.74	**0.002**	12.73	**0.002**
TGL	5.498	**<0.001**	5.498	**<0.001**
TC	6.905	**<0.001**	6.905	**<0.001**
NON-HDLC	8.171	**<0.001**	8.171	**<0.001**
HBA1C	8.805	**<0.001**	8.750	**<0.001**
FBS	10.04	**<0.001**	10.04	**<0.001**
Post Prandial	15.818	**<0.001**	15.818	**<0.001**

Significant values are in bold*

The table reveals significant differences between CAD and non-CAD groups in both lifestyle and sleep with significant p value <0.05.

Table 5. Independent t-test of non- biochemical parameters between two groups.

Variable	CAD group		Non-CAD group	
	T value	P value	Value	P value
Lifestyle	2.803	0.007	2.796	0.007
Sleep	4.387	0.000	4.394	0.000

Significant values are in bold*

The table below displays the frequency distribution of the ADIPOQ rs2241766 locus across study subjects, highlighting significant differences between CAD and non-CAD groups. The T:T genotype is notably absent in the CAD group but present in 26.9% of the non-CAD group, while the G:G genotype is more common in the CAD group (69.2%) than in the non-CAD group (7.7%). Additionally, allele frequencies vary significantly, with the T allele being predominant in the non-CAD group (56%) and the G allele more frequent in the CAD group (84%).

Table 6. Frequency distribution of the ADIPOQ rs2241766 locus across study subjects.

SNP site	Genotype /Allele	CAD group	NON-CAD group	P value
Rs2241766	T:T	0 (0.23)	7(26.9)	**<0.0001**
	T:G	7 (26.9)	23(88.5)	
	G:G:	18(69.2)	2(7.7)	
	T	8(0.16)	28 (0.56)	**0.0001**
	G	42(0.84)	28(0.56)	

Significant values are in bold*

The bar chart shows the frequency distribution of the ADIPOQ rs2241766 locus among study participants. The GG genotype and G allele were most common in individuals with coronary artery disease (CAD), followed by the TT genotype. Conversely, the TT and TG genotypes, as well as the T allele, were more prevalent in the non-CAD group compared to the CAD group.

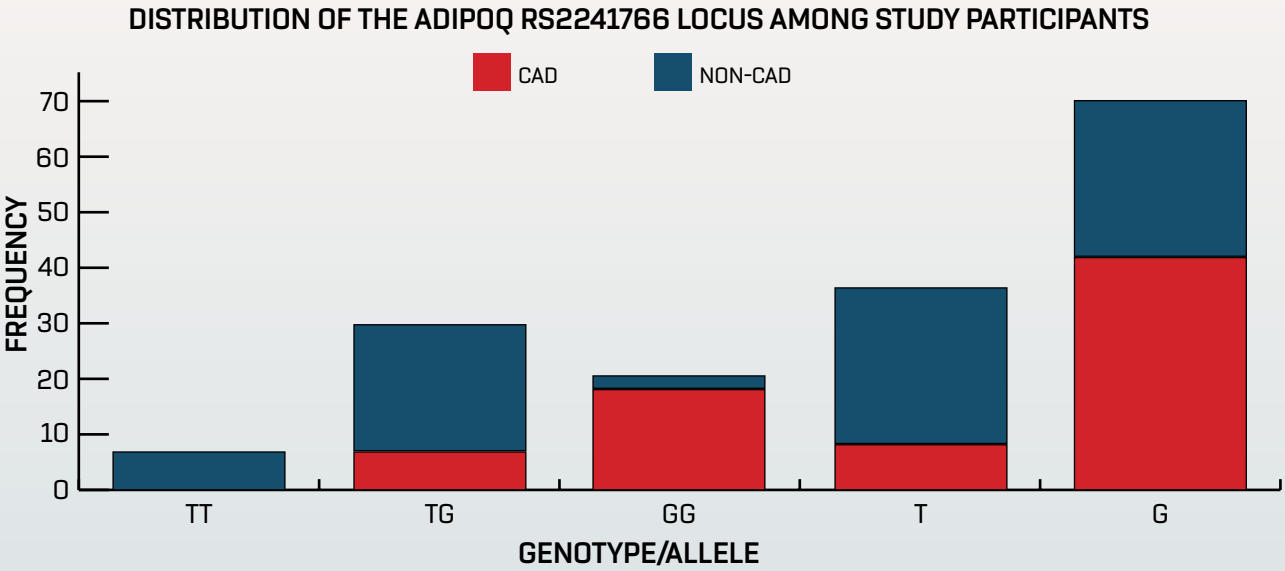

Figure 2. Distribution of the ADIPOQ rs2241766 locus among study participants.

In the CAD group, the GG genotype are much less likely to consume red meat compared to those without this genotype (OR<1). The statistically significant p-value (0.0182) and confidence interval (0.0085 to 0.6428) highlight a strong inverse association. While the GT genotype shows no significant. In the Non-CAD group, neither genotype demonstrated a significant effect with red meat.

Table 7. Interaction between genotypes and red meat consumption.

GROUP	GENE-DIET INTERACTION	OR	95%CL	P VALUE
CAD	GG VS RED MEAT	0.0741	0.0085 to 0.6428	**0.0182**
	GT VS RED MEAT	1.1953	0.3703 to 3.8582	0.7654
NON-CAD	GG VS RED MEAT	1.3788	0.4529 to 4.1973	0.5717
	GT VS RED MEAT	0.8366	0.2592 to 2.7004	0.7654

***CAD**: Coronary Artery Disease ***OR**: Odds Ratio ***CL**: Confidence Limit
(Abbreviations used here also correspond to other tables where applicable.)

Significant values are in bold*

In the CAD group, there was a notable association between the GG genotype and fast food consumption, whereas the GT genotype did not show a significant impact. In the Non-CAD group, neither genotype had a significant effect on fast food intake.

Table 8. Interaction between genotypes and Fast food consumption.

GROUP	GENE-DIET INTERACTION	OR	95%CL	P VALUE
CAD	GG VS fast food	0.0409	0.0077 to 0.2177	**0.0002**
	GT VS fast food	0.2188	0.0226 to 2.1137	0.1891
NON-CAD	GG VS fast food	0.3750	0.1058 to 1.3289	0.1286
	GT VS fast food	1.0000	0.3225 to 3.1006	1.0000

Significant values are in bold*

In the CAD group, a strong association was found between the GG genotype and high salt intake, while the GT genotype showed no significant effect. In the non-CAD group, neither genotype had a notable impact.

Table 9. Interaction between genotypes and high salt consumption.

GROUP	GENE-DIET INTERACTION	OR	95%CL	P VALUE
CAD	GG VS high salt	0.0247	0.0014 to 0.4511	**0.0125**
	GT VS high salt	0.2188	0.0226 to 2.1137	0.1891
NON-CAD	GG VS high salt	0.0036	0.1058 to 1.3289	0.1286
	GT VS high salt	1.6579	0.4051 to 6.7851	0.4820

Significant values are in bold*

The genotypes showed no significant effect on seafood consumption in either the CAD or Non-CAD groups.

Table 10. Interaction between genotypes and seafood consumption.

GROUP	GENE-DIET INTERACTION	OR	95%CL	P VALUE
CAD	GG VS seafood	0.2188	0.0226 to 2.1137	0.1891
	GT VS seafood	0.0625	0.0756 to 2.7550	0.3926
NON-CAD	GG VS seafood	1.6579	0.4051 to 6.7851	0.4820
	GT VS seafood	0.8512	0.2796 to 2.5912	0.7767

In the non-CAD group, a significant association was observed between the GT genotype and vegetarian diet, whereas the GG genotype had no significant effect. In the CAD group, neither genotype had a significant impact.

Table 11. Interaction between genotypes and vegetarian consumption.

GROUP	GENE-DIET INTERACTION	OR	95%CL	P VALUE
CAD	GG VS vegetarian	0.4565	0.0756 to 2.7550	0.3926
	GT VS vegetarian	3.5000	0.9206 to 13.3068	0.0660
NON-CAD	GG VS vegetarian	0.2188	0.0226 to 2.1137	0.1891
	GT VS vegetarian	5.6875	1.5098 to 21.4245	**0.0102**

Significant values are in bold*

In the CAD group, both the GG and GT genotypes were strongly associated with obesity, while in the non-CAD group, neither genotype showed a significant impact.

Table 12. Interaction between genotypes and obesity consumption.

GROUP	GENE-DIET INTERACTION	OR	95%CL	P VALUE
CAD	GG VS obesity	22.1538	2.5837 to 189.9581	**0.0047**
	GT VS obesity	5.1429	1.5220 to 17.3779	**0.0084**
NON-CAD	GG VS obesity	2.0870	0.1769 to 24.6160	0.5590
	GT VS obesity	1.7143	0.5245 to 5.6028	0.3724

Significant values are in bold*

DISCUSSIONS

In this study, the total sample size is 50 including 25 CAD and 25 non-CAD group. Descriptive statistical analysis was conducted on the study subjects and presented in Table 1. It shows that the sample was qualitatively heterogeneous with a broad age range and varying health conditions (such as abnormal glucose metabolism, poor lipid profiles, hypertension myocardial infarction and heart failure), which introduces variability in the primary data. The differences between the CAD and non-CAD groups are significant, emphasizing the need to interpret the findings with attention to the underlying health differences within the sample.

In Table 2, among the study subjects, the non-vegetarian group exhibited the highest prevalence, followed by the vegetarian and eggetarian groups. Specifically, in the CAD group, non-vegetarians were the most prevalent, followed by equal percentages of vegetarians and eggetarians. In contrast, in the non-CAD group, vegetarians were the most prevalent, followed by non-vegetarians and eggetarians, respectively. Utilizing the food frequency questionnaire (FFQ) to identify major dietary frequencies based on the highest consumption, four categories were delineated in (fig. 1). The “red meat-based” category, representing 80% of respondents, is characterized by a high intake of animal organ meats and fresh meat. The “fast food-based” category, constituting 100%, is typified by high-fat and high-carbohydrate items like burgers, fries, and sugary drinks. And “high salt-based diet” category involves frequent consumption of salt-rich foods like dry fish, pickles, pappads, fries, and chips. Lastly, the “seafood-based” category, representing 88%, encompasses various fish and shellfish varieties, including dry fish.

The study conducted correlation analysis, revealing significant associations between dietary habits and various health parameters. Specifically, high consumption of red meat and fast food showed positive correlations with LDL, VLDL, triglyceride (TGL), and total cholesterol (TC) levels, while displaying negative correlations with HDL levels. Conversely, seafood intake exhibited a positive correlation with HDL levels but negative correlations with TGL and VLDL levels. Moreover, a high-salt diet demonstrated positive correlations with systolic and diastolic blood pressure levels, as indicated in Table 3. Cahill LE et al. noted a strong link between fried food intake and type II diabetes mellitus, as well as moderate connections to coronary artery disease risk. These relationships were influenced by factors such as body weight, hypertension, and hypercholesterolemia (16).

The independent sample t-test demonstrated significant differences between CAD and non-CAD groups across various parameters, as outlined in Table 4. Babić Z. et al.’s findings indicate that patients with more narrowed coronary arteries tended to exhibit higher BMI and waist circumference. Furthermore, the waist-to-hip ratio showed associations with certain health markers with a significance level below 0.005, underscoring the study’s findings. Additionally, Alam et al. study found significant differences in serum cholesterol and HDL concentrations between Case and Control groups across all age groups ($P<0.0001$) in coronary heart disease patients proves the study (17, 18).

The independent T-test of lifestyle and sleep with CAD and non-CAD groups shows there is a significant difference in both lifestyle and sleep with $p<0.05$ given in Table 5.

The allele frequencies for the SNP in the ADIPOQ gene in both the CAD and non-CAD groups did not show significant deviation from Hardy-Weinberg equilibrium. Fisher’s exact test revealed a significant association between allele and genotype with CAD. In the genotype frequency distribution of the ADIPOQ gene in (Fig. 2), the TT wild-type was more prevalent in the non-CAD

group, while the GG mutant type was more common in the CAD group described in Table 6. This suggests that individuals with the GG genotype may have a higher risk of CAD compared to those with the TT genotype.

The results presented in Table 7 shows the link between genotypes and red meat intake in CAD and non-CAD groups. In the CAD group, the GG genotype was significantly less associated with red meat consumption (OR = 0.0741, 95% CI: 0.0085–0.6428, p = 0.0182), indicating that individuals with the GG genotype are less likely to consume red meat. The GT genotype showed no significant association (OR = 1.1953, p = 0.7654). In the non-CAD group, neither the GG nor GT genotypes were significantly linked to red meat intake. Janiszewska et al. discovered that increased intake of processed red meat may lower adiponectin levels, particularly among women, potentially mediated by BMI. This study underscores the link between diets high in red meat and saturated fats, such as typical Western diets, and reduced adiponectin levels (19).

In Table 9 and 10, there is a significant difference observed between the GG genotype and elevated intake of high-salt diet. However, no significant difference was found regarding seafood consumption and genotypes. The Table 11 demonstrate the potential link between the GT genotype and a vegetarian diet in non-CAD group, while no association was noted between GG genotype and vegetarianism in any group. This indicates a lower prevalence of the G allele among vegetarian consumers in the non-CAD group

Table 12 reveals that GG and GT genotypes were strongly linked to obesity in CAD group, while in the non-CAD group; neither genotype had a significant effect. Ogundele O. E., et al. studied ADIPOQ gene variations in young Nigerian adults, linking the rs266729 SNP to obesity indicators like BMI and waist circumference. The -11377G allele increased the risk of overall and abdominal fat, while the rs1501299 SNP showed no significant correlation with obesity measures (20). In non-CAD group, there is no significant association between genotypes and obesity.

Our study possesses several strengths and limitations. Notably, This South Indian study is pioneering in its exploration of the association between ADIPOQ genetic polymorphisms and dietary patterns in relation to CAD, marking a significant contribution to the field.

Nevertheless, the study is constrained by a relatively small sample size warranting larger studies for more robust results. Additionally, the cross-sectional design limits the assessment of temporal relationships, suggesting that a potential prospective study approach could yield more impactful findings. Furthermore, while our research focused on a single SNP, future investigations may benefit from exploring multiple SNPs to elucidate gene-gene interactions in larger populations.

CONCLUSIONS

The study explored the association between dietary patterns, genetic variations in the ADIPOQ gene, and obesity measures in CAD and non-CAD subjects. Dietary habits, including the consumption of red meat, fast food, seafood, and high-salt foods, showed significant correlations with lipid profiles and blood pressure levels. Furthermore, genetic variations in the ADIPOQ gene were associated with dietary preferences and obesity risk. Specifically, the rs2241766 GG genotype was linked to high red meat and fast-food consumption, while the GT genotype was associated with a vegetarian diet in the non-CAD group. In the CAD group, the GG and GT genotypes were significantly associated with obesity, indicating a potential genetic predisposition to obesity in CAD patients. These findings underscore the complex interplay between diet, genetics, and obesity in cardiovascular disease.

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CONFLICT OF INTEREST The authors declare no conflict of interest.

ETHICS APPROVAL Approval for this study was secured from the Ethics Committee of the Chettinad Hospital and Research Institute (approval ID: IHEC-I/2464/24).

CONSENT TO PARTICIPATE All participants in this study provided written informed consent.

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