THE EFFECTS OF GENETIC VARIANTS TOWARDS WEIGHT AND BIOCHEMICAL CHANGES IN WEIGHT MANAGEMENT PROGRAM

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Abstract

Genetic variation research indicates that 25% to 70% of body weight is determined by genetics. This study aimed to identify the influence of genetic variants on weight and biochemical data changes in participants who underwent a weight management program. A total of 30 obese participants were randomly assigned to either intervention or control groups. The study consisted of three phases: Phase I (pre-assessment), Phase II (intervention phase), and Phase III (post-assessment). The intervention and control groups were selected using block randomisation. The study involved 30 participants aged between 31 and 41 with a BMI of 32.8 ± 6.12 kg/m2. By examining the available data, it is possible to observe trends suggesting potential associations between certain genotypes and weight changes. Two specific variants, rs1726866 and rs1800497, significantly impacted glucose levels. Additionally, these two variants and another variant called rs1051168 were observed to influence cholesterol levels. These findings contribute to our understanding of the genetic factors that can potentially influence glucose and cholesterol metabolism and may have implications for personalised approaches to managing glucose and cholesterol-related conditions towards weight management programs.

Keywords: Obesity, Diet, Weight Management Program, Genotyping

Introduction

Obesity has become a significant public health burden worldwide in recent years, with many people being overweight or obese. Out of 50.1% of Malaysian adults, 30.4% were overweight, and 19.7% were obese (1). The prevalence of diet-related diseases and obesity are influenced by genetic and environmental factors such as over-nutrition and a sedentary lifestyle. Genetic variations can impact metabolic processes, including how the body utilises and stores energy from food. For example, certain variations in genes such as FTO (fat mass and obesity-associated gene) and MC4R (melanocortin 4 receptor gene) have been associated with increased hunger, reduced satiety, and a higher preference for high-calorie foods. These variations can make it more challenging for individuals to control their food intake and contribute to weight gain and obesity. Genetic variations may predispose individuals to obesity and affect weight loss and weight management strategies for those who are overweight or obese (2). The concept of nutrigenomics has emerged as awareness of food modifications to reduce the risk of obesity-related diseases has increased. Nutrigenomics refers to the interactions between nutrition and lifestyle factors on genetic expression. Changes in eating patterns and lifestyles have made people increasingly vulnerable to diet-related issues (3).

A person's risk for childhood obesity can increase if they have a family history of obesity. Studies have shown that parental obesity or overweight is associated with an elevated risk of childhood obesity, which can then be a predictor of adult obesity (4). Furthermore, research on genetic variation has demonstrated that approximately 25%–70% of body weight is genetically determined, and more than 600 chromosomes may play a role in the heritability of obesity (5). Heritability accounts for 40% to 70% of the variance in body mass index (BMI). Various factors, including gene-diet interactions and genetic variations influenced by ethnicity, environment, disease/condition, genes, genetic variants, or nutrients, can contribute to develop obesity-related diseases (6). The impact of a specific genotype on weight loss outcomes can vary significantly, even when different patterns of energy restriction or dietary changes are prescribed (5).

Nutrigenomics has generated considerable interest due to the potential for dietary modifications to improve health and reduce the risk of diet-related diseases (7).

According to the Centre for Disease Control and Prevention (CDC), genetic factors play a crucial role in the body's ability to adapt to environmental changes, and studies on family members, twins, and adoptees indicate that hereditary factors contribute significantly to variations in adult weight (8). Several genes, such as FTO, ADIPOQ, and MC4R, have been identified with variants that may increase the risk of obesity by influencing appetite and food consumption.

For instance, the fat mass and obesity-related gene (FTO, rs9939609) have been associated with a 30% higher risk of becoming overweight. FTO was the first obesity-related gene to be discovered, and research has revealed its association with obesity and an increased risk of various cancers across different racial groups using genome-wide association studies (GWAS) analysis (9).

GWASs have shed light on the role of genetic factors, including single nucleotide polymorphisms (SNPs), in an individual's susceptibility to obesity. These studies examine genetic variations across the entire human genome, including SNPs, copy number variations (CNVs), and other structural variants, using whole-genome sequencing analysis conducted by international genome projects. Among these variations, SNPs are humans' most common type of genetic variation (10).

Genetic factors, including specific gene variants and SNPs, have been identified as significant contributors to obesity susceptibility. The discovery of genes like FTO, along with advances in GWAS technology, has provided insights into the genetic underpinnings of obesity and its associated risks in diverse populations. This study aimed to identify the influence of genetic variants on weight and biochemical data changes in participants who underwent a weight management program.

Materials and Methods

Research design

An experimental design with an intervention-control approach was utilised for this study.

Study location

Health Clinic; Diet Care Centre, and Integrative Pharmacogenomics Institute, UiTM, Selangor Malaysia

Sampling

The study used quota sampling to select the sample, which does not require a survey frame or a list of all members of the population of interest. A total of 30 participants were selected. The sample was divided into two groups, an intervention group and a control group, each with n=15 participants, randomly assigned a number from 1-30 and equally divided into the two groups. To be eligible for participation in this study, individuals must meet certain inclusion criteria, which include being between 18 and 60 years old, being able to understand either the Malay or English language, having a BMI of more than 27.5 kg/m2, and working, studying, or residing in Selangor. Participants must also be willing to take part in the study voluntarily. However, individuals diagnosed with chronic diseases or pregnant mothers will be excluded from the study.

Measurement

This study used various instruments, including a questionnaire to gather information on socio-demographic data and a Food Frequency Questionnaire (FFQ) to assess the participants' dietary intake. Based on the information collected, the dietitian creates a personalised dietary plan for each participant. This plan considers their specific nutritional needs, caloric requirements, and weight management goals. The recommendations may include guidance on portion sizes, macronutrient distribution, meal planning, and food choices. The dietitian also educates the participants about the importance of balanced nutrition and offers practical tips for incorporating healthier food choices into their daily routines. Blood samples were collected for genotyping and biochemistry tests, such as fasting blood sugar (FBS) and fasting lipid profile (FLP).

Data collection

This study consisted of three phases, namely pre-assessment (Phase I), intervention phase (Phase II), and post-assessment (Phase III), conducted over a period of 10 weeks. Participants were briefed about the study protocol in Phase I and asked to provide their socio-demographic information. Their body weight was measured, and blood samples were collected to analyse FBS, FLP, and genotyping. Phase II involved the distribution of a food frequency questionnaire (FFQ) during the first week of the intervention phase to determine the participants' dietary habits and lifestyles. The FFQ used was from the National Health and Morbidity Survey. In Phase III, weight measurements were taken, and blood samples were collected for FBS and FLP analysis. The difference between the pre and post-assessment was then evaluated.

Laboratory analysis

Glucose and Lipid Fasting Profile

The nurse or qualified phlebotomist collected six (6) ml of blood from each participant from the venous vein. After 8 to 12 hours of fasting, blood was sampled. Participants were not allowed to eat or drink during the fasting period prior to the withdrawal of blood samples. Three (3) ml of blood was used for FLP, while another 3 ml was for FBS. All sample was analysed at the Department of Clinical Diagnostic Laboratories, UiTM Medical Specialist Centre.

Genotyping

Five (5) ml of blood was collected from the participants' venous veins. Genomic DNA was extracted from the EDTA anti-coagulated blood. Genotyping was conducted for all participants using validated allele-specific PCR assays. All samples were analysed at Integrative Pharmacogenomics Institute (iPROMISE), UiTM.

Ethical approval

Ethical approval was obtained from the UiTM Research Ethics Committee ref.no: (REC/07/2021 (FB/46).

Data analysis

The data from continuous variables were compared between the intervention and control groups using a two-sample t-test with independent samples. Pre- and post-intervention, the data of continuous variables were compared using paired samples T-test and two related sample tests. Normally distributed data were analysed by T-test. The non-parametric test was used to analyse data that was not normally distributed. Pearson or Spearman correlation was used to determine the correlation depending on the normality of the data.

Results

Demographic characteristics of respondents

Table 1 shows the mean and frequency of socio-demographic profiles of the participants for the intervention and control groups. The mean age of participants in the intervention group is 36.2 years, with a standard deviation of 4.90. This indicates that, on average, participants in this group are in their mid-thirties. In the intervention group, 50% of the participants are male, while the other 50% are female. In the control group, 46.7% are male, and 53.3% are female. These percentages indicate a relatively equal distribution of gender in both groups. The mean BMI in the intervention group is 32.8, with a standard deviation of 6.12. BMI is a measure of body fat based on height and weight, and a BMI of 32.8 suggests that, on average, participants in the intervention group are in the obese range.

Table 1: Demographic data

Socio-demographic category	Interventions group n (%)	Control group n (%)	Mean (SD)
Age			36.2 (4.90)
Gender			
Male	5 (33.3)	7 (46.7)	
Female	10 (66.7)	8 (53.3)	
Body Mass Index (BMI)			32.8 (6.12)

Dietary and pre-biochemical data between intervention-control group

Variations in the frequency of eating or drinking and the different types of meals or liquids between the control and intervention groups were compared (Table 2). Based on the findings, most dietary intake is low to moderate, with only vegetable intake high.

Table 2: Frequency of foods or drinks consumption

	Intervention	Control	
Frequency of Foods or Drinks Consumption	**Mean±SD**	**Mean±SD**	*p*[c]
N	**15**	**15**	
Cereals and cereals product (meals/week)[a]	2.87±1.13	3.08±0.99	0.97
Fast food consumption (meals/week)[b]	2.79±2.42	2.71±1.07	0.69
Meat and meats product (meals/week)[b]	2.42±1.74	3.36±1.80	0.15
Fish and sea foods (meals/week)a	2.77±1.70	3.49±1.64	0.26
Eggs (eggs/week)b	1.97±1.30	2.45±1.69	1.00
Legumes and legumes product (cups/week)[b]	2.85±1.53	1.96±1.37	0.24
Milk and milk products (glasses/week)[b]	2.34±1.89	2.47±1.90	0.74
Vegetables (cups/week)[b]	5.24±3.99	7.55±7.01	0.97
Fruits (cups/week)[a]	3.21±1.99	2.66±1.13	0.58
Drinks (glasses/week)[b]	1.42±0.79	2.07±1.67	0.79
Confectioneries (meals/week)[b]	3.72±2.59	2.60±1.20	0.92
Bread spreads (tablespoons/week)[b]	1.49±0.92	1.49±0.96	0.55
Flavors/ seasoning (tablespoons/week)[a]	3.32±1.72	3.73±2.05	0.40

[a] two-sample independent test.

[b] Man-Whitney Test.

[c] p-value, statistical significant $p < 0.05$

Correlation between pre-biochemical and post-biochemical data and dietary habit

Table 3 shows an analysis of the correlation between dietary habits and pre-biochemical and post-biochemical data by Pearson's correlation coefficient to determine the correlation between the variables involved. In the correlation analysis, the correlation of the data can be described as strong, moderate, fair and weak depending on its r and r_s values. The strong association indicate when

the r value > 0.75, followed by moderate 0.50 < r < 0.75, fair 0.25 < r < 0.50 and weak is when the r < 0.25.

Correlation of genetic variant with biochemical data and weight

Table 4 displays the distribution of weight and biochemical data pre-and post-test, categorised based on the genotypes of each SNP. The SNP rs1726866, associated with eating disinhibition, exhibits a notable difference in glucose levels between individuals with GG and GA alleles. Moreover, individuals with the GG genotype have higher cholesterol levels than those with the AA genotype. The GG allele for rs1800497 significantly affects glucose and cholesterol levels. In contrast, the pre-and post-test results show that individuals with the TT allele for rs9939609 have considerably lower cholesterol levels. Concerning obesity genotyping, individuals with CC alleles for rs17782313 have a significantly higher value for HDL levels in the pre-test of biochemical data than those with TT and TC alleles. However, there is no significant difference between the individual's genotyping and weight and biochemical data before and after the post-test for the other alleles.

Discussion

The genetic variations section of the study encompasses 14 types of genotyping that impact changes in biochemical data and weight between the two groups studied. Research has consistently shown that incorporating whole grains into one's diet reduces the risk of non-communicable diseases, including obesity, cardiovascular disease, type 2 diabetes, and colorectal cancer (10). On the other hand, consumption has been found to have limited or even detrimental effects on health outcomes. Studies have demonstrated that a higher intake of cereal or cereal products, especially those made from processed grains, is often associated with elevated glucose levels. Additionally, a higher intake of refined grains can raise blood sugar levels, especially in individuals already diagnosed with diabetes mellitus. However, based on the study's findings, there is only a fair and modest association between grain consumption and biochemical value, suggesting no correlation between grain intake and biochemical value. Although cereals may satisfy our sweet cravings, they also sabotage glucose levels. Many popular kinds of cereal have refined grains and sugars as their top ingredients, which provide little nutritional value and high empty calories that can increase blood glucose levels.

Additionally, previous research has shown strong evidence linking sugar-sweetened beverages to weight gain or obesity in children and adolescents (11). Our study also found a significant association between the consumption of sweetened beverages and triglyceride levels.

Previous research findings suggest that many individuals consume protein higher than the recommended daily allowance (RDA). This higher protein intake aligns with recommendations to promote muscle health, regardless of an individual's body weight or intake of calories, carbohydrates, or fats. Moreover, increased dietary protein consumption has been associated with a reduced risk of cardiometabolic disorders, particularly in individuals with obesity. Those who follow a diet rich in protein tend to have a lower body mass index (BMI) and waist circumference, along with higher levels of high-density lipoprotein (HDL) cholesterol than those who consume protein at the RDA level. These findings highlight the potential benefits of incorporating higher protein intake into a balanced diet for improved body composition and metabolic health (12).

The study reports that higher consumption of red meat was associated with an increased risk of hypercholesterolemia, hyper-LDL cholesterolemia, and dyslipidemia in both men and women, with a 34% and 10% increase in the risk of hypercholesterolemia, respectively. Similarly, a 58% and 17% increase in the risk of hyper-LDL cholesterolemia and dyslipidemia was observed in men consuming red meat. Processed meat intake was also associated with an increased risk of hypercholesterolemia, hypertriglyceridemia, and dyslipidemia in both men and women, with a 38% and 9% increase in the risk of hypercholesterolemia, respectively (13). The findings suggest that consuming meat and meat products can substantially increase cholesterol and LDL levels and validate the conclusions of prior research.

Based on the current study, egg intake has only a moderate correlation with triglyceride and LDL levels. In contrast, a previous study (14) found that consuming eggs can increase total cholesterol, LDL, and HDL levels. However, there was no significant influence on the LDL: HDL, TC: HDL, or triglyceride concentrations. The heterogeneity among studies may be due to differences in study design and participant response to dietary cholesterol. Additionally, the current study suggests that the moderate intake of meat, meat products, and eggs may not have a strong association with biochemical effects.

A negative correlation exists between vegetable intake and the two variables under consideration. A negative correlation means that when one variable increases, the other decreases, or vice versa. Evidence suggests that fruits and vegetables are associated with a lower risk of cardiovascular diseases, such as high blood pressure, cholesterol, triacylglycerol, and soluble fiber. As the average intake of vegetables is high, we might expect a significant correlation between vegetable intake and changes in biochemical data. However, since the correlation is insignificant, we may conclude that there is no substantial linear relationship between the population's biochemical data and vegetable intake (15).

The study revealed that the consumption of certain food categories was moderate to low, suggesting that the impact on biochemical values is likely to be minimal. However, high consumption of these food categories can significantly affect the values, particularly in individuals with underlying conditions such as diabetes mellitus, hyperlipidemia, or hypercholesterolemia. For those without underlying conditions, their values may not vary much after consuming

Table 3: Correlation of each type of food product intake with pre- and post-biochemical data

Type of food product	Glu pre-		Glu post-		Chol pre-		Chol post-		HDL pre-		HDL post-		TG pre-		TG post-		LDL pre-		LDL post-	
	r^a	p^b	r^a	p^b	r^a	p^b	r^a	p^b	r^a	p^b	r^a	p^b	r^a	p^b	r^a	p^b	r^a	p^b	r^a	p^b
Cereals and cereals	0.016	0.95	**-0.440**	0.04	0.315	0.15	-0.023	0.92	-0.175	0.44	-0.156	0.49	0.329	0.14	0.094	0.68	0.375	0.09	0.106	0.64
Fast food	0.145	0.52	-0.032	0.89	0.360	0.10	0.165	0.46	-0.040	0.86	-0.092	0.68	0.271	0.22	0.022	0.92	0.342	0.12	0.097	0.67
Meat and meats	0.034	0.88	-0.316	0.15	**0.622**	0.02	0.098	0.66	-0.081	0.72	-0.127	0.57	0.346	0.12	0.118	0.60	**0.518**	0.01	0.115	0.61
Fish and sea foods	-0.011	0.96	-0.260	0.24	0.217	0.33	-0.264	0.34	-0.155	0.49	-0.154	0.49	0.161	0.47	-0.013	0.96	0.227	0.31	-0.186	0.41
Eggs	0.131	0.56	-0.230	0.30	0.317	0.15	0.171	0.45	-0.036	0.88	-0.340	0.12	**0.495**	0.02	0.188	0.40	**0.442**	0.04	**0.416**	0.05
Legumes and legumes	0.057	0.80	0.256	0.25	0.322	0.14	-0.012	0.96	0.030	0.59	-0.095	0.67	0.359	0.10	0.233	0.30	0.329	0.14	0.027	0.90
Milk and milk products	0.012	0.96	-0.296	0.18	0.264	0.24	0.201	0.37	-0.063	0.78	-0.328	0.14	0.123	0.58	-0.023	0.92	0.181	0.42	0.378	0.08
Vegetables	-0.245	0.27	-0.202	0.37	-0.056	0.80	-0.084	0.71	-0.203	0.36	0.243	0.28	-0.097	0.67	0.148	0.51	-0.098	0.66	-0.214	0.34
Fruits	0.393	0.07	0.322	0.14	0.582	**0.005**	0.242	0.28	-0.174	0.44	-0.333	0.13	**0.449**	0.04	0.062	0.78	0.601	0.003	0.259	0.25
Drinks	0.330	0.13	-0.272	0.22	0.312	0.16	0.132	0.56	0.059	0.79	0.009	0.97	**0.442**	0.04	0.079	0.85	0.393	0.07	0.136	0.55
Confectioneries	0.098	0.37	0.056	0.81	0.474	0.001	0.155	0.49	-0.115	0.72	-0.174	0.51	**0.247**	0.007	0.044	0.85	**0.461**	0.001	0.136	0.55
Bread spreads	0.107	0.64	-0.170	0.45	0.256	0.25	-0.197	0.38	-0.109	0.63	-0.062	0.78	**0.579**	0.005	**0.527**	0.01	0.307	0.17	-0.146	0.52
Flavours	-0.381	0.08	0.222	0.32	0.047	0.83	-0.057	0.80	0.116	0.61	**0.427**	0.05	0.145	0.52	0.128	0.57	-0.038	0.87	-0.307	0.17

Abbreviation: Glu = Glucose; Chol = cholesterol; HDL= High Density Lipoprotein, TG = triglyceride; LDL= Low Density Lipoprotein.

[a] indicate value of Pearson correlation.

[b] p-value, statistically significant $p < 0.05$.

Table 4: Mean pre and post-test of biochemical data and weight for all the participants involved

Genotyping	Alleles	Glu_pre	Glu_post	Chol_pre	Chol_post	HDL_pre	HDL_post	Tri_pre	Tri_post	LDL_pre	LDL_post	Wt_pre	Wt_post
	GG	5.65	***5.62**	6.57	***6.20**	1.5	1.46	1.73	1.28	4.33	4	31.33	29.33
rs1726866[a]	GA	5	***5.00**	5.15	4.72	1.53	1.58	0.99	0.74	3.2	3	34.1	33.6
	AA	4.93	5.04	5.31	5.38	1.41	1.55	1.1	1.07	3.33	3.33	32.89	32.89
	GG	5.33	***5.45**	6.89	***5.33**	1.34	1.36	1.74	1.18	5	3.5	32	30.75
rs1800497[a]	GA	5.09	5.1	5.07	5.17	1.48	1.49	1	0.86	3.07	3.36	34.79	34.36
	AA	4.69	4.76	5.14	5.1	1.62	1.94	1	1.02	3	2.75	29	29
	GG	5.66	5.48	4.12	4.38	1.51	1.5	1.07	0.81	3.8	2.5	34	33
rs1051168[a]	GT	5.06	4.96	5.87	5.25	1.43	1.42	1.32	0.94	3.78	3.6	33.4	33.8
	TT	4.97	5.1	5.43	5.28	1.5	1.6	1.09	0.97	3.47	3.27	33.07	32.33
rs9939609[b]	TT	5.16	5.06	***5.68**	***5.51**	1.43	1.49	1.24	1.06	3.77	3.69	32.46	32.15
	TA	4.91	5.16	5.03	4.73	1.56	1.64	0.98	0.77	2.89	2.67	34.33	33.56
rs2025804	GA	5.06	5.1	5.41	5.2	1.48	1.55	1.13	0.95	3.41	3.27	33.23	32.73
rs5400	GG	5.08	5.1	5.29	5.3	1.52	1.57	1.06	0.96	3.29	3.35	32.53	32
	GA	5	5.07	5.83	4.83	1.36	1.48	1.41	0.9	3.8	3	35.6	35.2
rs4680[b]	GG	5.08	5.08	5.47	5.04	1.51	1.56	0.83	0.9	3.5	3.19	34.31	33.81
	GA	5	5.15	5.26	5.6	1.4	1.52	1.09	1.07	3.17	3.5	30.33	29.83
rs8179183	GG	5.06	5.1	5.42	5.19	1.48	1.55	1.14	0.95	3.41	3.27	33.23	32.73
rs17782313[a]	TT	5.01	5.06	5.35	5.36	1.45	1.54	1.08	0.96	3.23	3.38	32	31.69
	TC	5.24	5.2	5.47	4.57	1.32	1.37	1.33	0.98	3.8	3	34.4	33.6
	CC	5	5.1	5.56	5.42	***1.79**	1.8	1.09	0.85	3.5	3.25	35.75	35
rs17366568	GG	5.06	5.1	5.42	5.2	1.5	1.55	1.14	0.95	3.41	3.27	33.23	32.73
rs4994[b]	AA	4.99	5.09	5.42	5.22	1.44	1.51	1.14	0.98	3.42	3.32	33.11	32.63
	AG	5.51	5.19	5.38	4.99	1.77	1.76	1.07	0.76	3.33	3	34	33.33
rs17300539	GG	5.06	5.1	5.41	5.19	1.5	1.55	1.14	0.94	3.41	3.27	33.23	32.73
rs1801282[b]	CC	5	5.09	5.3	5.13	1.51	1.58	1.07	0.93	3.3	3.2	32.9	32.4
	CG	5.6	5.26	6.61	5.72	1.18	1.22	1.76	1.11	4.5	4	36.5	36
rs7903146	CC	5.05	5.11	5.43	5.21	1.47	1.53	1.16	0.97	3.43	3.29	33.52	33

Abbreviation: Glu = Glucose; Chol = cholesterol; Tri = triglyceride; Wt = weight.

[A] one-way ANOVA/Kruskal-Wallis Test.

[b] T-test/Man Whitney test.

[*] significance, $p < 0.05$.

any meal. It is essential to note that the data was obtained through an FFQ, and any inaccuracies in completing the questionnaire may result in significant variations in the variables. Therefore, a better understanding of the questionnaire's completion procedures are critical in determining the association.

Several studies in human nutrition have demonstrated that TAS2R38 polymorphisms may affect food intake and nutritional status (16). The TAS2R38 gene is associated with food aversion and has a bitter taste. Individuals with GG and GA allele profiles often have a modified perception of bitter foods such as broccoli and cabbage and drinks like coffee. Super-tasters are less likely to enjoy leafy greens, broccoli, Brussels sprouts, dark chocolate, bitter beverages, dark roast coffee, and hot peppers, suggesting they cannot stop eating in response to stimuli. This variation in taste perception can lead to eating disinhibition. Individuals with the AA allele profile cannot tolerate bitter flavours, leading them to avoid bitter foods like vegetables and coffee. The study found that the control group consumed more vegetables than the experimental group. The results

show no significant changes in biochemical data between pre- and post- between the two groups. Weight reduction was recorded between the pre- and post-test. This may be due to other external factors such as education and socioeconomic status, family or peer influences, and other factors that affect how they eat and indirectly affect the participant's biochemical value and weight (17).

A previous study found that individuals homozygous for the TAQ1A polymorphism (rs1800497) had lower body fat and central adiposity, even after accounting for established variables affecting body composition. A dietary pattern characterised by increased energy consumption and a larger proportion of calories from sugar may contribute to the less healthy phenotype found in those carrying two TAQ1A risk alleles (18). However, the current study's results show a significant mean for TG in the intervention group and HDL value for the control group without significantly affecting other lipid profiles. While previous research suggests that carrying two alleles increases sugar consumption, the results of this study contradict that finding, as both groups consumed sweet foods and beverages in small amounts. It is essential to note that preliminary analysis suggests a larger sample population is needed to validate the TAQ1A allele's outcome.

The NMB (rs1051168) gene has been found to influence satiety signals. It has been linked to dietary disinhibition, sensitivity to hunger, and changes in fat mass. Previous study by Dotson et al. (19) has suggested that NMB may regulate eating behaviour and affect body weight. However, the results of this study indicate that individuals who carry homozygous NMB alleles only showed a small change in biochemical data and no significant differences in biochemical or weight measurements for both groups studied.

The FTO (rs9939609) gene is linked to how our bodies respond to different types of fats. People with this gene's TT and TA variations tend to do well on diets high in unsaturated and low in saturated fats. However, those with the AA variation usually have a more typical fat metabolism and are at a higher risk of obesity. This study confirms previous research that shows individuals with the A-A genotype have a higher risk of obesity than those with the A-T and T-T genotypes. Another study found that the FTO rs9939609 A-A genotype was more common in obese individuals than those with normal weight (20). Both groups in the study engaged in physical activity. However, there was a significant difference in the intervention and biochemical control values for people with the TT allele. This finding aligns with previous research on the AA allele, as no participants in the study had the AA allele.

The rs5400 gene is commonly associated with a preference for food high in sugar. Individuals with the GG allele typically have a strong preference for sugary food. In contrast, those with the GA allele have a slightly increased preference. However, in this study, both groups consumed only a moderate amount of sugary food and drink. External factors such as socioeconomic status or culture may have influenced dietary intake and obscured the trait of carrying these alleles. Furthermore, because all participants had only the GA and GG alleles, the reliability of the assertion is limited compared to individuals with the AA allele.

Homozygous individuals for the rs4680 allele (GG) have an increased tendency to overeat, as they struggle to feel full after eating, which can lead to obesity. However, the dietary intake scores for both groups in this study were only in the low to moderate range for all food categories. Therefore, it cannot fully support the genotype description. Additionally, while there were changes in the values of biochemical data and weight for the intervention group, there were only slight changes in biochemical values for the control group.

The MC4R gene, commonly associated with obesity, was also examined. The rs17782313 single nucleotide polymorphism in the MC4R gene has been debated regarding its role in obesity (21). Homozygous or compound heterozygous MC4R variations are associated with a higher prevalence of severe obesity than heterozygous variants, suggesting a codominant inheritance pattern (22). The analysis revealed that individuals with the CC allele are more likely to have a higher body mass index than those with the CT and TT alleles. The impact of the CT and TT alleles on body mass index remains unclear due to insufficient evidence. The analysis also showed that participants with the CC allele had a greater weight than those with the CT or TT alleles.

The ADRB3 gene, like the MC4R gene, is often associated with obesity. However, there is still uncertainty about the genetic impact of the ADRB3 rs4994 polymorphism on childhood and adolescent overweight/obesity, despite previous intervention studies (23). An individual with the AA allele has only the typical risk of childhood and adolescent overweight/obesity. On the other hand, those with the AG allele are more likely to have an increased risk of childhood and adolescent overweight/obesity. However, the intervention group participants' results challenge this assumption by showing lower weight than individuals with the AA allele. The assertion can be supported by the fact that the mean value of participants in the control group carrying the AG allele is higher than those with the AA allele. This suggests that the intervention received by the participants in the intervention group, which involved 10 weeks of module advice, may have contributed to the difference in mean values observed between the two groups.

The rs1801282 genotype is commonly associated with the risk of obesity. Evidence suggests that the PPARG SNPs, which are nuclear receptors, play a significant role in regulating glucose and lipid metabolism (24). Homozygous carriers of the GG allele are believed to have a higher risk of increasing their body mass index. However, weight loss and a reduction in body fat percentage can be achieved by consuming monounsaturated fats. However, the study's findings indicate that both groups only consist of individuals carrying the CC and CG alleles, which confer

a slightly elevated risk of increasing body mass index, with monounsaturated fats aiding in weight loss. Results demonstrate that participants carrying the CC allele have experienced changes in their lipid profile and glucose levels in both groups. However, those carrying the CG allele showed no significant changes in their mean values.

The genetic variant rs7903146 is associated with BMI and relates to type 2 diabetes risk. Individuals carrying the TCF7L2 rs7903146 TT genotype and who are obese have a 2.62-fold increased risk of developing type 2 diabetes compared to individuals with other genotypes (25). The analysis also revealed that both allele carriers in the intervention group experienced weight changes. However, as the control group only includes individuals with the CC allele, no differences were observed in this group. Participants with the CT allele had lower glucose levels than those with the CC allele. Moreover, based on the biochemical data, both groups' fasting blood glucose values are within the normal range.

Conclusion

In conclusion, we can see patterns that suggest connections between specific gene types and changes in weight. Two specific gene variants, rs1726866 and rs1800497, significantly affected glucose levels. Furthermore, these two variants and another variant called rs1051168 were found to influence cholesterol levels. These discoveries enhance our knowledge of genetic factors that can impact glucose and cholesterol metabolism and could have implications for tailoring approaches to managing glucose, cholesterol, and weight conditions in personalised weight management programs.

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Competing interests

The authors declare there is no conflict of interest.

Ethical clearance

The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Universiti Teknologi MARA (UiTM) (REC/07/2021 (FB/46).

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