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A High-Carbohydrate Diet Effects on the A Allele of Hepatic Lipase Polymorphism on the apoB100/apoAI Ratio in Young Chinese Males*

Wpływ wysokowęglowodanowej diety i allelu A polimorfizmu lipazy wątrobowej na wskaźnik apoB100/apoAI u młodych Chińczyków

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Abstract

Background. Diet induces changes in plasma lipid profiles, and the plasma lipid profiles vary among different genetic backgrounds.
Objectives. The aim of this study was to investigate how a high-carbohydrate (high-CHO) diet interacts with hepatic lipase G-250A promoter polymorphism to affect the ratios of plasma lipids and apolipoproteins (apo) in a young Chinese population.
Material and Methods. Experiments were conducted on 56 university students. A stabilization diet was given for 7 days and a high-CHO diet was followed for 6 days. The diets used in this study were described by Song et al. and the following parameters were evaluated: total plasma triglyceride (TG), total plasma cholesterol (TC), plasma high-density lipoprotein cholesterol (HDL-C), plasma low-density lipoprotein cholesterol (LDL-C), apoB100 and apoAI. The plasma lipid and apoB100/apoAI ratios were also calculated and hepatic lipase G-250A polymorphism was analyzed.
Results. At baseline, no significant difference was detected for subjects with different genotypes and genders. All the parameters showed significant differences before and after the high-CHO diet, and these differences are gender-specific: after the high-CHO diet, the TG/HDL-C ratios significantly increased in females (GG genotype: $P = 0.004$; A carriers: $P = 0.005$). The TC/HDL-C ratios significantly decreased in GG genotype males ($P = 0.007$), A carrier males ($P < 0.0001$) and A carrier females ($P = 0.016$) and the LDL-C/HDL-C ratios significantly decreased in the GG genotype males ($P = 0.011$), A carrier males ($P < 0.0001$) and A carrier females ($P = 0.001$). However, comparing the apoB100/apoAI ratio before and after the high-CHO diet, a significant difference only existed in male A carriers ($P = 0.009$).
Conclusions. The results of this study show that the high-CHO diet induces the positive effects on the lipid ratios in general, only except the TG/HDL-C ratio in females. Noticeably, the decreased apoB100/apoAI ratio is associated with the A allele of hepatic lipase G-250A polymorphism only in young Chinese males (**Adv Clin Exp Med 2012, 21, 6, 751–757**).

Key words: diet, dyslipidemia, hepatic lipase, cardiovascular diseases.

Streszczenie

Wprowadzenie. Dieta wywołuje zmiany w profilu lipidów w osoczu, a profile lipidów w osoczu różnią się w zależności od tła genetycznego.
Cel pracy. Zbadanie, jak wysokowęglowodanowa dieta oddziałuje na polimorfizm promotora G-250A lipazy w wątrobie i wpływa na iloraz lipidów w osoczu i apolipoprotein (APO) w młodej populacji chińskiej.

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Materiał i metody. Badania przeprowadzono na 56 studentach. Dietę stabilizacyjną podawano przez 7 dni, a wysokowęglowodanową przez następne 6 dni. Diety użyte w tym badaniu opisali Song et al. Oceniano następujące wskaźniki: triglicerydy całkowite w osoczu (TG), całkowity cholesterol w osoczu (TC), lipoproteiny o dużej gęstości w osoczu (LDL-C), lipoproteiny o małej gęstości (LDL-C), apoAI i apoB100. Obliczono również lipidy w osoczu i wskaźnik apoB100/apoAI. Analizowano polimorfizm G-250A lipazy w wątrobie.
Wyniki. Na początku badania nie stwierdzono istotnych różnic u osób z różnymi genotypami i różnej płci. Wszystkie parametry istotnie zmieniły się po zastosowaniu diety wysokowęglowodanowej, a różnice te są związane z płcią: po zastosowaniu diety wysokowęglowodanowej, wskaźnik TG / HDL-C znacznie zwiększył się u kobiet (genotyp GG: P = 0,004; nośniki: P = 0,005). Wskaźnik TC / HDL-C znacznie zmniejszył się u mężczyzn z genotypem GG (P = 0,007), u mężczyzn nośników A (p <0,0001) i kobiet nośników A (p = 0,016), a wskaźniki LDL-C/HDL-C znacząco zmniejszyły się u mężczyzn z genotypem GG (p = 0,011), u mężczyzn nośników A (p < 0,0001) i kobiet nośników A (P = 0,001). Porównując współczynnik apoB100/apoAI przed i po zastosowaniu diety wysokowęglowodanowej, istnieje znacząca różnica tylko), u mężczyzn nośników A (p = 0,009).
Wnioski. Wyniki tego badania wskazują, że wysokowęglowodanowa dieta ma korzystny wpływ na wskaźniki lipidów w ogóle, z wyjątkiem TG / HDL-C u kobiet. Warto zauważyć, że zmniejszenie ilorazu apoB100/apoAI jest związane z allelem A polimorfizmu G-250A lipazy w wątrobie tylko u młodych mężczyzn pochodzenia chińskiego (**Adv Clin Exp Med 2012, 21, 6, 751–757**).

Słowa kluczowe: dieta, dyslipidemia, lipaza wątroby, choroby układu krążenia.

Dyslipidemia accounts for at least 50% of cardiovascular disease (CVD)'s risk [1]. It was conventionally considered that the increases in total plasma triglyceride (TG), total plasma cholesterol (TC) and plasma low-density lipoprotein cholesterol (LDL-C) and the decrease in plasma high-density lipoprotein cholesterol (HDL-C) were the major factors causing CVD [2]. However, accumulating evidence has suggested that the HDL-C related ratios (TG/HDL-C, TC/HDL-C and LDL-C/HDL-C) might be better to predict CVD or as therapeutic targets for dyslipidemia [3, 4]. In addition, recent data has suggested that another new CVD marker that is even better than the HDL-C-related ratios in CVD risk assessment might be the apolipoprotein B100/apolipoprotein AI (apoB100/apoAI) ratio [5].

A high-carbohydrate (high-CHO) diet induces a multitude of biochemical changes on lipid metabolism. After digestion, carbohydrates can be converted into triacylglycerols and cholesterol mainly in the liver. Accumulating studies have shown that a high-CHO diet could cause hypertriglyceridemia [6, 7]. And it has also been shown that the lower plasma TC and LDL-C concentrations after the high-CHO diet can decrease CVD risk [6]. Although the effects of a high-CHO diet on lipid profile changes have been extensively reported in the past several decades, few have been reported on the lipid and alipoprotein ratios in subjects with different hepatic lipase gene polymorphism backgrounds. Since the Chinese diet contains more carbohydrates and less fat compared to other diets [8, 9], in order to develop a personalized diet suitable for this population, it is important to investigate its effects on different genetic backgrounds.

In lipid metabolism, hepatic lipase is a key enzyme that has monoglyceride, diglyceride, triglyceride hydrolase, and phospholipase AI activities [10]. Hepatic lipase is synthesized and secreted by the liver, and found extracellularly in the liver and in steroidogenic organs, primarily bound to proteoglycans [11]. Hepatic lipase catalyzes the hydrolysis of phospholipids and triglycerides in plasma lipoproteins, and is involved in the processing of several lipoproteins [12]. Hydrolysis of phospholipids and triglycerides in HDL by hepatic lipase may induce cholesterol and/or cholesteryl ester flux to the lipase-containing tissues [13]. The hepatic lipase gene is located in chromosome 15q21 and consists of nine exons [14]. Four different promoter polymorphisms have been identified (G-250A, C-514T, T-710C, and A-763G) [15]. The variants have been widely studied in relation to hepatic lipase activity and lipoprotein metabolism [16], however the interaction of G-250A polymorphism with a high-CHO diet on plasma lipid ratios has not been reported before.

Previous research has shown that either a high-CHO diet or hepatic lipase G-250A polymorphism can affect plasma lipid and lipoprotein profiles [17, 18]. In the current study, the authors were interested in investigating how the different genotypes of hepatic lipase G-250A polymorphism interact with a high-CHO diet to modify the plasma lipid and apolipoprotein ratios. This may help us find an approach to better reduce the risk of cardiovascular disease, and may lead to personalized dietary recommendations as an ultimate goal.

Material and Methods

The subjects consisted of 27 males and 29 females from the West China Medical Center (Table 1). The Human Research Ethics Committee of Sichuan University had approved the present protocol and

Table 1. Demographic and biochemical characteristics of the subjects

Tabela 1. Demograficzna i biochemiczna charakterystyka pacjentów

Variables (Zmienne)	Males (n = 27) (Mężczyźni)	Females (n = 29) (Kobiety)	All (n = 56) (Razem)
Age – years (Wiek – lata)	22.9 ± 1.9	22.8 ± 1.6	22.8 ± 1.8
Weight – kg (Masa ciała – kg)	62.9 ± 11.6	50.9 ± 7.0*	56.7 ± 11.2
Body mass index – kg/m^2 (Wskaźnik masy ciała – kg/m^2)	21.8 ± 4.1	20.2 ± 2.5	21.0 ± 3.4
Waist circumference – cm (Obwód w talii – cm)	74.8 ± 10.0	67.5 ± 6.2*	71.1 ± 9.0
Waist-to-hip ratio (Stosunek obwodu talii do bioder)	0.89 ± 0.06	0.83 ± 0.04*	0.86 ± 0.06
Heart rate – bpm (Częstość serca)	72.3 ± 10.4	74.0 ± 9.3	73.2 ± 9.8
Systolic blood pressure – mm Hg (Ciśnienie skurczowe)	117.0 ± 14.0	104.6 ± 6.4*	110.6 ± 12.3
Diastolic blood pressure – mm Hg (Ciśnienie rozkurczowe)	75.9 ± 12.2	67.9 ± 7.1*	71.7 ± 10.6
Triglyceride – mg/dL (Triglicerydy)	89.1 ± 55.6	65.2 ± 24.2*	76.5 ± 43.4
Total cholesterol – mg/dL (Cholesterol całkowity)	148 ± 21	156 ± 28	152 ± 25
HDL-C – mg/dL	56.0 ± 14.0	65.8 ± 13.0*	61.2 ± 14.3
LDL-C – mg/dL	81.5 ± 20.7	79.8 ± 21.5	80.6 ± 21.0
Apolipoprotein AI – mg/dL	193 ± 26	213.7 ± 15.9*	204 ± 23
Apolipoprotein B100 – mg/dL	65.8 ± 22.7	69.3 ± 18.3	67.7 ± 20.3
Glucose – mg/dL (Glukoza)	4.00 ± 0.53	4.01 ± 0.54	4.01 ± 0.53
Insulin – μU/mL (Insulina)	4.80 ± 3.72	5.37 ± 2.29	5.11 ± 3.03

Data is shown as mean ± SD.
* $p < 0.05$ for females vs. males (unpaired *t*-test).

Dane przedstawiono jako średnie ± SD.
* $P < 0,05$ dla kobiet *vs* mężczyzn (niesparowany test *t*)

Table 2. Composition of the stabilization diet and the high-CHO diet

Tabela 2. Skład diety stabilizacyjnej i wysokowęglowodanowej

Ingredients (Składniki)	Stabilization diet – 7 days (Dieta stabilizacyjna – 7 dni)	High-CHO diet – 6 days (Dieta wysoko-węglowodanowa – 6 dni)
Caloric intake (cal/d)	2289 ± 848	2999 ± 1110
Protein (% of total energy)	15.8 ± 1.8	16.2 ± 1.6
Carbohydrate (% of total energy)	54.1 ± 2.4	70.1 ± 2.8
Fiber (g/day)	11.6 ± 2.3	15.4 ± 3.6
Fatty acids (% of total energy)	30.1 ± 3.6	13.8 ± 1.4
Saturated fatty acids (% of total energy)	7.5 ± 0.9	3.6 ± 0.5
Monounsaturated fatty acids (% of total energy)	16.1 ± 1.4	7.3 ± 0.8
Polyunsaturated fatty acids (% of total energy)	6.4 ± 1.5	2.8 ± 0.3
Fatty acid composition		
Palmitic fatty acids (16:0) (% of total fatty acids)	15.9 ± 4.4	18.9 ± 5.8
Stearic fatty acids (18:0) (% of total fatty acids)	6.9 ± 1.3	7.4 ± 0.9
Palmitoleic fatty acids (16:1) (% of total fatty acids)	2.1 ± 0.7	2.0 ± 0.4
Oleic fatty acids (18:1) (% of total fatty acids)	30.7 ± 6.5	32.1 ± 3.7
Linoleic fatty acids (18:2) (% of total fatty acids)	13.2 ± 3.3	17.0 ± 5.1

Data is reported as mean ± SD. Calories from food intake were not restricted for each meal. The volunteers ate to satiation as usual in their daily life. The diets were supplied by the Department of Nutrition, West China Hospital, Sichuan University.

Dane podaje się jako średnią ± SD. Kalorie z pokarmu nie były ograniczone do każdego posiłku. Ochotnicy jedli do sytości, jak zwykle w życiu codziennym. Diety były dostarczane przez Zakład Żywienia, West China Hospital, Sichuan University.

a written consent was signed by all the subjects. Details of the experiment's design, blood collection and plasma analysis procedures have been described in authors' previous publication [19].

DNA Extraction and Genotyping

Blood was collected in tubes containing EDTA as an anticoagulant, and tubes were store at 4°C before genomic DNA isolation, which was carried out in less than 3 days from the time of collection. A DNAout kit (Tiandz, Mianyang, China) was used to isolate genomic DNA from peripheral blood leukocytes. Standard company protocol was strictly followed (http://www.tiangen.com). Genotyping was performed as described by Meng et al. [20]. A 411 bp sequence of the hepatic lipase gene was amplified by PCR by using oligonucleotide primers 5′- GGCAAGGGCATCTTTGCTTC-3′and 5′-GGTCGATTTACAGAAGTGCTTC-3′. DNA templates were denatured at 94°C for 5 minutes, and then PCR was subjected to 35 cycles, each consisting of 40 seconds of denaturation at 94°C, 40 seconds of annealing at 58°C, and 90 seconds of extension at 72°C. The PCR products were digested with 10 U of restriction endonuclease D*ra*I and the fragments were separated by electrophoresis on a 2% agarose gel. The GG genotype is homozygote for the absence of the site (band at 411 bp), GA genotype is heterozygote for the presence and absence of the site (bands at 411, 301 and 110 bp), and AA genotype is homozygote for the presence of the site (bands at 301 and 110 bp). Allele and genotype frequencies of HL G-250A polymorphisms are shown in Table 3.

Table 3. Allele and genotype frequencies of HL G-250A polymorphisms

Tabela 3. Allele i częstotliwość genotypu HL G-250A polimorfizmu

	Males (n = 27) n (%)	Females (n = 29) n (%)	All (n = 56) n (%)
Genotype frequencies (Częstotliwość genotypu)			
GG	9 (33.3%)	6 (20.7%)	15 (26.8%)
GA	15 (55.6%)	18 (62.1%)	33 (58.9%)
AA	3 (11.1%)	5 (17.2%)	8 (14.3%)
Allele frequencies (Częstotliwość alleli)			
G	0.61	0.52	0.56
A	0.39	0.48	0.44

No deviation was found from the Hardy-Weinberg equilibrium in the distribution of genotypes ($\chi^2 = 1.923$, $p = 0.165$).

Nie stwierdzono odchylenia od równowagi Hardy'ego-Weinberga w rozkładzie genotypów ($\chi^2 = 1{,}923$; $p = 0{,}165$).

Statistical Analysis

Four volunteers did not complete the study with good compliance because of personal reasons. Their data was excluded. All the results are expressed as mean ± SD. The normality of each variable was analyzed by Shapiro-Wilk test. An unpaired *t*-test was performed to calculate the differences between male and female subjects of the study population. Differences of variables between subjects were assessed by one-way analysis of variance (one-way ANOVA). Differences of the variables before and after high-CHO diet were assessed by two-tailed paired *t*-tests. Statistical significance was set at $P < 0.05$. All analyses were performed using SPSS 16.0 (SPSS Inc., USA).

Results

Baseline Lipid and Apolipoprotein Ratios According to the Hepatic Lipase G-250A Genotypes

Due to the small number of the homozygotes for the less common allele, persons with GA and AA genotypes were grouped together as the A carriers. There was no significant difference in the ratios between the GG genotype and the A carriers (ANOVA) at the beginning of the stabilization diet. And unpaired *t*-tests also did not indicate that there was any significant difference between the females and the corresponding males (Table 4).

After the High-CHO Diet, Changes in Total Caloric Intake and Changes In Plasma Lipid and Apolipoprotein Ratios

Compared to those before the high-CHO diet (Table 5), in all genders and genotypes, the total caloric intake ($P < 0.0001$) was significantly increased. Females have significantly lower total caloric intake (before the high-CHO diet: $P = 0.003$ for the GG genotype and $P < 0.0001$ for the A carriers; after the high-CHO diet: $P = 0.003$ for the GG genotype and $P < 0.0001$ for the A carriers) than the corresponding males (unpaired *t*-test). Comparing different genotypes, A carriers had

Table 4. Lipid and apolipoprotein ratios in plasma at baseline

Tabela 4. Lipidy i wskaźniki apolipoprotein w osoczu na początku badania

Variables (Zmienne)	Males (Mężczyźni)		Females (Kobiety)		All (Razem)	
	GG (N = 9)	A carriers (N = 18)	GG (N = 6)	A carriers (N = 23)	GG (N = 15)	A carriers (N = 41)
Age (years)	22.2 ± 2.0	23.3 ± 1.8	22.3 ± 1.2	22.9 ± 1.7	22.2 ± 1.7	23.1 ± 1.7
TG/HDL-C	2.00 ± 1.82	1.64 ± 1.66	1.07 ± 0.72	1.05 ± 0.55	1.63 ± 1.52	1.31 ± 1.19
TC/HDL-C	3.10 ± 1.17	2.29 ± 1.02	2.46 ± 0.34	2.41 ± 0.49	2.85 ± 0.96	2.36 ± 0.76
LDL-C/HDL-C	1.79 ± 0.88	1.41 ± 0.72	1.22 ± 0.30	1.25 ± 0.39	1.56 ± 0.74	1.32 ± 0.56
ApoB100/ApoAI	0.36 ± 0.15	0.28 ± 0.17	0.33 ± 0.08	0.32 ± 0.08	0.35 ± 0.12	0.30 ± 0.12

Data is shown as mean ± SD.

Dane przedstawiono jako średnie ± SD.

Table 5. Lipid and apolipoprotein ratios in plasma before and after the high-CHO diet

Tabela 5. Lipidy i wskaźniki apolipoprotein w osoczu przed i po zastosowaniu diety wysokowęglowodanowej

Variables (Zmienne)	Males (Mężczyźni)		Females (Kobiety)		All (Razem)	
	GG (N = 9)	A carriers (N = 18)	GG (N = 6)	A carriers (N = 23)	GG (N = 15)	A carriers (N = 41)
Age (years)	22.2 ± 2.0	23.3 ± 1.8	22.3 ± 1.2	22.9 ± 1.7	22.2 ± 1.7	23.1 ± 1.7
Caloric intake (cal/d)						
before	3153 ± 792	2899 ± 419	1942 ± 207‡	1564 ± 481‡	2668 ± 866	2150 ± 807#
after	4130 ± 1038*	3798 ± 549*	2544 ± 271*‡	2049 ± 630*‡	3496 ± 1135*	2817 ± 1058*#
TG/HDL-C						
before	2.18 ± 1.69	1.63 ± 0.88	1.05 ± 0.59	1.19 ± 0.39‡	1.73 ± 1.45	1.39 ± 0.68
after	2.27 ± 1.54	1.53 ± 0.87	1.32 ± 0.67*	1.39 ± 0.53*	1.89 ± 1.32	1.45 ± 0.69
TC/HDL-C						
before	2.98 ± 0.97	2.77 ± 0.80	2.27 ± 0.21	2.42 ± 0.38	2.70 ± 0.83	2.57 ± 0.62
after	2.39 ± 0.70*	2.27 ± 0.59*	2.23 ± 0.16	2.26 ± 0.39*	2.33 ± 0.54*	2.26 ± 0.48*
LDL-C/HDL-C						
before	1.47 ± 0.71	1.46 ± 0.64	1.12 ± 0.11	1.23 ± 0.39	1.33 ± 0.57	1.33 ± 0.52
after	1.06 ± 0.41*	1.06 ± 0.47*	0.99 ± 0.18	1.00 ± 0.30*	1.03 ± 0.33*	1.03 ± 0.38*
ApoB100/ApoAI						
before	0.36 ± 0.19	0.36 ± 0.15	0.30 ± 0.07	0.30 ± 0.08	0.34 ± 0.15	0.33 ± 0.12
after	0.35 ± 0.17	0.34 ± 0.15*	0.29 ± 0.09	0.30 ± 0.08	0.33 ± 0.14	0.32 ± 0.12*

Data is shown as mean ± SD.
Dane przedstawiono jako średnie ± SD.

* $p < 0.05$ for after the high-CHO diet vs. after stabilization diet (paired *t*-tests).
* P < 0,05 dla osób po diecie wysokowęglanowej vs po diecie stabilizacyjnej (sparowany test t).

# $P < 0.05$ for subjects with different genotypes (ANOVA).
# P <0,05 dla osób z różnymi genotypami (ANOVA).

‡ $p < 0.05$ for females vs. males (unpaired *t*-tests).
‡ p < 0,05 dla kobiet vs mężczyzn (niesparowany test t).

significantly lower total caloric intake than the GG genotype (before the high-CHO diet: P = 0.042; after the high-CHO diet: P < 0.0001).

For the plasma lipid and apolipoprotein ratios, both TC/HDL-C (P = 0.012 for the GG genotype and P < 0.0001 for the A carriers) and LDL-C/ /HDL-C (P = 0.005 for the GG genotype and P < 0.0001 for the A carriers) decreased significantly, and apoB100/apoAI decreased significantly in the A carriers (P = 0.043) in all the subjects. When gender was considered, the TG/HDL-C significantly increased in females (P = 0.004 for the GG genotype and P = 0.005 for the A carriers) after the high-CHO diet. And the TG/HDL-C (P = 0.039) significantly decreased in female A carriers compared to the corresponding male A carriers before the high-CHO diet. Compared to those before the high-CHO diet, TC/HDL-C decreased significantly for the GG genotype males (P = 0.007), the A carrier males (P < 0.0001), the A carrier females (P =

0.016), and also decreased for the GG genotype females, although not significantly; LDL-C/HDL-C decreased significantly for the GG genotype males ($P = 0.011$), the A carrier males ($P < 0.0001$), the A carrier females ($P = 0.001$), and also decreased for the GG genotype females, although not significantly. ApoB100/apoAI decreased significantly in the A allele males ($P = 0.009$) after the high-CHO diet intervention.

Discussion

Hepatic lipase is an enzyme that regulates LDL, IDL, and HDL particle metabolism [21]. It is known that hepatic lipase catalyzes the hydrolysis of TG and phospholipids in large, modifying LDL to form more atherogenic small, dense LDL particles [22]. High hepatic lipase activity is also associated with low HDL-C level, and hepatic lipase can convert large, TG-rich HDL2-C into small, dense HDL3-C [23]. Hepatic lipase activity is determined by diet, genotype, gender, visceral obesity, insulin resistance, etc. Approximately 20–30% of the individual variation in hepatic lipase activity is due to the presence of common polymorphisms in the hepatic lipase gene promoter [17]. The G-250A substitution in the hepatic lipase gene promoter has been found to be associated with modifications of plasma lipid concentrations and the risk of coronary artery disease [24, 25]. However, the interactions of this G-250A substitution with a high-CHO diet on the plasma lipid and apolipoprotein ratios have not been reported before. At the baseline, there was no significant difference in the ratios (Table 4). After the high-CHO diet, the apoB100/apoAI ratios of the A carriers were only significantly decreased in males but not in females. These results indicate that the A allele of the hepatic lipase G-250A polymorphism is associated with a lower apoB100/apoAI ratio in males on high-CHO diets. Apparently, the studied population's lipoprotein metabolisms did not get changed much by other genetic and environmental factors since this is a 6-day short-term diet intervention. Therefore, after the high-CHO diet intervention, the plasma lipid and apolipoprotein ratio changes were most likely because of the individuals' genetic background [19].

After the high-CHO diet, the apoB100/apoAI ratio significantly decreased only in the A allele carrier males but not in the GG genotype males (Table 5). It appears that only in males, the high-CHO diet induces the positive effect of the A allele on the apoB100/apoAI ratio. In females, it is noticeable that the high-CHO diet can negatively influence the TG/HDL-C ratios. Higher levels of estrogen might be an explanation. Estrogen can significantly inhibit the activity of lipoprotein lipase, an enzyme which plays the key role in TG hydrolysis [26].

The authors concluded that the high-CHO diet has general favorable effects on the TC/HDL-C and LDL-C/HDL-C ratios. However, a favorable apoB100/apoAI ratio was only induced in A carrier males, and the healthy young Chinese females might have higher risk of hypertriacylglycerolemia. Therefore, present data suggests that the male A carriers would be a good target for diet intervention but our findings need future larger sample studies. Once confirmed, it will lead to the ultimate goal of personalized dietary recommendations.

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