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Original Article

Independent Association of Plasma Triglycerides and Hyperhomocysteinemia: A Cross-Sectional Study

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SUMMARY

Background: Hyperhomocysteinemia (HHcy) and dyslipidemia are indicators of atherosclerosis, and despite limited data, studies have suggested an association between lipid metabolism and HHcy. This study investigated the correlation between plasma triglyceride (TG) and homocysteine (Hcy) levels in a relatively healthy Taiwanese adult population.
Methods: This cross-sectional study included 3615 participants aged 15–89 years who underwent health checkups at a single medical center in northern Taiwan between 2008 and 2018. TG values were natural log-transformed. HHcy was defined as a plasma Hcy level greater than 15.0 μmol/L. Multivariable logistic regression analyses were conducted to evaluate the association between TG levels and HHcy by adjusting for potential confounding factors such as sex, age, body mass index, smoking, alcohol consumption, blood pressure, plasma glucose, and lipid profile. Subgroup analyses were also conducted.
Results: Of the 3615 participants, the mean age was 53.0 years, with 53.4% being men. The mean plasma Hcy level was 8.9 ± 3.5 μmol/L, with 4.0% (145 subjects) exhibiting HHcy. The median (IQR) plasma TG level was 114 (81–165) mg/dL. Log_e triglycerides (lnTGs) showed an independent association with HHcy. In logistic regression analyses, lnTG levels demonstrated a significant positive correlation with HHcy (adjusted odds ratio = 1.61, 95% CI 1.05–2.45, p = 0.029). Subgroup analyses revealed a significant positive association between lnTG and an increased HHcy risk in those under 65 years of age.
Conclusion: This study demonstrated that elevated plasma TG levels are associated with a higher risk of HHcy, particularly in younger populations.

1. Introduction

Atherosclerotic cardiovascular disease (ASCVD) is the leading cause of death worldwide. Homocysteine (Hcy) is an amino acid produced in the body as part of normal metabolism. Hyperhomocysteinemia (HHcy), which is detectable in up to 30% of patients with coronary artery disease and 42% of patients with cerebrovascular disease, has emerged as an independent risk factor for ASCVD.[1,2] Prospective studies have revealed that an elevated plasma homocysteine (Hcy) concentration doubles the risk of cardiovascular disease.[3] Recent research in healthy populations also suggests an association between elevated Hcy levels and peripheral vascular disease.[4–7] In addition, HHcy has been recognized as a contributing risk factor for vascular-related cognitive impairment, dementia, and Alzheimer's disease.[8] Therefore, HHcy should be identified in patients with progressive or unexplained atherosclerosis and managed accordingly.[6] The estimated prevalence of HHcy is 5% to 7% in the general population.[9] Hcy levels tend to rise with advancing age and can be influenced by various habits, such as smoking, alcohol consumption, and sedentary lifestyle.[10] Strong associations with sedentary time were observed for triglyceride (TG).[11] The ATTICA study found that there was a significant 6% increase in plasma Hcy levels for every 20 mg/dL rise in TG (p = 0.01) in healthy adults.[12]

Hypertriglyceridemia affects approximately 10% of adults and shows notable regional variations in prevalence.[13] While low-density lipoprotein cholesterol (LDL-C) is well established as a central factor in the development of ASCVD, plasma TG levels have also been shown to play a crucial and independent role in predicting the risk of coronary heart disease and stroke, particularly in the Asia-Pacific region.[14] The PROCAM study revealed increased cardiovascular event risks with rising TG levels, persisting even after accounting for other major risk factors.[15] According to the latest American Heart Association/American College of Cardiology guidelines, hypertriglyceridemia is recognized as an additional risk factor for ASCVD.[16] One proposed hypothesis is that Hcy induces endoplasmic reticulum stress, leading to the activation of sterol regulatory element-binding proteins, which are involved in TG biosynthesis, ultimately resulting in increased TG levels.[17] Alternatively, additional studies show hy-

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pertriglyceridemia as part of insulin resistance, with links between elevated plasma Hcy and reduced insulin sensitivity.[18,19]

Both TG and Hcy are involved in the atherogenic inflammatory cascade, which is closely associated with atherosclerosis, plaque formation, and infarction.[19] While numerous studies have explored the role of HHcy as a risk factor for cardiovascular disease, relatively little research has focused on the lipid profile association with HHcy, specifically TG.[4,20] Few studies have reported that detecting TG is highly feasible and can be used to screen high-risk patients for HHcy, enabling early identification of cardiovascular risk and facilitating timely intervention.[4,19] Therefore, further research is needed to substantiate these findings. This study aimed to investigate the clinical significance and correlation between TG and HHcy in relatively healthy Taiwanese adults.

2. Materials and methods

2.1. Study population

This cross-sectional study was conducted at a single center in Taipei, Taiwan. Ambulatory subjects undergoing health checkups at MacKay Memorial Hospital's Medical Examination Center from 2008 to 2018 were included. The study protocol was evaluated and approved by the Human Research Ethics Committee of the MacKay Memorial Hospital (project research number 18MMHIS137). All patient identifiers have been removed or anonymized to prevent any possibility of reidentification, as indicated by the waiver granted by the institutional research board. Participants under 15 years old, those above 89 years old, and those with incomplete biochemical parameters were excluded. A total of 3615 participants were included in the analysis. All participants underwent measurements of anthropometric and biochemical parameters, including plasma Hcy levels, with recorded medical histories. Guided by a trained nurse, participants completed a questionnaire on demographic details, medical history, current medications, smoking, and alcohol consumption, along with measurements of blood pressure, body weight, and height. Participants were instructed to observe an 8-hour fasting period before venous blood collection. Blood biochemical tests, including Hcy, TG, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and fasting plasma glucose (FPG), were conducted using a Hitachi 7170 automatic analyzer.

2.2. Definition

Based on previously published studies, HHcy was defined as Hcy levels $\geq$ 15.0 μmol/L.[5,21] Participants were split using a 15.0 μmol/L cut-off for further analysis. In several studies investigating premature coronary heart disease, the cut-off point for age is set at 45 years,[22] while the National Institute on Aging defines individuals aged 65 years as elderly. In this study, BMI categories adhered to the Taiwan Ministry of Health and Welfare definitions: underweight (BMI < 18.5 kg/m^2), lean (BMI $\geq$ 18.5 and < 24.0 kg/m^2), overweight (BMI $\geq$ 24.0 kg/m^2 and < 27 kg/m^2), and obese (BMI $\geq$ 27.0 kg/m^2).[23]

2.3. Statistical analysis

Data were presented as numbers and frequencies with percentages for categorical variables. Normally distributed continuous parameters were reported as mean $\pm$ standard deviation (SD), while non-normally distributed continuous variables were described as median and interquartile range (median [IQR 25–75]). The TG values were naturally log-transformed to address the extreme right skewness in regression analysis. Univariate and multistep logistic regression analyses were performed to assess the association between TG and HHcy. The analysis included crude models (model 1) and adjusted models (models 2–4), which accounted for potential confounding factors, such as sex, age, BMI, smoking, alcohol consumption, hypertension (HTN), diabetes mellitus (DM), systolic blood pressure (SBP), diastolic blood pressure (DBP), FPG, TC, and HDL-C. Subgroup analyses were conducted to explore potential risk subgroups stratified by sex, age, smoking status, and alcohol consumption after adjusting for the aforementioned covariates. Interaction effects were tested using interaction terms in the models, with significance evaluated by the p-value for interaction. Statistical significance was set at $p < 0.05$. Statistical analyses were performed using the SPSS software (version 22.0; IBM Corp., Armonk, NY, USA).

3. Results

The baseline characteristics of the study participants are summarized in Table 1. A total of 3615 participants with a mean age of 53

Table 1
Clinical characteristics of the study population.

N	3615
Hcy (μmol/L), mean $\pm$ SD	8.9 $\pm$ 3.5
Median (IQR)	8.3 (6.7–10.3)
Min-Max	2.5–49.3
TG (mg/dL), mean $\pm$ SD	135.9 $\pm$ 87.4
Median (IQR)	114 (81–165)
Min-Max	26–1255
Sex, N (%)	
Men	1930 (53.4%)
Women	1685 (46.6%)
Age, mean $\pm$ SD, N (%)	
All	53.0 $\pm$ 11.6
< 20 years old	17 $\pm$ 1.7, 6 (0.2%)
$\geq$ 20, < 45 years old	33.5 $\pm$ 4.4, 465 (12.9%)
$\geq$ 45, < 65 years old	52.9 $\pm$ 6.6, 2597 (71.8%)
$\geq$ 65 years old	70.5 $\pm$ 4.8, 547 (15.1%)
BMI (kg/m^2), mean $\pm$ SD, N (%)	
All	24.3 $\pm$ 3.8
Underweight	17.3 $\pm$ 1.0, 133 (3.7%)
Lean	21.7 $\pm$ 1.4, 1672 (46.3%)
Overweight	25.3 $\pm$ 0.9, 1036 (28.7%)
Obese	29.7 $\pm$ 2.8, 774 (21.4%)
Smoking status, N (%)	
Current and former	1023 (28.3%)
Never	2592 (71.7%)
Alcohol consumption, N (%)	
Current and former	929 (25.7%)
Never	2686 (74.3%)
HTN, N (%)	877 (24.3%)
DM, N (%)	326 (9.0%)
SBP (mmHg), mean $\pm$ SD	124.7 $\pm$ 18.2
DBP (mmHg), mean $\pm$ SD	77.1 $\pm$ 11.0
FPG (mg/dL), mean $\pm$ SD	101.9 $\pm$ 24.9
TC (mg/dL), mean $\pm$ SD	204.5 $\pm$ 38.1
HDL-C (mg/dL), mean $\pm$ SD	
All	53.6 $\pm$ 15.8
Men	47.3 $\pm$ 12.6
Women	60.8 $\pm$ 16.0

Note: BMI, body mass index; DBP, diastolic blood pressure; DM, diabetes mellitus; FPG, fasting plasma glucose; Hcy, homocysteine; HDL-C, high-density lipoprotein cholesterol; HTN, hypertension; IQR, interquartile range; N, number of participants; SBP, systolic blood pressure; SD, standard deviation; TC, total cholesterol; TG, triglyceride.
BMI < 18.5 kg/m^2: underweight; $\geq$ 18.5, < 24.0 kg/m^2: lean; $\geq$ 24.0, < 27.0 kg/m^2: overweight; $\geq$ 27.0 kg/m^2: obese.

years (standard deviation (SD) 11.6) were included in the study. Among the participants, 1930 were men (53.4%) and 1685 were women (46.6%). The mean BMI was 24.3 kg/m^2 (SD 3.8); 28.3% of the participants were smokers (current or former), and 25.7% were alcohol consumers (current or former). The median (interquartile range (IQR)) plasma TG level was 114 mg/dL (81–165), and the mean plasma Hcy level was 8.9 μmol/L (SD 3.5).

The HHcy group comprised 145 patients (4.0%), with 125 men and 20 women. The age of the participants ranged from 43 to 70 years old (mean ± SD: 56.4 ± 13.6). In the HHcy group, the median (IQR) plasma TG level was 136 mg/dL (103–188), whereas in the normal Hcy group, it was 112 mg/dL (80–164). The two 15-year-old participants (Hcy levels: 5.14 and 6.50 μmol/L) and the one 17-year-old participant (Hcy: 8.17 μmol/L) did not meet the diagnostic criteria for HHcy.

The results of the univariate and multistep logistic regression analyses are presented in Table 2. Multistep logistic regression analyzed the independent association of HHcy with TG, adjusting for potential confounders. In Model 1, which represented the univariate analysis, $\log_e$ triglyceride (lnTG) showed a significant association with HHcy (adjusted odds ratio (aOR) = 1.95, 95% confidence interval (CI) 1.44–2.64, p < 0.001). In the subsequent models, adjustments were made to account for potential confounding factors. In Model 2, with age and sex as covariates, lnTG maintained a significant positive correlation with HHcy (aOR = 1.54, 95% CI 1.12–2.13, p = 0.009). In Model 3, additional adjustments were made for age, sex, and BMI. The association between lnTG and HHcy remained significant (aOR = 1.50, 95% CI 1.07–2.10, p = 0.020), indicating that BMI has a modest effect on the association. In Model 4, further adjustments were made for age, sex, BMI, smoking, alcohol consumption, HTN, DM, SBP, DBP, FPG, TC, and HDL-C. Even after adjusting for correlated plasma lipid profiles, lnTG remained positively and significantly associated with HHcy (aOR = 1.61, 95% CI 1.05–2.45, p = 0.029). This suggests that HHcy is independently associated with TG beyond other lipid factors.

Table 3 displays the association between elevated lnTG and HHcy in various subgroups. A significant age-dependent interaction was observed (P for interaction = 0.004), with elevated TG levels associated with hyperhomocysteinemia in participants aged < 65 years (odds ratio (OR) = 1.98, 95% CI 1.20–3.28; p = 0.008), but not in those ≥ 65 years (p = 0.61). In sex-stratified analysis, a nominally significant association was found in men (OR = 1.59, 95% CI 1.01–2.52; p = 0.047) but not in women (p = 0.90), with no significant interaction by sex (P for interaction = 0.81). No statistically significant subgroup effect was observed when stratified by smoking and alcohol consumption.

Table 2
Logistic regression for effects of TG ($\log_e$TG) on hyperhomocysteinemia.

	OR (95% CI)	p value
Model 1	1.95 (1.44–2.64)	< 0.001
Model 2	1.54 (1.12–2.13)	0.009
Model 3	1.50 (1.07–2.10)	0.020
Model 4	1.61 (1.05–2.45)	0.029

Note: CI, confidence interval; OR, odds ratio; TG, triglyceride.
Model 1: univariate analysis.
Model 2: adjusted for age and sex.
Model 3: adjusted for age, sex and body mass index.
Model 4: adjusted for age, sex, body mass index, smoking status, alcohol consumption, hypertension, diabetes mellitus, systolic blood pressure, diastolic blood pressure, fasting plasma glucose, total cholesterol, high-density lipoprotein cholesterol.

4. Discussion

This retrospective cross-sectional study, based on the health examination records of relatively healthy adults, revealed that elevated TG levels were independently and positively associated with an increased risk of HHcy. Our findings indicate that elevated TG levels are strongly associated with HHcy, particularly in participants aged < 65 years, suggesting that age may act as an effect modifier in this relationship. These results support the potential value of screening for HHcy in individuals with high TG levels, although further interventional studies are required to clarify the implications for treatment.

Most hypertriglyceridemia classifications follow the National Cholesterol Education Program Adult Treatment Panel III guidelines, which define normal TG levels as < 150 mg/dL, borderline high as 150–199 mg/dL, high as ≥ 200–499 mg/dL, and very high as > 500 mg/dL.[24] In this study, due to extreme right skewness, TG values were log-transformed for regression analysis. More covariates were adjusted for, including BMI, blood pressure, FPG, TC, HDL-C, smoking, and alcohol consumption, all previously linked to Hcy levels.[5,25]

Several studies have demonstrated that hypertriglyceridemia is considered one of the components of insulin resistance,[18] and numerous other studies have observed a correlation between plasma Hcy levels and a decrease in insulin sensitivity.[19] Systemic insulin resistance, by promoting dyslipidemia and other metabolic abnormalities, is part of the "proatherogenic milieu".[26] Various triglyceride-rich lipoproteins (TRLs), including very-low-density lipoprotein (VLDL-

Table 3
Subgroup analyses of the association between $\log_e$TG levels and hyperhomocysteinemia.

Subgroups	OR (95% CI)	p value	Cases/Subjects	P for interaction
Sex				0.81
Men	1.59 (1.01–2.52)	0.047	124/1923	
Women	1.08 (0.32–3.64)	0.90	20/1682	
Age				0.004
≥ 65 years old	0.81 (0.36–1.82)	0.61	45/543	
< 65 years old	1.98 (1.20–3.28)	0.008	99/3062	
Smoking				0.45
Yes	1.18 (0.72–1.96)	0.51	70/1019	
No	1.62 (0.89–2.95)	0.12	74/2586	
Alcohol consumption				0.51
Yes	1.63 (0.82–3.22)	0.16	58/927	
No	1.57 (0.91–2.73)	0.11	86/2678	

Note: BMI, body mass index; CI, confidence interval; OR, odds ratio; TG, triglyceride. Smoking: current or former smoker.
Data were analyzed using logistic regression models after adjusting for age, sex, and BMI.

C) and its remnants, hinder the atheroprotective and anti-inflammatory effects of HDL-C. Both TRLs and chylomicron remnants independently contribute to atherosclerosis, distinct from the impact of LDL-C.[27,28]

This study found that plasma TG levels were significantly associated with HHcy, which aligns with findings from a health examination database study involving 817 participants in Southern Taiwan.[4] A study conducted in India also demonstrated a positive relationship between plasma Hcy and TG, as well as VLDL-C levels, in subjects with coronary artery disease.[29] In China, this was subsequently validated by a positive correlation between plasma TG and Hcy in many studies of Chinese patients undergoing physical examination[5,30] and community populations.[21,31] However, it is worth noting that not all prior studies have found consistent correlations between HHcy and lipid profiles. For example, Yadav et al. observed no significant correlation between plasma Hcy and TG in patients with ischemic heart disease.[32] Likewise, another study found no significant association between Hcy and plasma lipids among patients with DM.[33] Differences in demographic characteristics, including age, sex, race, geographic regions, physical activity levels, and various confounding factors may explain discrepancies across studies.[5,33]

A defining threshold for HHcy in adolescents is the 90th percentile at 8.23 μmol/L, nearly half the level observed in adults (15.0 μmol/L).[34] Hcy levels increase with age, irrespective of sex.[35] Although it is possible that estrogens might lead to reduced Hcy levels, the specific mechanisms by which estrogen achieves this effect are still unknown.[35] A recent study utilizing logistic regression analysis demonstrated an independent association of Hcy levels with age, men, vitamin B12 and folate levels, and the metabolic syndrome.[36] As one component of the metabolic syndrome, hypertriglyceridemia was specifically focused on. In this study, the stronger association between elevated TG and HHcy in men, consistent with previous findings, and in those under 65 years suggests a potential clinical benefit of maintaining normal TG and Hcy levels in the non-elderly. However, causal inference cannot be drawn from observational data, and further interventional studies are needed to confirm this relationship.

Smokers tend to have significantly higher levels of VLDL-C, TC, and plasma TG than non-smokers.[37] Additionally, tobacco smoke boosts interleukin-6 production, reducing cystathionine beta-synthase activity in Hcy metabolism.[38] On the other hand, alcohol-induced hypertriglyceridemia results from heightened VLDL-C secretion and impaired lipolysis.[39] Elevated ethanol intake inhibits methionine synthase gene expression, impacting the re-methylation process and hindering methionine formation from Hcy.[40] Due to the significant influence of smoking and alcohol on both TG and Hcy, our subgroup analysis, stratified by smoking and alcohol consumption, revealed no significant differences while remaining consistent with the primary outcome.

Our study has several limitations. First, the retrospective and cross-sectional design raises concerns about reverse causation. Second, we did not measure plasma folic acid and vitamin B12 levels, which regulate Hcy. Third, binary classification of smoking and alcohol consumption oversimplifies exposure by ignoring recovery post-cessation, recency of quitting, and exposure intensity or duration. Fourth, uncollected covariates like diet, vitamin B supplementation, and physical activity may influence the Hcy-lipid association. Additionally, the single-center, Taiwanese population focus limits external validity. Lastly, the mechanisms linking TG and Hcy remain unclear. Future large-scale, prospective studies are needed to clarify this association and explore underlying mechanisms.

5. Conclusion

This study demonstrated that elevated plasma TG levels are associated with an increased risk of HHcy, particularly in younger populations. Further studies are warranted to evaluate its predictive value for cardiovascular and cerebrovascular events and to provide more comprehensive insights into its relevance in preventive and therapeutic strategies.

Declaration of conflict of interest

The authors declare that they have no competing financial and non-financial interests.

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