

# A Correlation Study Between Non-alcoholic Fatty Liver Disease and Serum Triglyceride Level After an Oral Fat Tolerance Test

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## Research Article

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# Abstract

**Background:** Non-alcoholic fatty liver disease (NAFLD) has become one of the most common chronic liver diseases worldwide. High postprandial serum lipid concentrations have been reported in patients with metabolic syndrome. Meanwhile, postprandial triglyceride (TG) was found to be a potential replacement when fasting TG is not available. We aimed to investigate the relationship between postprandial TG concentration during oral fat tolerance testing (OFTT) and NAFLD.

**Methods :** A total of 472 Chinese adults, aged 25 to 65 years enrolled in this study. All the participants underwent OFTT. Serum triglyceride and other lipid concentrations were measured, and their relationships with NAFLD were analyzed.

**Results:** Of the 472 participants, 155 were diagnosed with NAFLD. The fasting and postprandial TG concentrations of the participants with NAFLD were higher than those of healthy participants ( $P<0.05$ ). The TG concentrations of the healthy participants peaked 4 h postprandially, whereas those of the participants with NAFLD peaked 6 h postprandially and had higher peak values. Postprandial triglyceride concentration showed significant association with higher risk of NAFLD.

**Conclusions:** Higher postprandial TG concentration is positively related to higher risk of NAFLD, and postprandial TG concentrations of NAFLD patients were higher than healthy individuals with a delayed peak time. Hence, 4h postprandial TG maybe a potential indicator for NAFLD.

**Trial registration number:** ChiCTR1800019514

## Highlights

- An oral fat tolerance test with a large sample was firstly conducted in a Chinese cohort.
- Postprandial serum triglyceride concentration is positively correlated with higher risk of non-alcoholic fatty liver disease.
- Non-alcoholic fatty liver disease patients have higher lipid responses after given a high fat load than healthy individuals and the peak time delayed.

## 1. Introduction

Non-alcoholic fatty liver disease (NAFLD) is the most common liver disease worldwide. It is a clinicopathological syndrome which is characterized by excessive lipid deposition in hepatocytes, and can progress from non-alcoholic fatty liver (NAFL) to non-alcoholic steatohepatitis (NASH) and more severe diseases, featuring hepatocellular injury and fibrosis, including cirrhosis and hepatocellular carcinoma [1-2]. The proportion of cases of hepatocellular carcinoma that are caused by NAFLD has been increasing rapidly worldwide since 2000, and NAFLD has become the most frequent reason for liver transplantation in USA [3-5]. Thus, the early identification of people who are more susceptible to NAFLD

and the identification of patients in the early stages of NAFLD are of great importance for the prevention and treatment of NAFLD.

The exact mechanism of development of NAFLD is as yet unclear, but various theories exist, including the “double-hit” and “multiple-hit” hypotheses. The “double-hit” hypothesis, which was first proposed by Day, states that excessive lipid deposition in hepatocytes caused by insulin resistance, hyperlipidemia, type 2 diabetes (T2DM), and metabolic syndrome leads to steatosis, which induces inflammation and fibrosis [6]. Hence, abnormal lipid metabolism plays an important role in the development of NAFLD. Dyslipidemia in Asians manifests as a low serum high-density lipoprotein-cholesterol (HDL-C) concentration and a high triglyceride (TG) concentration [7].

Fasting lipid profile is most commonly used in clinical practice. However, non-fasting lipidemia, such as postprandial TG concentration is also important for the assessment of lipid metabolism in an individual. Previous studies have suggested that postprandial lipemia is positively associated with chronic cardiovascular disease (CVD) and NAFLD [8-12] and that postprandial hyperlipidemia is a risk factor for T2DM[13]. The dyslipidemia that characterizes T2DM typically consists of high fasting and postprandial TG concentrations in the form of triglyceride-rich lipoproteins, high apolipoprotein B (ApoB) levels, low HDL-C, and a large number of small dense low-density lipoprotein particles, and these defects are a major cause of the associated high CVD risk[14].Furthermore, atherogenic dyslipidemia persists after the return of low-density lipoprotein cholesterol (LDL-C) to target levels during treatment and contributes to residual vascular risk[15].A study analyzing postprandial lipidemia response in the CORDIOPREV study shows that the postprandial response increases progressively according to non-diabetic, prediabetic and type 2 diabetic state and it is higher in patients with liver insulin-resistance[16]. Other studies have shown that high non-fasting plasma TG concentration is a strong and independent predictor of CVD and all-cause mortality and that cholesterol or remnant cholesterol concentrations are strong predictors of CVD[17-19].

High postprandial TG can be the result of the overproduction, with or without lower catabolism, of TG-rich lipoproteins, which is a consequence of the interaction of pathogenic genetic variants and coexisting medical conditions, particularly obesity, insulin resistance, or T2DM [20]. The postprandial accumulation of lipoproteins also contributes to the induction of oxidative stress, inflammation, and endothelial dysfunction, which increase the risk of NAFLD.

Recent studies have shown that dietary composition affects postprandial metabolism [21-22]. A comparison of the effects of a Western-style high-fat diet, a Western-style high-carbohydrate diet, and a Mediterranean diet on lipid and glucose metabolism showed that an energy-rich diet is associated with hyperglycemia, hyperlipidemia, an inflammatory response, and reductions in the levels of antioxidant markers [21]. Another study showed that meals that are high in saturated fat and carbohydrate affect the postprandial lipidemia of men with high waist circumferences (WC) [22]. However, although various studies have shown a role for postprandial lipidemia, the definition of hypertriglyceridemia varies in different guidelines [23-24]. A joint consensus statement from the European Atherosclerosis Society (EAS) and European Federation of Clinical Chemistry and Laboratory Medicine suggested that non-

fasting TG levels in subjects whose fasting TG < 1.7mmol/L are expected to be below 2.0mmol/L (175mg/dL) [25]. Meanwhile, an expert panel statement in Greece defined TG concentrations  $\leq 2.5$  mmol/L (220 mg/dL) at all time-points after an oral fat tolerance test (OFTT) as a desirable postprandial response [26]. Therefore, to further explore the relationship between hypertriglyceridemia and the metabolic abnormalities of NAFLD, it is important to establish a standardized oral fat tolerance test (OFTT) and explore the correlation between postprandial triglyceride concentration and NAFLD.

In the present study, we aimed to assess the relationship between postprandial TG concentration and the incidence of NAFLD by analyzing the postprandial TG concentration during an OFTT, in order to evaluate the potential role of postprandial lipidemia in NAFLD.

## 2. Methods

### 2.1 Study population

We randomly recruited 472 volunteers at the Endocrinology Department of Hebei General Hospital, China, between May 2018 and December 2019. The study complied with the principles of the Declaration of Helsinki, the protocol was approved by the ethics committee of Hebei General Hospital, and all the participants gave their written informed consent. The study has been registered with the China Clinical Trial Registry (registration number: ChiCTR1800019514, registration date: November 15, 2018. <http://www.chictr.org.cn/index.aspx>).

Vegetarians; patients with malignant tumors, CVD, diabetes mellitus, thyroid dysfunction, kidney disease, hematologic disease, infectious disease, or psychiatric disorders; individuals who had experienced stroke or had been pregnant in the preceding 3 months; those who were taking drugs that influence lipid metabolism and inflammation, including fish oil, contraceptives, hormones,  $\beta$  receptor blockers, or diuretics; and patients who had experienced serious infection, surgery, trauma, or a body mass change of >3 kg were excluded. After providing their written informed consent, physical examination and oral glucose tolerance testing (OGTT) were performed in all the participants. All the participants also underwent OFTT, as described below.

### 2.2 Oral fat tolerance testing

All the participants were asked to stick to their normal diet for 1 week before commencing the study. All the participants were studied in Hebei General Hospital after a 10-h overnight fast. Venous blood was collected in the morning, and then the participants consumed a high-fat meal within the following 10 min. The high-fat meal was prepared by professional dieticians, and provided 1,500 kcal in total, with fat, protein, and carbohydrate contents of 60%, 20%, and 20%, respectively. Blood samples were collected 2 h, 4 h, 6 h, 8 h, and 10 h after the consumption of the high-fat meal and the derived serum samples were stored at  $-80^{\circ}\text{C}$  (Haier MDR-382E, China). During this 10-h period, subjects were allowed to drink only water and prohibited to smoke, drink wine or eat any food. Strenuous exercises were not recommended, and only slow walking was allowed.

### *2.3 Measurement of anthropometric, clinical, and biochemical parameters*

Serum glucose, total cholesterol (TC), TG, HDL-C, LDL-C, ApoA1, and ApoB concentrations were measured by laboratory technicians in the Physical Examination Center of our hospital using a Hitachi 7600 automatic biochemical analyzer (Instrument Hitachi Ltd., Tokyo, Japan). Fasting plasma insulin concentration (FINS) was measured by chemiluminescence in the Nuclear Medicine Department of our hospital. Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated using the fasting insulin and glucose (FPG) concentrations by dividing insulin ( $\mu\text{U/ml}$ ) and glucose ( $\mu\text{mol/l}$ ) by 22.5 [27] ( $\text{HOMA-IR} = \text{FBG}(\text{mmol/L}) \times \text{FINS}(\text{mIU/L}) / 22.5$ ). Body mass index (BMI) was calculated as body mass divided by height squared ( $\text{kg/m}^2$ ).

Body mass, height, WC, and blood pressure were measured by trained professionals.

### *2.4 Diagnosis of non-alcoholic fatty liver disease*

Abdominal ultrasonography was performed by a specialist technologist in the Physical Examination Center of our hospital. A diagnosis of NAFLD was made according to The Guidelines for the Prevention and Treatment of Non-alcoholic Fatty Liver Disease (2018 update) [28] and the position statement on NAFLD/NASH based on the EASL 2009 special conference [29]. Subjects with fatty liver and without of alcohol abuse (men with alcohol consumption  $<30$  g/day, and women with alcohol consumption  $<20$  g/day) were diagnosed as having NAFLD [29].

Ultrasonographic examinations were performed by a specialist technologist. Diffuse fatty liver was diagnosed if two of the following were present: (1) diffuse enhancement of the near-field echo of the liver ("bright liver") and far-field echo attenuation; (2) unclear intrahepatic duct structure; (3) gradual attenuation of the far-field echo; and (4) mild-to-moderate hepatomegaly with a blunt leading edge.

### *2.5 Funding statement*

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

### *2.6 Statistical analysis*

SPSS 21.0 software (SPSS Inc, IBM Corporation, Armonk, New York, USA) was used for statistical analysis. Numerical data were tested for normality using the Shapiro-Wilk test. Normally distributed data are expressed as mean  $\pm$  standard deviation and non-normally distributed data are expressed as median and interquartile range. The independent sample t-test was used to compare data between two groups. Pearson's chi-square test was used to compare the prevalence of NAFLD among the groups. Single-factor ANOVA was used to compare data among three groups. Binary logistic regression analysis, with NAFLD as the dependent variable, was used to determine the influence of each parameter on the prevalence of NAFLD. Statistical significance was accepted at  $p < 0.05$ .

### 3. Results

All participants ingested the high-fat meal during the OFTT and it was well tolerated.

#### *3.1 Basic characteristics*

There were 472 participants in the present study, of whom 224 were male and 248 were female. The mean age of the men was  $45\pm 13$  years old and that of the women was  $44\pm 13$  years old (Table 1).

The participants were allocated to the two groups according to their liver ultrasonographic findings. The BMI, WC, systolic blood pressure, and diastolic blood pressure of the NAFLD group were higher than those of the control group ( $P<0.01$ ). The FPG, FINS, TC, TG, LDL-C, ApoB, and HOMA-IR of the NAFLD group were higher than those of the control group ( $P<0.001$ ). The HDL-C and ApoA1 concentrations and the ApoA1/ApoB ratio in the NAFLD group were lower than in the control group ( $P<0.001$ ,  $P=0.001$ , and  $P<0.001$ , respectively) (Table 1).

The differences in the BMI, FBG, FINS, HOMA-IR, TC, TG, HDL-C, LDL-C ApoA1, ApoB, and ApoA1/ApoB of the NAFLD and control groups were affected by gender. The BMI, FBG, FINS, and HOMA-IR of male and female participants with NAFLD were higher than those of the same gender in the control group ( $P<0.001$ ). The fasting TC, TG, and LDL-C concentrations in the NAFLD group were higher than those in the control group among participants of the same gender, but HDL-C was lower than that in the control group ( $P<0.05$ ) (Table 2).

#### *3.2 Circulating lipid concentrations at time points during the OFTT*

The TC, TG, and LDL-C concentrations in the fasting state and 2 h, 4 h, 6 h, 8 h, and 10 h after the start of the OFTT were higher in the NAFLD group than in the control group, whereas the HDL-C concentrations were lower ( $P<0.001$ ) (Table 3).

#### *3.3 Triglyceride peak concentration and time*

We next constructed a graph of the TG concentration at time points during the OFTT. The TG in each group increased gradually after the ingestion of the high-fat meal in OFTT. In the control group, the TG peaked at 4h postprandial and had returned to near the fasting concentration after 10 h. In contrast, the TG of the NAFLD group peaked 6h postprandially and had not returned to the fasting concentration by 10h postprandially ( Figure 1).

#### *3.4 Comparison of parameters in groups with differing fasting triglyceride concentrations*

According to their fasting triglyceride concentration, all the participants were allocated to one of two groups. Those with fasting TG concentrations  $\leq 1.7$  mmol/L were placed in the normal-triglyceride group (NFTG group) and those with TG  $>1.7$  mmol/L were placed in the high fasting triglyceride group (HFTG group). The BMI, FBG, FINS, and HOMA-IR of the HFTG group were higher than those of the NFTG group

( $P < 0.001$ ). The incidence of NAFLD in our participants is 32.8%. Pearson's chi-square test showed that the incidence of NAFLD in the HFTG group was higher than that in the NFTG group ( $P < 0.001$ ) (Table 4).

### *3.5 Risk factors for NAFLD*

The effects of BMI, WC, TC, TG, FBG, FINS, HOMA-IR, HDL-C, LDL-C, and fat load on NAFLD were determined using binary logistic regression analysis, and the factors that influenced or protected against NAFLD were further clarified by the creation of a forest plot. The parameters that were shown to be associated with the prevalence of NAFLD were BMI, WC, TG, 2-h postprandial TG, 4-h postprandial TG, HDL-C, LDL-C, and HOMA-IR (Table 5, Model 1, Figure 2A). After adjusting age and gender, factors listed above are still associated with NAFLD (Table 5, Model 2, Figure 2B). In order to exclude the effect of fasting TG, we further adjusted age, gender and fasting TG, result showed that higher postprandial TG concentration still associated with higher risk of NAFLD (Table 5, Model 3, Figure 2C).

### *3.6 Characteristics of participants with differing levels of fat tolerance*

The participants were allocated to three groups according to their fasting and 4-h postprandial TG concentrations. 4h postprandial triglyceride concentration higher than 2.5mmol/L was defined as high postprandial triglyceride (HTG) [26].

Those with normal fasting and postprandial TG concentrations were placed in the NFT group, those with normal fasting but HTG were placed in the impaired fat tolerance (IFT) group, and those with high fasting TG concentration were placed in the fasting hypertriglyceridemia (FHT) group.

Across the three groups, BMI, WC, FINS, HOMA-IR, TC, TG, and LDL-C all increased with decreasing fat tolerance, whereas HDL-C decreased with decreasing fat tolerance ( $P < 0.05$ ). The age and FPG of the IFT and FHT groups were higher than those of the NFT group ( $P < 0.05$ ), but there were no significant differences in age or FPG between the IFT and FHT groups. In addition, the incidence of NAFLD increased as the fat tolerance decreased ( $P < 0.001$ ) (Table 6).

## **4. Discussion**

We found that NAFLD is very common among the recruited group of participants of Han ethnicity with a prevalence of 32.8%. By using binary logistic regression, several risk factors such as BMI, WC and TG were found to be associated with NAFLD including TG after fat load in an OFTT. After adjusted age and gender, those with higher postprandial TG concentration still have higher risk of NAFLD (TG2h: OR, 1.94, 95%CI:1.62-2.32; TG4h: OR, 1.55,95%CI:1.37-1.76).

By analogy with OGTT, we used the fasting and 4h postprandial TG concentrations as criteria to define fat tolerance. We allocated all the participants to three groups of different fat tolerance according to fasting and postprandial TG concentration. According to the Greece consensus [26], we considered postprandial triglyceride concentration at 4h after ingesting the high fat meal in the OFTT higher than 2.5mmol/L as high postprandial triglyceridemia. Among participants with fasting TG concentration

within range while postprandial TG higher than the desirable level, incidence of NAFLD was higher than those with normal fasting and postprandial TG concentration. Thus, only detecting fasting TG in our daily clinical routine may cause some NAFLD patients missed diagnosis. Bring postprandial TG into routine physical examination may help detect NAFLD patients in early stages which may lead to better clinical outcomes.

Furthermore, the use of a 4-h postprandial value to define hypertriglyceridemia is consistent with practices reported by multiple authors in other countries [30-33]. A study assessing the determinant of postprandial TG in healthy young adults found a strong correlation between 4 h postprandial TG and fasting TG and came to the conclusion that 4 h postprandial TG might potentially replace fasting TG when measurement of fasting TG is not feasible [34].

A meta-analysis conducted by Mihas et al showed that a high TG concentration 4 h after a fat load is indicative of an excessive response [30]. The meta-analysis also suggested that a fat load of 70–79 g is the optimal dose. However, the high-fat meal administered in the present study contained 100g fat. Different fat load may affect postprandial lipid, and different ethnicity of the participants may affect result as well. However, though the meta-analysis only included studies of Caucasians and the participants in our study were all Chinese, the TG concentration also peaked at 4 h postprandially in our study. To determine the most suitable time point of OFTT in Chinese people, further studies are required.

However, the definition of high postprandial triglyceridemia is still unclear. The criteria used in the present study was higher than that after a daily meal recommended by the EAS consensus statement [25] and that after a daily Chinese breakfast in overweight people [35] while lower than that in another Mexican study [36]. These disparities may result from different ethnical groups and difference in dietary calories and fat content.

Participants with NAFLD were found to have higher BMI, WC, SBP, DBP, and HOMA-IR, which is consistent with the findings of a previous study [37]. In addition, by analyzing the characteristics of participants with differing fasting TG concentrations, we found that BMI is higher in individuals with high fasting concentrations. The previous study by Nogueira et al. showed that overweight and obesity are associated with a high prevalence of dyslipidemia [38], and another study showed that obese and insufficiently active male adolescents are more likely to have the metabolic syndrome [39].

High TC, TG, and LDL-C concentrations are risk factors for CVD [40], whereas HDL-C has protective effects [41]. HDL-C is mainly involved in the reverse transport of cholesterol from extrahepatic tissues to the liver. It can also inhibit the uptake of LDL-C by arterial smooth muscle cells and prevent the accumulation of cholesterol in cells. Therefore, HDL-C has anti-atherosclerotic effects and is often used as a marker of CVD risk [42]. In the present study, we found that NAFLD patients have abnormal lipid metabolism featuring higher level of TC, TG, LDL-C and lower level of HDL-C. ApoB regulates the secretion of very low-density lipoprotein, which is present at high circulating concentrations in patients with obesity, T2DM, and metabolic syndrome. Therefore, the high ApoB concentration in the NAFLD group is further evidence of dyslipidemia in patients with NAFLD.

The TG concentration peaked later in the NAFLD group, which may imply that the meal-induced change in serum TG is larger in NAFLD patients than that in normal individuals. An impairment in TG metabolism is also suggested by the long period of time taken for the TG concentration to return to baseline following a high-fat meal.

Dyslipidemia is the principal etiologic factor in NAFLD in non-diabetic patients [43]. A previous study showed that dyslipidemia is common in lean non-diabetic NAFLD patients, with similar lipid profiles being identified to those of overweight and obese NAFLD patients [44]. This study also showed that in non-diabetic patients, high TG and low HDL-C concentrations may influence the development of NAFLD.

There are some limitations in our study. The results from this study cannot be extrapolated to other populations, since this study only include Chinese individuals. In a clinical trial, it is difficult to match participants for differences in the age and gender. However, after allocated the participants to different groups according to gender, NAFLD patients still showed higher fasting lipid profiles than the control group. Meanwhile, after adjusted for age and gender, 4h postprandial triglyceride was still positively related to higher risk of NAFLD. Nevertheless, the strengths of this study also warrant acknowledgement. To the best of our knowledge, a standardized OFTT with a large sample size to assess the relationship between postprandial TG and NAFLD was firstly conducted in a Chinese cohort. In this study, 4h postprandial triglyceride is significantly related to NAFLD. The time point of 4h postprandial is in line with a previous study which observed strong correlation between 4 h postprandial TG and fasting TG.

## 5. Conclusion

In conclusion, our study highlighted that people with 4h postprandial TG concentration were significantly and positively related to NAFLD. Meanwhile, subjects with high postprandial TG concentration had a higher prevalence of NAFLD. 4h postprandial TG maybe a potential indicator for NAFLD.

## Abbreviations

NAFLD: non-alcoholic fatty liver disease; NAFL: non-alcoholic fatty liver; NASH: non-alcoholic steatohepatitis; CVD: chronic cardiovascular disease; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; WC: waist circumference; FPG: fasting plasma glucose; FINS: fasting plasma insulin concentration; HOMA-IR: homeostasis model assessment of insulin resistance; TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein-cholesterol; LDL-C: low-density lipoprotein-cholesterol; ApoA1: apolipoprotein A1; ApoB: apolipoprotein B; Con: control group; NFTG: normal fasting triglyceride group; HFTG: high fasting triglyceride group; NFT: normal fat tolerance; IFT: impaired fat tolerance; FHT: fasting hypertriglyceridemia; TG2h: 2-h postprandial triglyceride; TG4h: 4-h postprandial triglyceride; 95% CI: 95% confidence interval.

## Declarations

## **Ethics approval and consent to participate**

The study complied with the principles of the Declaration of Helsinki, the protocol was approved by the ethics committee of Hebei General Hospital, and all the participants gave their written informed consent.

## **Consent for publication**

Not applicable

## **Availability of data and materials**

The datasets generated in this study and the protocol are available on reasonable request. Please contact the corresponding author.

## **Declaration of interest**

The authors declare no conflicts of interests.

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## **Author contributions**

Xiaoyu Hou: Formal analysis, Writing-Original draft; Yunpeng Guan: Formal analysis, visualization; An Song, Jiajun Zhao, Shuchun Chen, Limin Wei and Huijuan Ma: Conceptualization, Supervision; Yong Tang, Luping Ren: Methodology, Supervision, Writing-Reviewing; Guangyao Song: Conceptualization, Methodology, Supervision, Project administration, Writing-Reviewing and Editing. Xiaoyu Hou and Yunpeng Guan contributed equally to this manuscript.

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## Tables

**Table 1.** Basic characters of the participants

Group	Total n=472	Con n=317	NAFLD n=155	P
Age(Year)	44± 13	43± 14	47± 11 <sup>##</sup>	<0.001
Gender(Male/Female)	224/248	139/178	85/70	0.992
BMI(kg/m <sup>2</sup> )	25.93± 4.05	24.45± 3.25	28.97± 3.83 <sup>##</sup>	<0.001
SBP(mmHg)	127± 15	124± 15	133± 14 <sup>##</sup>	<0.001
DBP(mmHg)	78± 10	77± 9	83± 10 <sup>##</sup>	<0.001
WC(cm)	87.9± 11.9	83.9± 10.9	96.1± 9.4 <sup>##</sup>	<0.001
FBG(mmol/L)	5.41(5.07,5.87)	5.29(4.97,5.61)	5.78(5.38,6.46) <sup>##</sup>	<0.001
FINS(mmol/L)	10.65(7.35,15.24)	8.89(6.43,11.84)	16.15(11.19,19.90) <sup>##</sup>	<0.001
HOMA-IR	2.60(1.70,3.82)	2.09(1.47,2.95)	4.12(3.01,5.71) <sup>##</sup>	<0.001
TC(mmol/L)	4.72± 1.01	4.59± 0.96	5.00± 1.06 <sup>##</sup>	<0.001
TG(mmol/L)	1.66± 1.31	1.34± 1.07	2.32± 1.50 <sup>##</sup>	<0.001
HDL-C(mmol/L)	1.25± 0.28	1.30± 0.29	1.14± 0.22 <sup>##</sup>	<0.001
LDL-C(mmol/L)	2.98± 0.73	2.85± 0.69	3.23± 0.74 <sup>##</sup>	<0.001
ApoA1(g/L)	1.40(1.24,1.56)	1.42(1.27,1.60)	1.34(1.20,1.51) <sup>#</sup>	=0.001
ApoB(g/L)	0.79± 0.22	0.75± 0.20	0.88± 0.23 <sup>##</sup>	<0.001
ApoA1/ApoB	1.78(1.48,2.22)	1.94(1.61,2.36)	1.57(1.28,1.86) <sup>##</sup>	<0.001

Data are mean ±standard deviation or median (interquartile range).

#P<0.05, compared with the Con group. ##P<0.01, compared with the Con group.

**Abbreviations:** BMI: body mass index; WC: waist circumference; SBP: systolic blood pressure; DBP: diastolic blood pressure; FPG: fasting plasma glucose; FINS: fasting plasma insulin concentration; HOMA-IR: homeostasis model assessment of insulin resistance; TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein-cholesterol; LDL-C: low-density lipoprotein-cholesterol; ApoA1: apolipoprotein A1; ApoB: apolipoprotein B.

**Table 2.** Comparison of anthropometric, clinical, and fasting metabolic parameters in participants of each sex in the NAFLD and control groups

	Con		NAFLD	
	Male(n=139)	Female(n=178)	Male(n=85)	Female(n=70)
Age (Year)	45± 14	42± 13	44± 12	51± 10
BMI (kg/m <sup>2</sup> )	25.57± 3.17	23.58± 3.04	29.29± 3.55 <sup>##</sup>	28.58± 4.14 <sup>##</sup>
FBG (mmol/L)	5.40(5.07,5.79)	5.22(4.93,5.54)	5.79(5.40,6.47) <sup>##</sup>	5.78(5.37,6.49) <sup>##</sup>
FINS (mmol/L)	9.40(6.48,12.34)	8.63(6.14,11.60)	16.40(11.17,20.57) <sup>##</sup>	15.33(11.45,19.34) <sup>##</sup>
HOMA-IR	2.09(1.56,3.07)	2.07(1.39,2.76)	4.28(3.04,5.86) <sup>##</sup>	4.07(2.97,5.67) <sup>##</sup>
TC (mmol/L)	4.43± 0.86	4.71± 1.02	4.82± 0.99 <sup>#</sup>	5.21± 1.10 <sup>#</sup>
TG (mmol/L)	0.99± 0.30	0.86± 0.26	1.57± 0.48 <sup>##</sup>	1.78± 0.77 <sup>##</sup>
HDL-C (mmol/L)	1.18± 0.26	1.39± 0.29	1.06± 0.16 <sup>##</sup>	1.24± 0.24 <sup>##</sup>
LDL-C (mmol/L)	2.79± 0.60	2.90± 0.76	3.14± 0.71 <sup>##</sup>	3.34± 0.78 <sup>##</sup>

Data are mean ±standard deviation or median (interquartile range).

#P<0.05, compared with the Con group. ##P<0.01, compared with the Con group.

Abbreviations: Con: control group; BMI: body mass index; FPG: fasting plasma glucose; FINS: fasting plasma insulin concentration; HOMA-IR: homeostasis model assessment of insulin resistance; TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein-cholesterol; LDL-C: low-density lipoprotein-cholesterol.

**Table 3.** Lipid metabolism parameters during the oral fat tolerance test

		0h	2h	4h	6h	8h	10h
Con	TC	4.59± 0.96	4.56± 0.94	4.55± 0.96	4.70± 0.98	4.75± 0.99	4.70± 0.95
	TG	1.34± 1.07	2.20± 1.27	2.65± 1.75	2.59± 2.03	2.33± 2.04	1.57± 1.62
	HDL-C	1.30± 0.29	1.30± 0.29	1.24± 0.28	1.24± 0.29	1.29± 0.30	1.28± 0.29
	LDL-C	2.85± 0.69	2.78± 0.67	2.72± 0.65	2.80± 0.67	2.84± 0.68	2.88± 0.67
NAFLD	TC	5.00± 1.06 <sup>##</sup>	4.96± 1.03 <sup>##</sup>	5.05± 1.07 <sup>##</sup>	5.22± 1.09 <sup>##</sup>	5.24± 1.10 <sup>##</sup>	5.21± 1.20 <sup>##</sup>
	TG	2.32± 1.50 <sup>##</sup>	3.47± 1.59 <sup>##</sup>	4.32± 2.04 <sup>##</sup>	4.50± 2.54 <sup>##</sup>	4.25± 2.61 <sup>##</sup>	2.96± 2.45 <sup>##</sup>
	HDL-C	1.14± 0.22 <sup>##</sup>	1.15± 0.22 <sup>##</sup>	1.09± 0.21 <sup>##</sup>	1.07± 0.22 <sup>##</sup>	1.11± 0.22 <sup>##</sup>	1.10± 0.22 <sup>##</sup>
	LDL-C	3.23± 0.74 <sup>##</sup>	3.14± 0.72 <sup>##</sup>	3.09± 0.70 <sup>##</sup>	3.16± 0.69 <sup>##</sup>	3.20± 0.70 <sup>##</sup>	3.28± 0.75 <sup>##</sup>
Total	TC	4.72± 1.01	4.69± 0.99	4.72± 1.02	4.87± 1.05	4.91± 1.05	4.87± 1.06
	TG	1.66± 1.31	2.62± 1.50	3.20± 2.01	3.22± 2.38	2.96± 2.41	2.03± 2.04
	HDL-C	1.25± 0.28	1.25± 0.28	1.19± 0.27	1.19± 0.28	1.23± 0.29	1.22± 0.28
	LDL-C	2.98± 0.73	2.90± 0.71	2.84± 0.69	2.92± 0.70	2.96± 0.71	3.01± 0.72

Data are mean ±standard deviation.

# $P<0.05$ , compared with the control group. ## $P<0.01$ , compared with the control group.

**Abbreviations:** Con: control group; NAFLD: Non-alcoholic fatty liver disease; TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein-cholesterol; LDL-C: low-density lipoprotein-cholesterol;

**Table 4.** Comparison of the characteristics of the NFTG and HFTG

	Total	NFTG	HFTG	P
	n=472	n=322	n=150	
Age	44± 13	43± 14	47± 11	0.176
Gender(male/female)	224/248	133/189	91/59	0.084
BMI	25.93± 4.05	24.94± 3.76	28.06± 3.85**	<0.001
FBG	5.41(5.07,5.87)	5.32(5.00,5.73)	5.60(5.34,6.31)**	<0.001
FINS	10.65(7.35,15.24)	9.55(6.59,12.72)	13.85(10.20,18.83)**	<0.001
HOMA-IR	2.60(1.70,3.82)	2.24(1.52,3.11)	3.70(2.60,5.07)**	<0.001
TC	4.72± 1.01	4.50± 0.93	5.19± 1.02**	<0.001
TG	1.66± 1.31	1.04± 0.32	2.98± 1.62**	<0.001
HDL-C	1.25± 0.28	1.30± 0.28	1.14± 0.24**	<0.001
LDL-C	2.98± 0.73	2.82± 0.69	3.32± 0.70**	<0.001
NAFLD	155(32.8%)	64(19.9%)	91(60.7%)**	<0.001
Non-NAFLD	317(67.2%)	258(80.1%)	59(39.3%)	<0.001

Data are mean ±standard deviation, median (interquartile range), or number (percentage).

\* $P<0.05$ , compared with the NFT group. \*\* $P<0.01$ , compared with the NFT group.

**Abbreviations:** NFTG: normal fasting triglyceride group; HFTG: High fasting triglyceride group; BMI: body mass index; FPG: fasting plasma glucose; FINS: fasting plasma insulin concentration; HOMA-IR: homeostasis model assessment of insulin resistance; TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein-cholesterol; LDL-C: low-density lipoprotein-cholesterol; NAFLD: non-alcoholic fatty liver disease.

**Table 5.** Odds ratio of impacting factors for NAFLD

Variables	Unstandardized Coefficient (B)	SE	Odds ratio (95% CI)	P value
<b>Model 1</b>				
BMI	0.39	0.04	1.48(1.37-1.61)	<0.001
WC	0.13	0.01	1.13(1.10-1.16)	<0.001
TC	0.40	0.10	1.50(1.23-1.82)	<0.001
TG	0.74	0.11	2.09(1.67-2.61)	<0.001
TG2h	0.68	0.09	1.97(1.65-2.35)	<0.001
TG4h	0.46	0.06	1.59(1.41-1.79)	<0.001
HDL-C	-2.37	0.42	0.09(0.04-0.21)	<0.001
LDL-C	0.73	0.14	2.07(1.56-2.73)	<0.001
FBG	1.00	0.15	2.72(2.03-3.65)	<0.001
FINS	0.21	0.02	1.23(1.17-1.29)	<0.001
HOMA-IR	0.81	0.09	2.24(1.88-2.67)	<0.001
<b>Model 2</b>				
BMI	0.40	0.04	1.49(1.37-1.62)	<0.001
WC	0.15	0.02	1.17(1.13-1.21)	<0.001
TC	0.38	0.11	1.46(1.18-1.81)	<0.001
TG	0.69	0.11	2.00(1.60-2.49)	<0.001
TG2h	0.66	0.09	1.94(1.62-2.32)	<0.001
TG4h	0.44	0.06	1.55(1.37-1.76)	<0.001
HDL-C	-2.51	0.46	0.08(0.03-0.20)	<0.001
LDL-C	0.69	0.15	1.99(1.47-2.69)	<0.001
FBG	0.94	0.16	2.56(1.88-3.47)	<0.001
FINS	0.22	0.03	1.25(1.19-1.31)	<0.001
HOMA-IR	0.82	0.09	2.28(1.90-2.72)	<0.001
<b>Model 3</b>				
TG2h	0.63	0.15	1.88(1.40-2.54)	<0.001
TG4h	0.32	0.09	1.37(1.14-1.65)	<0.05

Model 1-Crude OR; Model 2- Adjusted age and gender; Model 3- Adjusted age, gender and fasting triglyceride.

**Abbreviations:** BMI: body mass index; WC: waist circumference; FPG: fasting plasma glucose; FINS: fasting plasma insulin concentration; HOMA-IR: homeostasis model assessment of insulin resistance; TC: total cholesterol; TG: triglyceride; TG2h: triglyceride 2h postprandially; TG4h: triglyceride 4h postprandially; HDL-C: high-density lipoprotein-cholesterol; LDL-C: low-density lipoprotein-cholesterol; 95% CI: 95% confidence interval.

**Table 6.** Characteristics of different fat tolerance groups

	NFT	IFT	HTG
	n=192	n=130	n=150
Age	40±13	48±13 <sup>##</sup>	47±11 <sup>##</sup>
Gender(male/female)	66/126	63/67	91/59
BMI	24.18±3.68	26.07±3.60 <sup>##</sup>	28.06±3.85 <sup>##**</sup>
WC	82.43±10.96	88.24±11.32 <sup>##</sup>	94.71±9.79 <sup>##**</sup>
FBG	5.32±0.56	5.72±1.36 <sup>#</sup>	6.00±1.29 <sup>##</sup>
FINS	9.56±5.20	12.18±6.95 <sup>#</sup>	15.95±9.91 <sup>##*</sup>
HOMA-IR	2.29±1.36	3.22±2.51 <sup>##</sup>	4.33±2.96 <sup>##*</sup>
TC	4.34±0.88	4.74±0.95 <sup>##</sup>	5.19±1.02 <sup>##**</sup>
TG	0.88±0.26	1.28±0.25 <sup>##</sup>	2.98±1.62 <sup>##**</sup>
HDL-C	1.34±0.29	1.23±0.26 <sup>##</sup>	1.14±0.24 <sup>##*</sup>
LDL-C	2.66±0.65	3.05±0.69 <sup>##</sup>	3.32±0.70 <sup>##**</sup>
NAFLD	24(12.5%)	40(30.7%) <sup>##</sup>	91(60.6%) <sup>##**</sup>

Data are mean ±standard deviation, median (interquartile range), or number (percentage).

<sup>#</sup>*P*<0.05, compared with the NFT group. <sup>##</sup>*P*<0.01, compared with the NFT group. <sup>\*</sup>*P*<0.05, compared with the IFT group. <sup>\*\*</sup>*P*<0.01, compared with the IFT group.

**Abbreviations:**BMI: body mass index; WC: waist circumference; FPG: fasting plasma glucose; FINS: fasting plasma insulin concentration; HOMA-IR: homeostasis model assessment of insulin resistance;

TC: total cholesterol; TG: triglyceride; HDL-C: high-density lipoprotein-cholesterol; LDL-C: low-density lipoprotein-cholesterol; NAFLD: non-alcoholic fatty liver disease.

## Figures

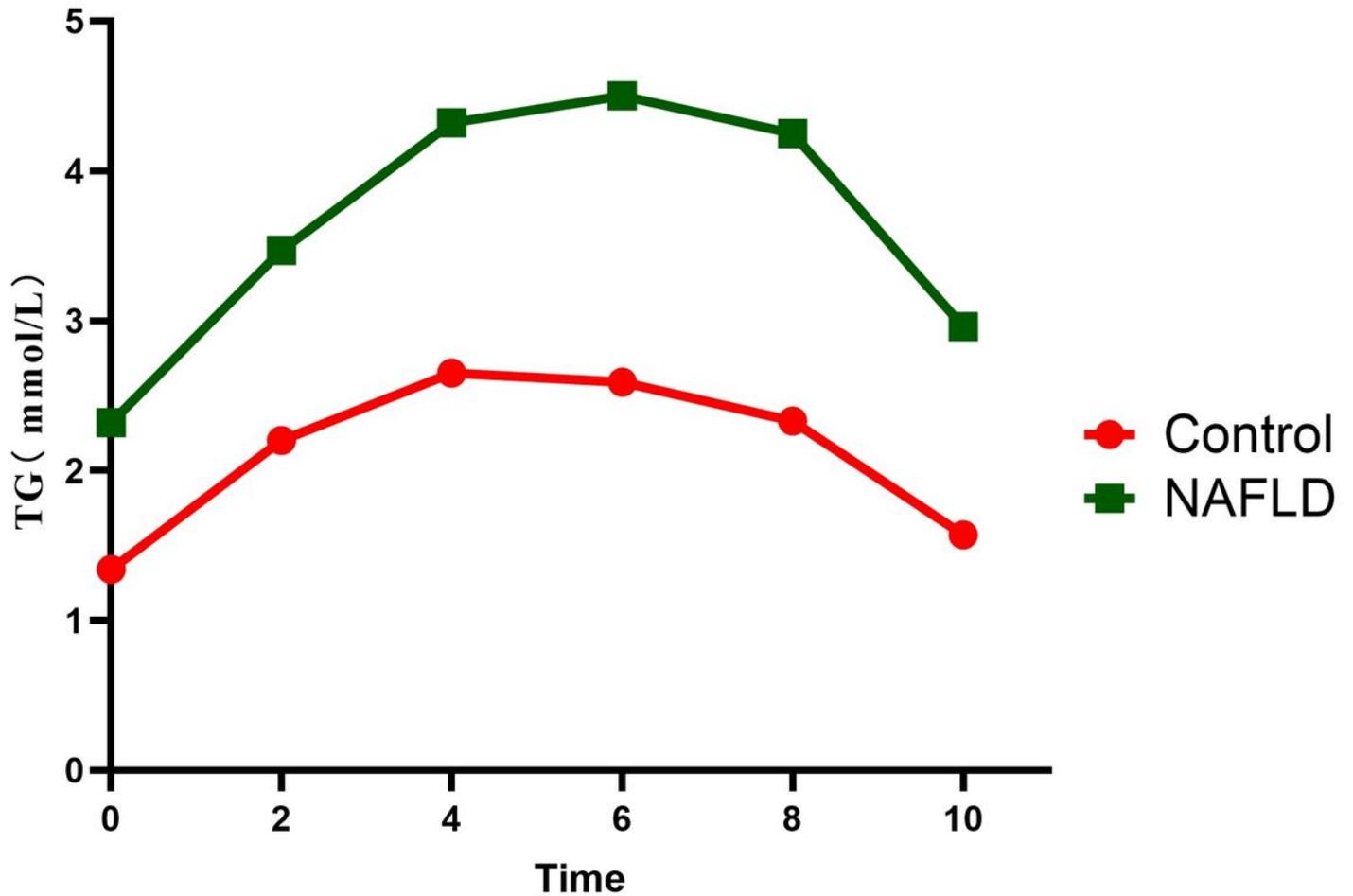
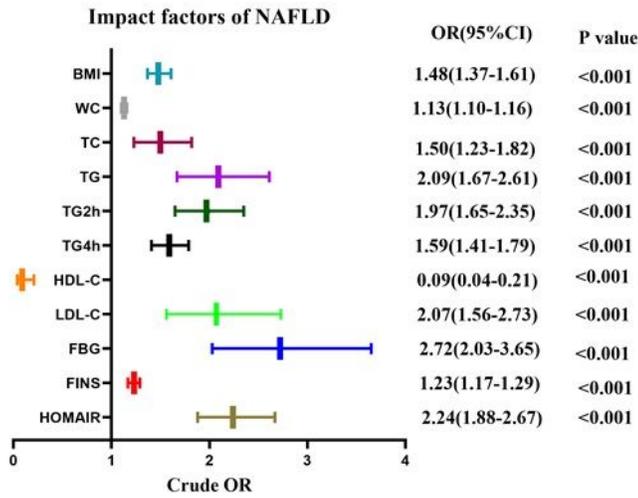
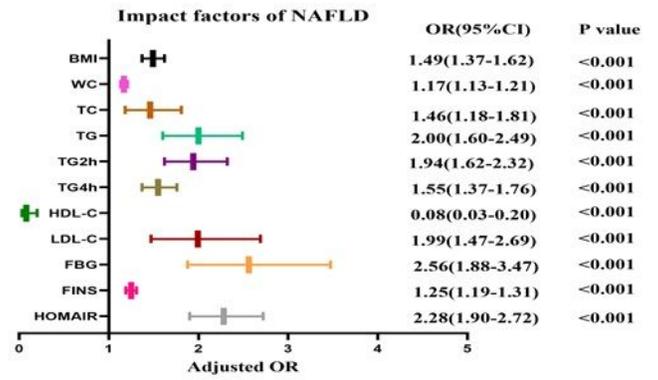


Figure 1

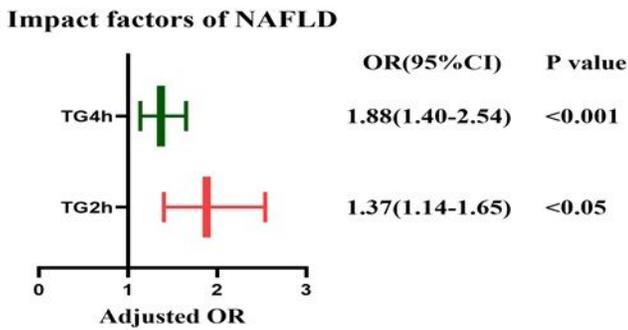
Triglyceride concentrations at time points during oral fat tolerance test; TG-triglyceride; NAFLD- non-alcoholic fatty liver disease



A



B



C

**Figure 2**

Odds ratio of factors influencing NAFLD A-Crude OR; B-Adjusted age and gender; C- Adjusted age, gender and fasting triglyceride; OR: odds ratio; 95%CI: 95% Confidence Interval; TG2h: triglyceride 2h postprandially; TG4h: triglyceride 4h postprandially;