

eISSN: 2581-9615 CODEN (USA): WJARAI Cross Ref DOI: 10.30574/wjarr Journal homepage: https://wjarr.com/

	WJARR	USSN 3591-8915 CODEN (USA): WUMRAI
S	W	JARR
	World Journal of Advanced	
	Research and	
	Reviews	
		World Journal Series INDIA

# Correlation study between lipid profile parameters and liver function enzymes in Babylon province

Mazin Eidan Hadi <sup>1</sup>, Karar Salih Mahdi <sup>2,\*</sup> and Ali abdalla Hindi <sup>3</sup>

<sup>1</sup> Department of Biology, College of Science for women, University of Babylon, Babylon, Hillah, 51001, Iraq.

<sup>2</sup> Department of Pathological analysis, College of Science, Al-Qasim Green University, Babylon, 51013, Iraq <sup>3</sup> Department of Biology, College of Science, Al-Qasim Green University, Babylon, 51013, Iraq.

Department of Diology, conege of science, Ar Quant dreen oniversity, Dubyton, 51013,

World Journal of Advanced Research and Reviews, 2024, 24(01), 1104–1118

Publication history: Received on 29 August 2024; revised on 05 October 2024; accepted on 08 October 2024

Article DOI: https://doi.org/10.30574/wjarr.2024.24.1.3088

## Abstract

**Objective:** The present study was an observational case control design. The data of study were collected in the period from first of January 2024 to March 2024. The study was conducted from clinical Laboratory private in Hilla City, Babylon province, Iraq.

**Material and methods:** A total number of subjects involved in this study was 150 patients, from both sex (male and female) with age range (5-85). Blood samples were taken from each one to determination lipid profile tests (total cholesterol, total triglyceride, High and Low-density lipoprotein cholesterol and liver function tests (ALT, AST, ALP, TSB) and investigation the correlation between them.

**Results:** The results showed that, these were significant correlation between these tests according to male groups, the strength and direction of the correlation between TC and TG levels. The value being significant suggests that there was a moderate strong relationship between these variables (0.491). A strong positive correlation between TC and LDL levels (0.799), HDL and ALP levels (0.290), ALT and AST levels (0.777). In addition, a positive association between AST and ALP levels (0.125), TC and LDL levels (0.799). On the other hand, a weak negative association TC and AST levels (0.045), TC and TSB levels (-0.185), TG and HDL levels (-0.04). TG and AST levels (-0.065), TG and TSB levels (-0.184), HDL and ALT levels (0.123), HDL and AST levels (-0.133), ALT and TSB levels (-0.08) and between ALP and TSB levels (-0.08). However, a moderate negative association between TG and LDL levels (-0.32) and a very weak negative association between HDL and TSB (-0.016).

**Conclusion:** In male group, a strong positive correlation between TC and LDL levels, HDL and ALP levels, ALT and AST levels. A positive association between AST and ALP levels, TC and LDL levels, these were significant correlation between these tests according to female groups, the strength and direction of the correlation between TC and TG levels, a moderate strong relationship between these variables, TC and LDL levels, ALT and AST levels, ALT and ALP levels, ALT and TSB levels, AST and ALP levels, AST and TSB levels and ALP and TSB levels. And moderately strong negative correlation between HDL and ALT levels. So, a relatively weak positive correlation between LDL and AST levels in female group. Moderately positive correlation between LDL and ALP levels.

Keywords: Lipid profile; Liver enzymes; LDL; HDL; AST; ALT and ALP

<sup>\*</sup> Corresponding author: Karar Salih Mahdi

Copyright © 2024 Author(s) retain the copyright of this article. This article is published under the terms of the Creative Commons Attribution Liscense 4.0.

# 1 Introduction

A lipid profile, also known as a lipid panel, is a blood test that measures the levels of various types of lipids (fats) and fatty substances in the blood. These lipids include total cholesterol, low-density lipoprotein (LDL), highdensity lipoprotein (HDL) and triglycerides (1). The total amount of cholesterol in the blood, including both "good" (HDL) and "bad" (LDL) cholesterol, as "bad" cholesterol, LDL cholesterol can build up on the walls of the arteries, leading to atherosclerosis and increasing the risk of heart disease and "good" cholesterol, HDL helps remove LDL cholesterol from the bloodstream, reducing the risk of heart disease (2).

Triglycerides are a type of fat found in the blood, high levels of triglycerides are associated with an increased risk of heart disease. A lipid profile is typically ordered by healthcare providers to assess a person's risk of developing cardiovascular disease or to monitor the effectiveness of treatments for high cholesterol. The results of a lipid profile can help guide lifestyle changes, medication prescriptions, and other interventions aimed at reducing the risk of heart disease (3).

The liver is a major organ only found in vertebrates which performs many essential biological functions such as detoxification of the organism, and the synthesis of proteins and biochemical necessary for digestion and growth (4).

In humans, it is located in the right upper quadrant of the abdomen, below the diaphragm. Its other roles in metabolism include the regulation of glycogen storage, decomposition of red blood cells, and the production of hormones. The various functions of the liver are carried out by the liver cells or hepatocytes. The liver is thought to be responsible for up to 500 separate functions, usually in combination with other systems and organs (5).

Currently, no artificial organ or device is capable of reproducing all the functions of the liver. Some functions can be carried out by liver dialysis, an experimental treatment for liver failure. The liver also accounts for about 20% of resting total body oxygen consumption (6).

The liver plays a crucial role in lipid metabolism. It synthesizes cholesterol and triglycerides and produces lipoproteins involved in transporting lipids in the bloodstream. Liver function abnormalities can impact lipid metabolism and lead to dyslipidemia (abnormal lipid levels) (7).

Liver function tests often measure levels of enzymes such as alanine transaminase (ALT) and aspartate transaminase (AST). Elevated levels of these enzymes can indicate liver damage or inflammation. Liver dysfunction can disrupt lipid metabolism, leading to changes in lipid profile parameters such as increased triglycerides and decreased HDL cholesterol (8).

Certain medications used to manage dyslipidemia or liver conditions can affect both lipid profile and liver function test results. For example, statins, commonly prescribed for high cholesterol, may cause elevations in liver enzymes in some individuals. Diabetes, obesity, and metabolic syndrome, can impact both liver function and lipid metabolism, leading to correlations between liver function test results and lipid profile parameters (9).

The correlation between lipid profile and liver enzymes can vary depending on several factors such as overall health status, underlying medical conditions, lifestyle factors, and medication use (10).

Elevated triglyceride levels are often associated with conditions such as non-alcoholic fatty liver disease (NAFLD) and metabolic syndrome. Liver enzymes such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) may be elevated in individuals with NAFLD, indicating liver inflammation or damage (11).

Elevated levels of total cholesterol, LDL cholesterol, and non-HDL cholesterol have been associated with an increased risk of cardiovascular disease. However, the correlation between cholesterol levels and liver enzymes is not as strong as with triglycerides (12). In some cases, elevated cholesterol levels may be associated with certain liver conditions such as cholestasis or liver cirrhosis, but the relationship is more complex and less direct compared to triglycerides (13).

Higher levels of HDL cholesterol are generally considered beneficial and may be associated with a lower risk of cardiovascular disease. There isn't a significant correlation between HDL cholesterol levels and liver enzymes in the same way as with triglycerides (14).

While lipid profile abnormalities may indicate underlying liver conditions, liver enzymes themselves may not necessarily predict lipid profile changes. However, in conditions such as NAFLD, where there is a disturbance in lipid metabolism, liver enzyme levels may be altered alongside changes in lipid profiles (15). The correlation does not imply causation, and abnormal lipid profiles and liver enzyme levels can often be manifestations of underlying health conditions such as obesity, diabetes, and metabolic syndrome (16).

Furthermore, lifestyle factors such as diet, exercise, and alcohol consumption can significantly influence both lipid profile and liver enzyme levels. Therefore, any interpretation of correlation between these factors should be done in the context of an individual's overall health and medical history, with consideration given to potential confounding variables (17).

### Aim of study

The aim of this study to Survey about correlations between lipid profile parameters and liver function enzymes in Babylon.

# 2 Materials and Methods

#### 2.1 Study design

The present study was an observational case control design. The data of study were collected in the period from first of January 2024 to March 2024. The study was conducted from clinical Laboratory private in Hilla City, Babylon province, Iraq.

## 2.2 Patients

A total number of subjects involved in this study was 150 patients, from both sex (male and female with age range (5-85), all patients and control were from the same ethnic group (Arabic).

## 2.3 Control group

Individuals were taken as a control group of the age-matched group ranging from (5-85) years in healthy control. A permission was taken from all subjects of control group after they were told about the aim and advantages of this study.

#### 2.4 Blood samples

A total of 150 hundred individuals who had undergone one or more abortions had their venous blood drawn using a 5-milliliter medical syringe and a 23-millimeter pointed needle. To separate the blood serum, put the tube in a centrifuge for 5 minutes after being allowed undisturbed for 15-30 minutes; then, freeze the serum until needed.

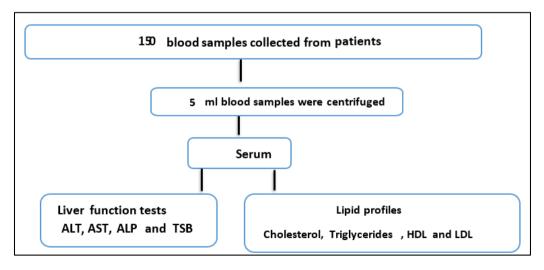


Figure1 Experimental design

## 2.5 Measurement of study parameters

The level of Alanine aminotransferase (ALT) and Aspartate aminotransferase (AST), alkaline phosphatase test (ALP) and Total Serum Bilirubin Test (TSB) were analysis according to their manufacture instructions.

### 2.5.1 Aspartate aminotransferase enzymatic method (AST)

The Aspartate Aminotransferase (AST) test was another common blood test used to assess liver health, as well as heart and muscle function. Follow the laboratory's procedures for processing the blood sample. This may involve centrifugation to separate serum from cells. Use the AST test kit to measure the enzymatic activity of aspartate aminotransferase in the serum sample. Typically, the enzymatic method involves coupling the AST reaction with other enzymatic reactions to produce a detectable color change or fluorescence. Measure the absorbance or fluorescence of the reaction product using a spectrophotometer. Compare the AST levels obtained from the analysis with reference ranges provided by the laboratory. Elevated AST levels may indicate liver damage, heart conditions, muscle injury, or other health issues. Document the AST levels in the patient's medical record.

## 2.5.2 Alanine aminotransferase analysis method (ALT)

The Alanine Aminotransferase (ALT) test, also known as the SGPT (Serum Glutamate Pyruvate Transaminase) test, was a common blood test used to assess liver health and function. Follow the laboratory's procedures for processing the blood sample. This may involve centrifugation to separate serum from cells. Use the ALT test kit to measure the enzymatic activity of alanine aminotransferase in the serum sample. Typically, the ALT analysis method involves coupling the ALT reaction with other enzymatic reactions to produce a detectable color change or fluorescence. Measure the absorbance or fluorescence of the reaction product using a spectrophotometer. Compare the ALT levels obtained from the analysis with reference ranges provided by the laboratory. Elevated ALT levels may indicate liver damage, such as hepatitis, cirrhosis, or liver injury from medications or toxins. Document the ALT levels in the patient's medical record.

## 2.5.3 Alkaline phosphatase test (ALP)

The alkaline phosphatase test (ALP) was a common blood test used to assess liver and bone health. Gather all necessary materials, including sterile equipment, alcohol swabs, gloves, tourniquet, and the vacutainer or syringe for blood collection. The patient's identification was confirmed according to the institution's protocol. Follow the laboratory's procedures for processing the blood sample. This may involve centrifugation to separate serum from cells. Analyze the serum sample using appropriate laboratory equipment, such as a spectrophotometer. Measure the alkaline phosphatase enzyme activity in the serum. Compare the ALP levels obtained from the analysis with reference ranges provided by the Document the ALP levels in the patient's medical record.

#### 2.5.4 Total Serum Bilirubin Test (TSB)

The Total Serum Bilirubin Test was a common blood test used to measure the total amount of bilirubin in the bloodstream. Follow the laboratory's procedures for processing the blood sample. This may involve centrifugation to separate serum from cells. Use the Total Bilirubin test kit to measure the total bilirubin concentration in the serum sample. The test typically involves the reaction of bilirubin with diazo reagents to form a colored product. Measure the absorbance of the reaction product using a spectrophotometer. Compare the total bilirubin levels obtained from the analysis with reference ranges provided by the laboratory. Elevated total bilirubin levels may indicate liver disease, bile duct obstruction, hemolytic disorders, or other health conditions.

Document the total bilirubin levels in the patient's medical record.

# 2.6 Determination of lipids profile

#### 2.6.1 Determination of Serum Total Cholesterol (TC)

Cholesterol concentration was evaluated enzymatically in accord with the method as shown in the following reactions:

Cholesterol esters +  $H_2O$   $\xrightarrow{Cholesterol esterose}$  Cholesterol + Fatty acids Cholesterol +  $O_2$   $\xrightarrow{Cholester oloxidase}$ 

#### Peroxidase

 $H_2O_2$  + Phenol + PAP  $\longrightarrow$  Quinonimine (pink) +4  $H_2O$ 

Reagents	Composition	Concentration
Reagent1 (Buffer)	Phosphate buffer	100 mmol/L
	Chloro-4-phenol Sodium chloride Triton x 1001.	5.0 mmol/L
	Preservative	2.3 mmol/L
		5 mmol/L
Reagent2 (Enzymes)	Cholesterol oxidase	100 IU/L
	Cholesterol esterase	170 IU/L
	Peroxidase	1200 IU/L
	Phenol aminophenazone (PAP)	0.25mmol/L 167µmol/L
	Polyethylene glycol 6000	
Reagent3	Cholesterol	200 mg/dL or
(Std.)		5.17mmol/L

The content of vial reagent 2 (Enzymes) was added to vial reagent 1 (Buffer), and mixed gently until it was completely dissolved (approximately 2- 5 min.) to prepare working reagent.

Table 2 Procedure of determination of serum total cholesterol

Reagents	Blank	Standard	Sample
Reagent	1ml	1ml	1ml
Deionized water (DW)	0.01ml	-	-
Std.	-	0.01ml	-
Sample	-	-	0.01ml

The tubes were mixed, then let stand for 5 min. at 37°C. Absorbance was recorded at lambda max 500 nm against blank. The colour was stable for 1 hour.

# 2.6.2 Determination of Serum High and Low-density lipoprotein cholesterol (HDL, LDL)

HDL and LDL were precipitated by phosphotungstic acid and magnesium chloride. HDL and LDL obtained in supernatant after centrifugation then assessed with reagent of total cholesterol.

**Table 3** Reagents of determination of serum HDL-LDL cholesterol

Reagents	Composition	Concentration
Reagent 1 (precipitant)	Phosphotungstic acid	13.9 mmol/L
	Magnesium chloride pH 6.2	490 mmol/L
Reagent 2 (Standard)	Cholesterol	100 mg/dl or 2.58mmol/L

A volume of 0.5mL samples (serum) was added into clean plain tubes.

A volume of  $50 \mu L$  precipitant was added.

The tubes were mixed vigorously, then let stand for 10 min. at room temperature.

Centrifuge 15 min. at 2000×g. Then next procedure was applied, which include measurement of cholesterol in supernatant.

**Table 4** Procedure of determination of serum HDL-LDL cholesterol

Reagents	Blank	Standard	Sample
Reagent	1ml	1mL	1mL
Distilled water	0.025ml	-	-
Standard	-	0.025ml	-
Supernatant	-	-	0.025ml

The tubes were mixed, then let stand for 5 min. at 37°C. Absorbance was recorded at lambda max 500 nm against blank. The colour was stable for 1 hour.

HDL-cholesterol (mmol/L)	= x	Sample absorbance	2.58
		Standard absorbance	

(Standard concentration = 2.58mmol/L)

2.6.3 Determination of Serum Triglycerides (T.G.)

Triglyceride concentration was estimated by enzymatic method in accord with the method described by Fossati, and principle method associated with Trinder reaction, as shown in the following reactions:

 $\begin{array}{c} \text{Lipase} \\ \text{Triglycerides} \longrightarrow \text{Glycerol+free fatty acids} \end{array}$ 

Glycerol Kinase Glycerol + ATP ← Glycerol-3-phosphate + ADP

$$\begin{array}{c} \mbox{Glycerol-3-phosphate oxidase} \\ \mbox{Glycerol-3-P} + O_2 & \longrightarrow \\ \end{array} \\ \begin{array}{c} \mbox{Glycerol-3-phosphate oxidase} \\ \mbox{Dihydroxyacetone-} p + H_2O_2 \\ \end{array} \\ \end{array}$$

H<sub>2</sub>O<sub>2</sub> + 4-chlorophenol + Phenol aminophenazone

Peroxidase →Quinonimine (pink) + 4H<sub>2</sub>O<sub>2</sub>

Absorbance of coloured complex (quinonimine), proportional to a concentration of TGs in the specimen.

Table 5 Reagents of determination of serum triglycerides

Reagents	Composition	Concentration
Reagent 1 (Buffer)	PIPES	100 mmol/L
	Magnesium chloride	9.8 mmol/L
	Chloro-4-phenol Preservative	3.5 mmol/L
Reagent 2 (Enzymes)	Lipase	1000 IU/L
	Peroxidase	1700 IU/L
	Glycerol-3-p-oxidase	3000 IU/L

	Glycerol kinase	660 IU/L
	Phenol aminophenazone adenosine triphosphate	0.5 mmol/L
		1.3 mmol/L
Reagent 3 (Standard)	Glycerol equivalent to TGs	200 mg/dl or
		2.28 mmol/L

The content of vial reagent 2 (Enzymes) was added to vial reagent 1 (Buffer), mixed gently until completing dissolution (approximately 2 minutes) to prepare work reagent.

**Table 6** Procedure of determination of serum triglyceride

Reagents	Blank	Standard	Sample
Reagent	1ml	1ml	1ml
Deionized water	0.01ml	-	-
Standard	-	0.01ml	-
Sample	-	-	0.01ml

The tubes were mixed, then let stand for 5 min. at 37°C. Absorbance was recorded at lambda max 500 nm against blank. Colour was stable for 1 hour.

Triglyceride (mmol/L) = x Sample absorbance 2.28 Standard absorbance

Standard of T.G = 2.28 mmol/ L

**Statistical analysis**: It was carried out using SPSS version 23. Categorical variables were presented as frequencies and percentages. Continuous variables were presented as (Means  $\pm$  SD). Student t-test was used to compare means between two groups. A p-value of  $\leq$  0.05 was considered as significant.

# 3 Results and Discussion

The results revealed highly percent of patients in this study was male other than female (figure 2), which may refer to increase whole percent of normal residential census in Babylon province, also highly percent of male and female appear in life stage among 31-51 year compare with other life stages that agreement with Gollob and Hanlon report, 2023 about Iraqi population, this configuration study gender percent may be refer to high fertility and child dependency face challenges in investing sufficient resources in the development of young people's human capital. They must address the high costs of older adults' medical and long-term care needs while also investing in the well-being of and future opportunities for younger generations (18).

The results showed significant correlation between these tests according to male groups as a results in Table 7, the strength and direction of the correlation between TC and TG levels. The value being significant suggests that there was a moderate strong relationship between these variables (0.491).

A strong positive correlation between TC and LDL levels (0.799), HDL and ALP levels (0.290), ALT and AST levels (0.777). In addition, a positive association between AST and ALP levels (0.125), TC and LDL levels (0.799).

On the other hand, a weak negative association TC and AST levels (0.045), TC and TSB levels (-0.185), TG and HDL levels (-0.04). TG and AST levels (-0.065), TG and TSB levels (-0.184), HDL and ALT levels (0.123), HDL and AST levels (-0.133), ALT and TSB levels (-0.08) and between ALP and TSB levels (-0.08). However, a moderate negative association between TG and LDL levels (-0.32) and a very weak negative association between HDL and TSB (-0.016).

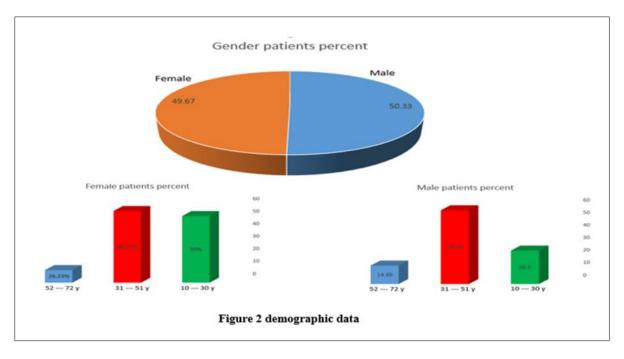


Figure 2 Demographic data

Parameters	Correlation coefficient	Parameters	Correlation coefficient
TC×TG	0.491**	TG×HDL	-0.04
TC×HDL	0.108	TG×LDL	-0.32
TC×LDL	0.799**	TG×ALT	0.095
TC×ALT	0.161	TG×AST	-0.065
TC×AST	0.045-	TG×ALP	0.070
TC×ALP	0.007	TG×TSB	-0.184
TC×TSB	-0.185	HDL×AST	-0.133
HDL×LDL	0.090	HDL×ALP	0.290**
HDL×ALT	-0.123	HDL×TSB	-0.016
LDL×ALT	0.132	ALT×AST	0.777**
LDL×AST	0.03	ALT×ALP	0.175
LDL×ALP	0.042	ALT×TSB	-0.08
LDL×TSB	0.103	AST × TSB	0.087
AST ×ALP	0.125	ALP ×TSB	-0.08

**Table 7** Correlations between lipid profile parameters and levels of liver function enzymes in male group

According to the findings, which indicate a strong and directionally related relationship between TC and TG levels, and these tests were significantly correlated with one another when broken down by male group. There seems to have been a somewhat strong link between the variables, since the value was significant. These findings corroborated those of a previous study of (19) that indicated a moderate to strong relationship between total cholesterol (TC) and triglycerides (TG), two forms of blood lipids that were commonly measured together in lipid profiles to evaluate cardiovascular health.

Similar metabolic pathways affected both TC and TG, according to a research of (20). They were controlled by hormones and enzymes that were part of the lipid metabolism pathway and were produced from carbs and dietary fats. Alterations to these pathways may have a multiplicative effect on TC and TG levels.

Other medical study (21) found the diets high in fat, particularly saturated and Tran's fats, may increase levels of TC and TG. The same holds true for diets heavy in sugar and simple carbs; they may increase TG levels. Therefore, there may be a link between TC and TG levels among people who have comparable eating habits.

Insulin resistance, in which cells do not react normally to insulin, was linked to increased TG levels and often to higher TC levels as well. Obesity and type 2 diabetes were associated with insulin resistance, which in turn contributes to the link between TC and TG levels (22).

Findings from the research of (23) suggest that hereditary variables may have a role in lipid metabolism and TC and TG levels. The two lipid markers may be correlated because certain genetic differences make people more likely to have elevated amounts of both TC and TG. Less TC and TG were seen in those who exercised often. On the other side, both lipid levels might rise as a result of inactivity. So, it's not surprising that people who were physically active tend to have comparable patterns of TC and TG levels, which helps explain why they're correlated (24).

Elevated TC and TG levels may be the result of lipid metabolism being affected by diseases including hypothyroidism, liver illness, or renal disease. Consequently, it was possible for TC and TG levels to be correlated in these people (25). While a study of (26) found levels of TC and TG may be correlated, they were still separate lipid markers that have independent effects on cardiovascular health. An increased risk of atherosclerosis and cardiovascular disease was linked to elevated TC levels, especially LDL cholesterol.

In this study, a strong positive correlation between TC and LDL levels, HDL and ALP levels, ALT and AST levels. In addition, a positive association between AST and ALP levels, TC and LDL levels. These results were agreement with results obtained by (27) who found that, when different biomarkers in the body showed substantial positive associations, it might be because they shared regulatory systems, physiological pathways, or risk factors.

On the other hand, the results in this study found a weak negative association TC and AST levels, TC and TSB levels, TG and HDL levels. TG and AST levels, TG and TSB levels, HDL and ALT levels, HDL and AST levels, ALT and TSB levels and ALP and TSB levels. However, a moderate negative association between TG and LDL levels and a very weak negative association between HDL and TSB.

These results were agreement with result of (28) who found that, the modest negative connection between TC and AST levels suggests that changes in one might not always correspond with changes in the other, even though both TC and AST levels were indicators of liver function. It might be because AST levels were affected by more than simply cholesterol levels.

A results of (29) found Triglycerides and highdensity lipoprotein cholesterol were two lipid markers that play distinct but weakly inverse functions in lipid metabolism. Since there seems to be only a weak negative correlation between TG and HDL levels, it was reasonable to assume that changes in one lipid parameter may not always correspond with changes in the other, maybe because of distinct physiological effects on the two markers.

Further clinical research (30) found there was a weak negative correlation between total serum bilirubin levels and alkaline phosphatase. Liver function affects these levels. There seems to be a modest negative correlation between ALP and TSB levels, which might indicate that bilirubin levels were not always correlated with ALP levels. This could be because each measure was affected by distinct physiological factors according to other study (31).

The correlation between total cholesterol and low-density lipoprotein levels was somewhat negative. One possible explanation for the moderate negative correlation between TG and LDL levels was that changes in triglyceride levels may interact with changes in LDL levels in a more consistent inverse fashion. The levels of total serum bilirubin were markers of liver function, and there was a very weak negative association between HDL and TSB levels. The link between HDL and TSB levels was minimal, but it may indicate that liver function has a slight effect on HDL levels (32).

In addition to that, the results showed that, these were significant correlation between these tests according to female groups as a results in Table 8, the strength and direction of the correlation between TC and TG levels, a moderate strong relationship between these variables (0.474), TC and LDL levels (0.606), ALT and AST levels (0.993), ALT and ALP levels

(0.488), ALT and TSB levels (0.88), AST and ALP levels (0.894), AST and TSB levels (0.971) and ALP and TSB levels (0.88).

However, a relatively weak positive correlation between TC and ALT levels (0.307), TC and AST levels (0.314), TC and TSB levels (0.302), TG and ALT levels (0.86), TG and AST levels (0.279), LDL and ALT levels (0.272), LDL and AST levels (0.270), LDL and ALP levels (0.338) and LDL and TSB levels (0.378). On the other hand, a correlation coefficient of (-0.62) indicates a moderately strong negative correlation between HDL and ALT levels. So, a correlation coefficient of (0.270) indicates a relatively weak positive correlation between LDL and AST levels. Furthermore, a correlation coefficient of 0.338 indicates a moderately positive correlation between LDL and ALP levels and between LDL and TSB levels (0.378).

These results were agreement with results of (33) who found that, not very high The Levels of Total Cholesterol and Triglycerides were Strongly Correlated. The two main lipids detected in the blood, total cholesterol (TC) and triglycerides (TG), were often tested combined in lipid profiles. Since there was a moderate-to-strong positive correlation between TC and TG levels, changes in TC levels will most likely be followed by corresponding changes in TG levels. Possible causes include hereditary variables influencing lipid metabolism, common metabolic pathways, or dietary effects as a results of (34).

Parameters	Correlation coefficient	Parameters	Correlation coefficient
TC×TG	0.474**	TG×HDL	0.02
TC×HDL	0.160	TG×LDL	0.48
TC×LDL	0.606**	TG×ALT	0.86*
TC×ALT	0.307*	TG×AST	0.279*
TC×AST	0.314*	TG×ALP	0.170
TC×ALP	0.369**	TG×TSB	0.228
TC×TSB	0.302*	HDL×LDL	0.68
HDL×ALP	-0.014	HDL×ALT	-0.62
HDL×TSB	-0.109	HDL×AST	-0.363
LDL×ALT	0.272*	ALT×AST	0.993**
LDL×AST	0.270*	ALT×ALP	0.488**
LDL×ALP	0.338*	ALT×TSB	0.88**
LDL×TSB	0.378**	AST × ALP	0.894**
ALP × TSB	0.88**	AST × TSB	0.971**

Table 8 Correlations between lipid profile parameters and levels of liver function enzymes in female group

According to study of (35) found the Levels of lowdensity lipoprotein and total cholesterol have a strong correlation. One of the most important parts of total cholesterol was LDL cholesterol. For this reason, TC and LDL levels were usually highly correlated. It was common for LDL levels to increase in tandem with TC levels. Among the many components that make up total cholesterol, LDL cholesterol was a major player.

The correlation between alkaline phosphatase (ALP) levels and acid transferase (ALT) was moderate. Although they participate in distinct metabolic pathways, the liver enzymes ALT and ALP were related. Because there was a modest positive correlation between ALT and ALP levels, it was reasonable to assume that liver illness or malfunction impacting one enzyme may also impact the other according to (36).

The correlation between ALT and TSB levels was moderate, a study of (37) found bilirubin was a byproduct of the liver's processing of red blood cells, and ALT was an enzyme that was mostly present in the liver. Elevated levels of ALT and

bilirubin may be caused by liver malfunction or illness, since there was a moderate positive association between the two markers.

Association between ALP and AST Levels was Moderate demonstrated by (38), it was found that, the liver was one of several tissues that contain the enzymes AST and ALP. Levels of both AST and ALP that were somewhat positively correlated may point to liver illness or malfunction. The levels of AST and TSB were somewhat related. Liver cells contain the enzyme AST, and bilirubin was a byproduct of the liver's processing of red blood cells. The modest positive correlation between AST and TSB levels implies that increased levels of both AST and bilirubin may be caused by liver malfunction or illness according of (39).

However, in this study, a relatively weak positive correlation between TC and ALT levels, TC and AST levels, TC and TSB levels, TG and ALT levels, LDL and ALT levels, LDL and ALT levels, LDL and ALT levels and LDL and TSB levels.

These results were agreement with results of (40) who found that, a relatively weak positive correlation between various biomarkers may indicate that while there was some association between the variables, it was not as strong as in other cases. Weak positive correlation between TC and ALT Levels. A study of (41) found ALT was an enzyme found primarily in liver cells. While liver function can influence cholesterol metabolism, a weak positive correlation may suggest that variations in TC levels were not consistently accompanied by proportional changes in ALT levels. This could be due to factors such as individual differences in lipid metabolism or the presence of other conditions influencing liver enzyme levels.

In a weak positive correlation, total cholesterol and total serum bilirubin levels were related. As a results of found the liver breaks down red blood cells and produces bilirubin as a byproduct. Changes in bilirubin levels may not always be correlated with changes in TC levels, according to a modest positive association between the two variables. Possible causes include diseases impacting bilirubin metabolism or the fact that TC levels were affected by variables other than liver function.

A modest positive association between LDL and AST levels may suggest that increases in LDL levels were not always followed by corresponding changes in AST levels, similar to the correlation with ALT. Muscle injury was one of the nonliver factors that may affect AST levels (42). The liver was one of several tissues that contain the ALP enzyme. A limited positive connection between LDL and ALP levels suggests that alterations in LDL levels were not regularly followed by corresponding changes in ALP levels, while liver function may impact lipid metabolism. This tenuous correlation can be due to other variables that influence ALP levels or LDL metabolism according to (43).

On the other hand, in this study, moderately strong negative correlation between HDL and ALT levels. So, weak positive correlation between LDL and AST levels. Furthermore, a moderately positive correlation between LDL and ALP levels and between LDL and TSB levels.

In a weak positive correlation, total cholesterol and total serum bilirubin levels were related. The liver breaks down red blood cells and produces bilirubin as a byproduct. Changes in bilirubin levels may not always be correlated with changes in TC levels, according to a modest positive association between the two variables. Possible causes include diseases impacting bilirubin metabolism or the fact that TC levels were affected by variables other than liver function (44).

Other variables impacting LDL metabolism or liver function could be to blame. Levels of LDL and AST weakly correlate positively. A modest positive association between LDL and AST levels may suggest that increases in LDL levels were not always followed by corresponding changes in AST levels, similar to the correlation with ALT. Muscle injury was one of the non-liver factors that may affect AST levels (45).

In this study, there was a moderately strong negative correlation between the levels of Alanine Aminotransferase and High-Density Lipoprotein. A study of (46) found elevated levels of ALT, an enzyme mostly present in liver cells, in the blood may suggest liver injury or illness. Because of its role in lowering blood cholesterol levels and its preventative effects against cardiovascular disease, HDL cholesterol was sometimes referred to as "good" cholesterol.

The relationship between HDL and ALT levels was somewhat negative, meaning that as HDL levels go up, ALT levels go down, and vice versa. This may suggest that levels of HDL cholesterol were inversely related to liver health. Lower HDL levels may be caused by liver malfunction or illness, which disrupts lipid metabolism (47).

# 4 Conclusions

In male group, a strong positive correlation between TC and LDL levels, HDL and ALP levels, ALT and AST levels. A positive association between AST and ALP levels, TC and LDL levels.

A weak negative association TC and AST levels, TC and TSB levels, TG and HDL levels. TG and AST levels, TG and TSB levels, HDL and ALT levels, HDL and AST levels, ALT and TSB levels and between ALP and TSB levels. A moderate negative association between TG and LDL levels and a very weak negative association between HDL and TSB in male group.

These were significant correlation between these tests according to female groups, the strength and direction of the correlation between TC and TG levels, a moderate strong relationship between these variables, TC and LDL levels, ALT and AST levels, ALT and ALP levels, AST and ALP levels, AST and TSB levels and ALP and TSB levels.

In female group, a relatively weak positive correlation between TC and ALT levels, TC and AST levels, TC and TSB levels, TG and ALT levels, TG and ALT levels, LDL and ALT levels, LDL and AST levels and LDL and TSB levels.

Moderately strong negative correlation between HDL and ALT levels. So, a relatively weak positive correlation between LDL and AST levels in female group. Moderately positive correlation between LDL and ALP levels and between LDL and TSB levels.

#### Recommendations

- Study about association between lipid profile and liver function tests in some disease condition such as Diabetic disease.
- Study the correlation of fasting lipid profile in patients with chronic Liver disease.
- Study of association of lipid profile and liver parameters with different grades of Non-alcoholic Fatty Liver Disease.

#### **Compliance with ethical standards**

#### Disclosure of conflict of interest

No conflict of interest to be disclosed.

#### Statement of ethical approval

Ethical committees, patients, and their supporters must all provide their stamp of approval before any medical procedures may begin. Additionally, prior to sample collection, all individuals were informed and given the necessary consent to conduct the research and publish the results (Ethics The human study was approved by the Al-Qasim Green University, Babylon Province, Iraq Review Board).

#### Authors' contributions

All authors had 1. Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; AND 2. Drafting the work or revising it critically for important intellectual content; AND 3. Final approval of the version to be published; AND 4. Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

#### References

[1] Schaefer, E. J., Tsunoda, F., Diffenderfer, M., Polisecki, E., Thai, N., & Asztalos, B. (2016). The measurement of lipids, lipoproteins, apolipoproteins, fatty acids, and sterols, and next generation sequencing for the diagnosis and treatment of lipid disorders.

- [2] Martin, S. S., Blaha, M. J., Elshazly, M. B., Toth, P. P., Kwiterovich, P. O., Blumenthal, R. S., & Jones, S. R. (2013). Comparison of a novel method vs the Friedewald equation for estimating low-density lipoprotein cholesterol levels from the standard lipid profile. Jama, 310(19), 2061-2068.
- [3] Navar, A. M. (2019). The evolving story of triglycerides and coronary heart disease risk. JAMA, 321(4), 347-349.
- [4] Hinton, D. E., Segner, H., & Braunbeck, T. (2017). Toxic responses of the liver. In Target organ toxicity in marine and freshwater teleosts (pp. 224268). CRC Press.
- [5] Zaefarian, F., Abdollahi, M. R., Cowieson, A., & Ravindran, V. (2019). Avian liver: the forgotten organ. Animals, 9(2), 63.
- [6] Deng, J., Wei, W., Chen, Z., Lin, B., Zhao, W., Luo, Y., & Zhang, X. (2019). Engineered liver-on-a-chip platform to mimic liver functions and its biomedical applications: A review. Micromachines, 10(10), 676.
- [7] Heeren, J., & Scheja, L. (2021). Metabolic-associated fatty liver disease and lipoprotein metabolism. Molecular metabolism, 50, 101238.
- [8] Kalas, M. A., Chavez, L., Leon, M., Taweesedt, P. T., & Surani, S. (2021). Abnormal liver enzymes: A review for clinicians. World journal of hepatology, 13(11), 1688.
- [9] Katsiki, N., Mikhailidis, D. P., & Mantzoros, C. S. (2016). Non-alcoholic fatty liver disease and dyslipidemia: an update. Metabolism, 65(8), 1109-1123.
- [10] Sookoian, S., & Pirola, C. J. (2017). Systematic review with meta-analysis: risk factors for non-alcoholic fatty liver disease suggest a shared altered metabolic and cardiovascular profile between lean and obese patients. Alimentary pharmacology & therapeutics, 46(2), 85-95.
- [11] Han, A. L. (2021). Association of Cardiovascular Risk Factors and Metabolic Syndrome with non-alcoholic and alcoholic fatty liver disease: a retrospective analysis. BMC Endocrine Disorders, 21(1), 91.
- [12] Deb, S., Puthanveetil, P., & Sakharkar, P. (2018). A population-based crosssectional study of the association between liver enzymes and lipid levels. International Journal of Hepatology, 2018.
- [13] Malhotra, P., Gill, R. K., Saksena, S., & Alrefai, W. A. (2020). Disturbances in cholesterol homeostasis and nonalcoholic fatty liver diseases. Frontiers in Medicine, 7, 467.
- [14] Castañer, O., Pintó, X., Subirana, I., Amor, A. J., Ros, E., Hernáez, Á., ... & Fitó, M. (2020). Remnant cholesterol, not LDL cholesterol, is associated with incident cardiovascular disease. Journal of the American College of Cardiology, 76(23), 2712-2724.
- [15] Agrawal, S., Dhiman, R. K., & Limdi, J. K. (2016). Evaluation of abnormal liver function tests. Postgraduate medical journal, 92(1086), 223-234.
- [16] Lim, S., Kim, J. W., & Targher, G. (2021). Links between metabolic syndrome and metabolic dysfunction-associated fatty liver disease. Trends in Endocrinology & Metabolism, 32(7), 500-514.
- [17] Rayyan, Y. M., & Tayyem, R. F. (2018). Non-alcoholic fatty liver disease and associated dietary and lifestyle risk factors. Diabetes & Metabolic Syndrome: Clinical Research & Reviews, 12(4), 569-575.
- [18] Bremner, J., Frost, A., Haub, C., Mather, M., Ringheim, K., & Zuehlke, E. (2010). World population highlights: Key findings from PRB's 2010 world population data sheet. Population Bulletin, 65(2), 1-12.
- [19] Wen, J., Huang, Y., Lu, Y., & Yuan, H. (2019). Associations of non-highdensity lipoprotein cholesterol, triglycerides and the total cholesterol/HDL-c ratio with arterial stiffness independent of low-density lipoprotein cholesterol in a Chinese population. Hypertension research, 42(8), 1223-1230.
- [20] Cheng, K., Song, Z., Zhang, H., Li, S., Wang, C., Zhang, L., & Wang, T. (2019). The therapeutic effects of resveratrol on hepatic steatosis in high-fat diet-induced obese mice by improving oxidative stress, inflammation and lipid-related gene transcriptional expression. Medical Molecular Morphology, 52, 187-197.
- [21] Hunter, J. E., Zhang, J., & Kris-Etherton, P. M. (2010). Cardiovascular disease risk of dietary stearic acid compared with trans, other saturated, and unsaturated fatty acids: a systematic review. The American journal of clinical nutrition, 91(1), 46-63.
- [22] Paniagua, J. A. (2016). Nutrition, insulin resistance and dysfunctional adipose tissue determine the different components of metabolic syndrome. World journal of diabetes, 7(19), 483.

- [23] Lu, X., Huang, J., Mo, Z., He, J., Wang, L., Yang, X., ... & Gu, D. (2016). Genetic susceptibility to lipid levels and lipid change over time and risk of incident hyperlipidemia in Chinese populations. Circulation: Cardiovascular Genetics, 9(1), 37-44.
- [24] Jones, P. R. (2020). The associations of physical activity, sedentary time, and aerobic fitness with lipoprotein particle profile in children.
- [25] Duntas, L. H., & Brenta, G. (2018). A renewed focus on the association between thyroid hormones and lipid metabolism. Frontiers in endocrinology, 9, 511.
- [26] Kosmas, C. E., Rodriguez Polanco, S., Bousvarou, M. D., Papakonstantinou, E. J., Peña Genao, E., Guzman, E., & Kostara, C. E. (2023). The triglyceride/high-density lipoprotein cholesterol (TG/HDL-C) ratio as a risk marker for metabolic syndrome and cardiovascular disease. Diagnostics, 13(5), 929.
- [27] Nimptsch, K., Konigorski, S., & Pischon, T. (2019). Diagnosis of obesity and use of obesity biomarkers in science and clinical medicine. Metabolism, 92, 61-70.
- [28] Lambert, N. G., ElShelmani, H., Singh, M. K., Mansergh, F. C., Wride, M. A., Padilla, M., ... & Ambati, B. K. (2016). Risk factors and biomarkers of age-related macular degeneration. Progress in retinal and eye research, 54, 64-102.
- [29] Desgagné, V., Guérin, R., Guay, S. P., Boyer, M., Hutchins, E., Picard, S., .. & Bouchard, L. (2019). Human high-density lipoprotein microtr
- [30] Xiao, Y., Lu, J., Chang, W., Chen, Y., Li, X., Li, D., ... & Yang, H. (2019). Dynamic serum alkaline phosphatase is an indicator of overall survival in pancreatic cancer. BMC cancer, 19, 1-8.
- [31] Mallikarjuna, M. N., & Uday, U. (2017). Diagnostic value of serum bilirubin in appendicular perforation. International Surgery Journal, 4(10), 34453449.
- [32] Gazzin, S., Masutti, F., Vitek, L., & Tiribelli, C. (2017). The molecular basis of jaundice: An old symptom revisited. Liver International, 37(8), 10941102.
- [33] Barkas, F., Elisaf, M., Liberopoulos, E., Liontos, A., & Rizos, E. C. (2016). High triglyceride levels alter the correlation of apolipoprotein B with low-and non-high-density lipoprotein cholesterol mostly in individuals with diabetes or metabolic syndrome. Atherosclerosis, 247, 58-63.
- [34] Gwynn, J., Sim, K., Searle, T., Senior, A., Lee, A., & Brimblecombe, J. (2019). Effect of nutrition interventions on diet-related and health outcomes of Aboriginal and Torres Strait Islander Australians: a systematic review. BMJ open, 9(4), e025291.
- [35] Yang, T., Liu, Y., Li, L., Zheng, Y., Wang, Y., Su, J., ... & Yu, C. (2022). Correlation between the triglyceride-to-highdensity lipoprotein cholesterol ratio and other unconventional lipid parameters with the risk of prediabetes and type 2 diabetes in patients with coronary heart disease: a RCSCD-TCM study in China. Cardiovascular diabetology, 21(1), 93.
- [36] Malakouti, M., Kataria, A., Ali, S. K., & Schenker, S. (2017). Elevated liver enzymes in asymptomatic patients–what should I do?. Journal of clinical and translational hepatology, 5(4), 394.
- [37] Newsome, P. N., Cramb, R., Davison, S. M., Dillon, J. F., Foulerton, M., Godfrey, E. M., ... & Yeoman, A. (2018). Guidelines on the management of abnormal liver blood tests. Gut, 67(1), 6-19.
- [38] Jalili, V., Poorahmadi, Z., Hasanpour Ardekanizadeh, N., Gholamalizadeh, M., Ajami, M., Houshiarrad, A., ... & Doaei, S. (2022). The association between obesity with serum levels of liver enzymes, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase and gamma-glutamyl transferase in adult women. Endocrinology, Diabetes & Metabolism, 5(6), e367.
- [39] Fazaa, A. H., Atya, A. K., & Kredy, H. M. (2022). Evaluation of Liver Function Tests and Their Correlation with HBV Viral Load in Patients with Hepatitis B Virus, Thi-Qar, Iraq. HIV Nursing, 22(2), 1112-1116.
- [40] Zheng, J. S., Sharp, S. J., Imamura, F., Koulman, A., Schulze, M. B., Ye, Z., ... & Wareham, N. J. (2017). Association between plasma phospholipid saturated fatty acids and metabolic markers of lipid, hepatic, inflammation and glycaemic pathways in eight European countries: a cross-sectional analysis in the EPIC-InterAct study. BMC medicine, 15, 1-12.
- [41] Sun, D. Q., Liu, W. Y., Wu, S. J., Zhu, G. Q., Braddock, M., Zhang, D. C., ... & Zheng, M. H. (2016). Increased levels of low-density lipoprotein cholesterol within the normal range as a risk factor for nonalcoholic fatty liver disease. Oncotarget, 7(5), 5728.

- [42] Kathak, R. R., Sumon, A. H., Molla, N. H., Hasan, M., Miah, R., Tuba, H. R., ... & Ali, N. (2022). The association between elevated lipid profile and liver enzymes: a study on Bangladeshi adults. Scientific reports, 12(1), 1711.
- [43] Wang, C., & Li, Z. (2021). Lipoproteins. Clinical Molecular Diagnostics, 179-193.
- [44] Jeong, H. R., Lee, H. S., Shim, Y. S., & Hwang, J. S. (2022). Positive associations between body mass index and hematological parameters, including RBCs, WBCs, and platelet counts, in korean children and adolescents. Children, 9(1), 109.
- [45] Lorenzo, C., Hanley, A. J., Rewers, M. J., & Haffner, S. M. (2013). The association of alanine aminotransferase within the normal and mildly elevated range with lipoproteins and apolipoproteins: the Insulin Resistance Atherosclerosis Study. Diabetologia, 56, 746-757.
- [46] Li, J., & Fan, J. G. (2020). Characteristics and mechanism of liver injury in 2019 coronavirus disease. Journal of clinical and translational hepatology, 8(1), 13.
- [47] Arvind, A., Osganian, S. A., Cohen, D. E., & Corey, K. E. (2015). Lipid and lipoprotein metabolism in liver disease.