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Comparative Study of Lipid Profile and ABO Blood Grouping

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ABSTRACT

The relationship between ABO blood groups and lipid profiles is gaining attention due to its potential implications for understanding cardiovascular disease risk and metabolic health. This study is aimed at assessing the relationship between the lipid profile of apparently healthy individuals with different ABO blood groups. The subjects used in this project were apparently healthy individuals of different ABO blood groups from Ambrose Alli University, Ekpoma, Edo State. We recruited a total of one hundred and sixty (160) subjects for this study, including thirty (30) from blood groups A, B, AB, and O. The results obtained show that the level of total cholesterol was higher in blood groups A (85.13±20.58 mg/dl) and B (89.08±17.19 mg/dl) compared to blood groups AB (81.00±11.65 mg/dl) and O (83.80±15.87 mg/dl). This increase was not statistically significant ($p > 0.05$). The level of triglyceride was higher in blood groups A (171.40±20.37) and B (170.40±18.36 mg/dl) compared to blood groups AB (169.90±15.80 mg/dl) and O (166.70±14.89 mg/dl). Also, the level of HDL was higher in blood group A (50.23±5.36 mg/dl) and B (51.60±5.17 mg/dl) compared to blood group AB (48.55±5.56 mg/dl) and O (49.88±5.55 mg/dl). Furthermore, levels of LDL were higher in blood groups A (104.35±16.91 mg/dl) and AB (105.80±13.88 mg/dl) compared to blood groups B (101.30±15.62 mg/dl) and O (100.33±12.54 mg/dl). From the results obtained in this study, it is evident that there was no significant difference in the levels of total cholesterol, triglycerides, HDL, and LDL within the different ABO blood group systems. Therefore, it could be concluded that lipid profile remained unaltered in blood groups A, B, AB, and O.

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Introduction

Lipids, commonly referred to as fats, represent a diverse group of organic compounds that are soluble in organic solvents but insoluble in water [1]. These compounds play a crucial role in energy storage, cellular structure, and hormonal functions. Phospholipids, which are vital components of cell membranes, are a key example of compound lipids. They consist of two fatty acids, a glycerol backbone, and a phosphate group, playing an essential role in maintaining the structural integrity and fluidity of cell membranes [1]. A lipid profile serves as a critical diagnostic tool in modern medicine, offering a comprehensive assessment of lipid levels, including triglycerides and cholesterol. As highlighted by Sidhu and Naugler, this panel not only aids in diagnosing genetic lipid disorders but also provides valuable insights into the risk of developing cardiovascular diseases, pancreatitis, and other metabolic conditions [2]. Used extensively to evaluate coronary heart disease risk, lipid profile testing is an essential part of preventative healthcare, offering a reliable prediction of life-threatening events like heart attacks or strokes caused

by atherosclerosis [3-5]. This indispensable tool underscores the intersection of lipid metabolism and cardiovascular health, cementing its role in clinical and public health frameworks.

The ABO blood group system is the most critical classification in human blood transfusion, serving as a cornerstone for safe and effective transfusion practices. Its associated anti-A and anti-B antibodies, primarily of the IgM type, are naturally developed early in life through environmental sensitization to antigens from food, bacteria, and viruses. Interestingly, the ABO blood group system is not exclusive to humans but is also observed in other species, including rodents and primates such as chimpanzees, bonobos, and gorillas [6]. Given the vital role of blood group systems in physiology and immunity, it is important to explore the potential influence of ABO blood groups on lipid profiles. Assessing lipid levels across different ABO blood groups will uncover variations that could have clinical or metabolic significance, potentially bridging the gap between genetics and lipid metabolism.

Atherosclerotic Coronary Artery Disease (CAD) is closely associated with abnormalities in lipid metabolism, underscoring the significance of understanding lipid profiles in disease predisposition [7,8]. Evaluating the lipid profiles of individuals with different ABO blood groups is crucial to exploring how these genetic markers contribute to cardiovascular disease risk factors [9]. Notably, emerging evidence has linked ABO blood types to susceptibility to various illnesses [10]. Variations in plasma or serum concentrations of key intestinal and liver enzymes, influenced by ABO blood type, have been implicated in increased disease susceptibility and differences in nutrient assimilation from the diet [11]. Despite these insights, there remains a paucity of information on the relationship between ABO blood type and lipid profile variations, leaving a critical gap in understanding how genetic factors interplay with lipid metabolism [5,12].

Recent genome-wide association studies suggest that ABO blood group antigens may influence the systemic inflammatory state, potentially impacting disease susceptibility. Since the identification of ABO blood groups, numerous studies have highlighted associations between these groups and the prevalence of various diseases, including stomach cancer, duodenal and gastric ulcers, cholera, and cardiometabolic conditions [13-18]. Particularly alarming is the rise of Cardiovascular Diseases (CVD) as the leading global cause of mortality. Major risk factors for CVD include age, obesity, smoking, diabetes mellitus, hypertension, and a family history of the condition. Notably, multiple studies have established a connection between non-O ABO blood group phenotypes and an increased risk of cardiovascular events, as well as other significant risk factors [18-20]. Despite these findings, the relationship between ABO blood type and lipid profiles—a key determinant of cardiovascular health-remains insufficiently explored and poorly understood. To address this gap, this study aims to assess the lipid profiles of apparently healthy individuals across different ABO blood groups, contributing to a deeper understanding of the interplay between genetic factors and lipid metabolism.

Materials and Methods

Area of Study

This study was carried out in Ekpoma, the headquarters of the Esan West Local Government area of Edo State. It is located at latitude 6° 45 N and longitude 6° 08' E. It is moderately populated, with the peoples' occupation being farming and trading. The main sources of water in the locality are rainfall and wells. The well is augmented by an irrigation scheme provided by the government for public use. The university is situated in this region. It is usually cold at night and very hot during the day. It also has undulating topography [21].

Study Population

The subjects used in this project were apparently healthy individuals of different ABO blood groups from Ambrose Alli University, Ekpoma, Edo State. We recruited a total of one hundred and sixty (160) subjects for this study, including forty (40) from blood groups A, B, AB, and O. We also obtained the

- **Research Design:** The research was designed as a cross-sectional study to evaluate the levels of Total Cholesterol, Triglycerides, Low-Density Lipoprotein (LDL), and High-Density Lipoprotein (HDL) in apparently healthy individuals of different ABO blood groups. This study was carried out over a five (5)-month period. A complete record of medical history was obtained for each subject, including their name, age, sex, dietary habits, and nutritional status.
- **Inclusion and Exclusion Criteria:** Apparently healthy individuals of different ABO blood groups from Ambrose Alli University, Ekpoma, Edo State, with no evident signs of illness, were recruited for the study. Conversely, individuals exhibiting clear symptoms or with a documented history of illness were excluded from participation.
- **Sample Collection:** Blood sample was collected from the antecubital vein into an accurately labelled plain bottle for each individual. Some aliquots of the blood sample were dispensed into EDTA containers for ABO blood grouping. Both ABO and Rhesus blood groups were determined using the tile method. The blood samples were centrifuged with a laboratory centrifuge within two hours of collection, and the serum was separated into clean, dry, plain tubes that were labeled corresponding to the initial blood sample bottle. Analysis was carried out for total cholesterol, triglyceride, high-density lipoprotein, and low-density lipoprotein.

Sample Analysis

ABO Blood Group Determinations

The ABO blood groups were determined using the tile method according to Cheesbrough [22]. When known sera (Anti A, B, AB, and D) react with an unknown red cell (antigen), it shows the presence of a corresponding antibody, e.g., when Anti A reacts with an unknown red cell (antigen) and shows agglutination or hemolysis, it therefore means that the red cell has a corresponding antigen. Two drops of individual serum were placed into each of the four precipitin tubes; one drop of 3% suspension of a known blood group A1 red cell was added to the first tube, group A2 red cells to the second tube, group B red cells to the third, and O red cells to the fourth. The cells and serum were mixed gently. The procedure was repeated using two drops of anti-A, anti-B, anti-AB, and anti-D sera and one drop of 3% test red cell suspension. It was centrifuged at 200g for 5 minutes after 30 minutes of incubation. It was examined for agglutination both macroscopically and microscopically.

Determination of Total Cholesterol

Total cholesterol levels were measured using the enzymatic endpoint method (CHOD-PAP) as described by Richmond [23]. This method involves the enzymatic hydrolysis of esterified cholesterol to free cholesterol by cholesterol esterase. The free cholesterol is subsequently oxidized by cholesterol oxidase, resulting in the formation of hydrogen peroxide. In the final reaction step, hydrogen peroxide reacts with phenol and 4-aminoantipyrine under the catalytic action of peroxidase, producing a red quinoneimine dye. The intensity of the dye is

directly proportional to the cholesterol concentration in the sample. For the procedure, 10 microliters each of distilled water (blank), standard solution, and sample were dispensed into appropriately labeled test tubes. To each tube, 1 milliliter of cholesterol reagent was added, and the mixtures were thoroughly mixed and incubated at 37°C for 5 minutes. The absorbance of the samples and standard were then measured against the blank at a wavelength of 500 nm using a spectrophotometer. The cholesterol concentration was calculated by comparing the sample absorbance to that of the standard.

Determination of Triglycerides

The triglyceride levels were determined using a colorimetric method as described by Trinder [24]. This method relies on enzymatic reactions to hydrolyze triglycerides into glycerol and fatty acids via lipoprotein lipase. The glycerol produced reacts with ATP in the presence of glycerol kinase to form glycerol-3-phosphate. This intermediate is subsequently oxidized by glycerol phosphate oxidase to generate hydrogen peroxide. The hydrogen peroxide then reacts with a phenolic compound and 4-aminoantipyrine under the catalytic action of peroxidase to produce a red-colored quinonimine dye. The intensity of this color is directly proportional to the triglyceride concentration in the sample. For the procedure, 10 microliters of distilled water (blank), standard solution, and sample were dispensed into appropriately labeled test tubes. To each tube, 1 milliliter of triglyceride reagent was added, and the mixtures were thoroughly mixed. The tubes were incubated in a water bath at 37°C for 5 minutes. After incubation, the absorbance of the standard and samples was measured against the blank at a wavelength of 500 nm using a spectrophotometer. The concentration of triglycerides in the samples was determined by comparing the absorbance of the samples to that of the standard

Estimation of High-Density Lipoprotein – Cholesterol

High-Density Lipoprotein Cholesterol (HDL-C) was determined using the precipitation method described by Lopes-Virella [25]. This method separates HDL cholesterol from other lipoproteins by precipitating Low-Density Lipoprotein (LDL), Very Low-Density Lipoprotein (VLDL), and chylomicron fractions with phosphotungstic acid in the presence of magnesium ions. After centrifugation, the HDL cholesterol remains in the supernatant and is quantified using the enzymatic cholesterol assay. Two hundred microliters of the standard or sample was dispensed into appropriately labeled test tubes. To each tube, 500 microliters of precipitant was added and mixed thoroughly. The mixture was allowed to stand for 10 minutes at room temperature and was then centrifuged at 4000 rpm for 10 minutes. The supernatant, containing HDL cholesterol, was carefully separated for further analysis. One hundred microliters of distilled water (blank), standard, and supernatant from the sample tubes were added into their respective labeled test tubes. One milliliter of cholesterol reagent was added to each tube, and the mixtures were thoroughly mixed. The tubes were incubated at 37°C for 5 minutes. After incubation, the absorbances of the standard and samples were measured against the blank at a wavelength of 500 nm using a spectrophotometer. The HDL-C concentration in the samples was calculated based on the absorbance values compared to the standard.

Estimation of Low-Density Lipoprotein-Cholesterol (LDL)

This was determined using the Friedewald formula described by Friedewald [26].

LDL- cholesterol (mmol/l) = Total cholesterol – (TG/ 2.2+ HDL-cholesterol)

Statistical Analysis

The data generated from the study were analyzed using SPSS statistical package to determine the mean, standard deviation as well as the comparison of the control with the test using Analysis of variance at 95% confidence limit.

Results

The results obtained shows that the level of Total cholesterol, triglyceride and HDL was higher in blood group A and B compared to blood group AB and O. This increase was not statistically significant ($p>0.05$). Also, levels of LDL were higher in blood group A and AB compared to blood group B and O (Table 1).

Table 1: Lipid Profile of Individuals with Different ABO Blood Groups

Parameters (mg/dl)	Blood Group A (n=40)	Blood Group B (n=40)	Blood Group AB (n=40)	Blood Group O (n=40)	F-Value	P-Value
TC	85.13±20.58[a]	89.08±17.19[a]	81.00±11.65[a]	83.80±15.87[a]	1.065	0.366
TG	171.40±20.37[a]	170.40±18.36[a]	169.90±15.80[a]	166.70±14.89[a]	0.523	0.667
HDL	50.23±5.36[a]	51.60±5.17[a]	48.55±5.56[a]	49.88±5.55[a]	1.563	0.201
LDL	104.35±16.91[a]	101.30±15.62[a]	105.80±13.88[a]	100.33±12.54[a]	0.895	0.446

Keys: TC= Total Cholesterol, TG= Triglyceride, HDL= High Density Lipoprotein, LDL= Low Density; Lipoprotein, n= Sample Size, $P<0.05$=Significant; $p>0.05$=Not significant. Values in a row with the same superscript are not statistically significant at $p<0.05$

When compared to blood groups B and O, the results reveal that total cholesterol levels were higher in blood groups A and B in both male and female subjects. This observation was not statistically significant ($p > 0.05$). Blood groups AB and O in males showed a non-significant increase in triglyceride levels ($p > 0.05$) compared to blood groups A and B. The triglyceride level did not significantly increase in the females ($p > 0.05$). However, the triglyceride level was higher in blood groups B and AB compared to blood groups A and O. Male subjects showed an increase in their HDL level in blood groups B and O compared to the other blood groups, whereas female subjects showed no significant increase in HDL level but showed a higher level in blood groups B and AB compared to other blood groups. Furthermore, blood groups B and AB showed an increase in the LDL level in male subjects compared to other blood groups, while blood groups A and O showed no significant increase in HDL level compared to other blood groups (Table 2).

Table 2: Lipid Profile of Males and Females with Different ABO Blood Groups

Parameters (mg/dl)	Blood Group A Male (n=22) Female(n=18)	Blood Group B Male (n=27) Female(n=13)	Blood Group AB Male (n=25) Female(n=15)	Blood Group O Male (n=28) Female(n=12)	F-Value	P-Value
TC Male Female	 86.15±21.23[a] 84.23±19.24[a]	 88.09±16.19[a] 89.11±15.45[a]	 81.00±11.65[a] 82.22±10.65[a]	 85.80±14.87[a] 82.12±13.33[a]	 1.056 1.023	 0.356 0.344
TG Male Female	 167.70±13.89[a] 168.21±20.12[a]	 171.30±17.36[a] 170.11±15.22[a]	 170.20±21.37[a] 170.34±22.30[a]	 172.80±17.80[a] 166.29±12.99[a]	 0.623 0.599	 0.669 0.722
HDL Male Female	 51.33±4.36[a] 50.21±4.23[a]	 53.70±5.17[a] 55.88±6.33[a]	 49.59±7.56[a] 51.00±6.77[a]	 53.85±2.55[a] 50.79±3.53[a]	 1.663 1.772	 0.301 0.442
LDL Male Female	 102.15±11.91[a] 103.11±12.12[a]	 102.40±11.62[a] 100.22±10.62[a]	 104.10±12.98[a] 102.11±11.11[a]	 101.23±14.54[a] 103.22±11.00[a]	 0.995 0.972	 0.546 0.441

Keys: TC= Total Cholesterol, TG= Triglyceride, HDL= High Density Lipoprotein, LDL= Low Density, Lipoprotein, n= Sample Size, P<0.05=Significant; p>0.05=Not Significant. Values in a row with the same superscript are not statistically significant at p<0.05.

The total cholesterol level was higher in blood groups B and O within the age range of 16–20 years compared to the other blood groups. Also, the total cholesterol level was higher in blood groups A and AB within the age range of 21-30 years when compared to the other blood groups. The triglyceride level of the various blood groups was not significant but was higher in blood groups B and AB between the ages of 16–20 and 21–30. This observation was not statistically significant ($p > 0.05$). While the HDL level was not statistically significant ($p > 0.05$), it did increase in blood groups B and O during the 16-20 years and 21-30 years age ranges. Blood groups AB and O experienced an increase in the LDL level ($p > 0.05$), while blood groups B and O experienced an increase in LDL values within the age range of 21-30 years. This was not statistically significant ($p > 0.05$) (Table 3).

Table 3: Lipid Profile of Individuals with Different ABO Blood Groups according to Age

Parameters (mg/dl)	Blood Group A 16-20 (n=21) 21-30(n=19)	Blood Group B 16-20 (n=26) 21-30(n=14)	Blood Group AB 16-20 (n=30) 21-30(n=10)	Blood Group O 16-20 (n=29) 21-30(n=11)	F-Value	P-Value
TC 16-20 years 21-30 years	 85.25±11.23[a] 84.23±11.24[a]	 87.09±11.19[a] 84.11±12.45[a]	 82.00±11.65[a] 84.22±14.65[a]	 86.70±11.87[a] 84.12±13.33[a]	 1.067 1.034	 0.365 0.341
TG 16-20 years 21-30 years	 171.20±22.37[a] 165.21±22.12[a]	 172.40±16.36[a] 171.12±14.22[a]	 173.70±15.80[a] 171.31±22.30[a]	 164.60±14.89[a] 162.90±13.99[a]	 0.630 0.622	 0.665 0.711
HDL 16-20 years 21-30 years	 52.33±4.36[a] 51.21±3.23[a]	 54.50±3.17[a] 56.68±9.33[a]	 48.91±4.56[a] 50.00±6.73[a]	 55.55±3.55[a] 52.59±4.53[a]	 1.763 1.752	 0.401 0.542
LDL 16-20 years 21-30 years	 104.12±12.11[a] 102.14±13.12[a]	 103.60±12.62[a] 104.42±11.62[a]	 105.12±14.78[a] 103.17±12.11[a]	 105.21±11.64[a] 107.23±10.60[a]	 0.885 0.772	 0.671 0.364

Keys: TC= Total Cholesterol, TG= Triglyceride, HDL= High Density Lipoprotein, LDL= Low Density, Lipoprotein, n= Sample Size, P<0.05=Significant; p>0.05=Not Significant. Values in a row with the same superscript are not statistically significant at p<0.05.

Discussion

The human ABO blood group system stands as the most critical classification in blood transfusion medicine, underscoring its central role in healthcare. Anti-A and anti-B antibodies, predominantly IgM in nature, are developed early in life through environmental exposures to elements such as food, bacteria, and viruses [6]. This immunological foundation highlights the potential for biological variability among individuals based on their ABO blood type. Given the significance of lipids in health and disease, this study explores the association between ABO blood groups and lipid profiles to uncover potential variations. The lipid profile—a comprehensive panel of blood tests—serves as a primary tool for detecting lipid abnormalities, including triglycerides and cholesterol levels [5]. By examining lipid profiles across different ABO blood groups, this study aims to provide insights into how blood type may influence metabolic health and disease predisposition.

Coronary Artery Disease (CAD) is a significant cardiovascular disorder that compromises the heart's blood supply, posing severe health risks globally [27]. This condition arises from the accumulation of cholesterol plaques along the walls of blood vessels, leading to their narrowing or obstruction. Such blockages impair the delivery of oxygen and essential nutrients to the heart muscle, undermining its functionality. In severe cases, the sudden loss of blood supply to a portion of the heart can result in the death of that tissue, culminating in a heart attack [27]. The heart plays a pivotal role in the circulatory system, functioning as the pump that ensures oxygen-rich blood reaches vital organs such as the brain, kidneys, and other critical systems. Any disruption in its

operation directly affects these organs, leading to tissue damage, organ failure, and ultimately, death. This underscores the systemic implications of cardiac abnormalities. Globally, ischemic heart disease remains the foremost cause of mortality, highlighting the urgent need for continued research and intervention strategies to mitigate its impact [27].

The results of this study revealed that the levels of total cholesterol, triglycerides, and HDL were higher in blood groups A and B compared to blood groups AB and O, although these differences were not statistically significant ($p>0.05$). Additionally, LDL levels were higher in blood groups A and AB in comparison to blood groups B and O. Various changes in the levels of total cholesterol, triglycerides, HDL, and LDL were observed across different blood groups when analyzed in relation to sex and age. In line with findings from Western countries, blood group O has been associated with a lower incidence of coronary heart disease compared to blood group A [19,20,28-31]. Specifically, studies have shown that males with blood group O tend to have lower mean cholesterol levels than those with blood group A, reinforcing the hereditary pathophysiology of ischemic heart disease. The present study's findings are consistent with this observation, as blood group O exhibited lower total cholesterol levels than other blood types. However, contrasting results from Amirzadegan and Sari indicated no significant correlation between the onset of coronary artery disease and blood type [32,33]. These studies found that individuals across different blood groups exhibited similar prevalences of major cardiovascular risk factors, which did not appear to influence the development of early coronary artery disease. The findings of this study align with these reports, as no significant correlation was observed between Total Cholesterol (TC), Triglycerides (TG), HDL, and LDL levels across the different blood groups.

There are numerous risk factors that contribute to the development of ischemic heart disease, even though the exact etiology is unknown. It has been demonstrated that reducing these risk factors lessens the severity and complications of the illness. When it comes to the prevalence of cardiac disease, there are clear differences between blood types [34]. The authors made a compelling case for the presence of a genetic component linked to blood group A that is distinct from the other risk variables that are likewise linked to a higher incidence of myocardial infarction. Blood type "A" is associated with a higher incidence of ischemic heart disease as well as a higher total serum cholesterol concentration, according to an eight- year research of 7662 males that was published in the esteemed British Medical Journal [20].

The findings of this study suggest that blood groups A and AB may be associated with an increased likelihood of heart disease, as evidenced by slight elevations in LDL levels and marginal decreases in HDL levels. These results align with previous studies by Bhattacharya and Banerjee and Datta, who noted a higher incidence of ischemic heart disease in individuals with blood group A. In their study, blood groups A and O were the most prevalent among the Sikkimese population, with blood group A showing the highest association with ischemic heart disease [35,36].

Our results also support the observations of Platt, Whincup, Garrison and Akhund who found correlations between ABO blood types and cardiovascular risk factors. Garrison demonstrated that blood group O had the lowest incidence of intermittent claudication, with a strong association between blood type and claudication [19,20,28,30]. Moreover, non-O blood types showed slight but non-significant increases in the occurrence of specific coronary heart disease (CHD) events and consistently higher cholesterol levels. In certain regions of Pakistan's Sindh province, individuals with blood group O were less likely to experience angina pectoris and myocardial infarction compared to those with blood group A [30,37].

The relationship between blood types and atherosclerosis has been widely studied, with several authors documenting correlations between non-O blood types and increased cardiovascular risk [29,31,38,39]. Stakisaitis identified blood groups A and B as genetically based risk factors for atherosclerosis in the Lithuanian population, while Stakisaitis suggested an association between the B blood group and coronary atherosclerosis in women. However, Cakir found no significant correlation between the Lewis genotype and subclinical atherosclerosis [40].

Suadicani highlighted those men with blood type O were at greater risk of ischemic heart disease due to long-term occupational exposure to airborne contaminants, compared to men with other ABO phenotypes [41]. Nydegger identified the ABO blood group B allele as an independent risk factor for myocardial infarction, while Von Beckerath reported that the O1 allele reduces the risk of myocardial infarction. Conversely, some studies have found no significant relationship between blood type and ischemic heart disease [32,33,42,43]. Amirzadegan concluded that the onset of coronary artery disease was unrelated to ABO blood types and that the prevalence of major cardiovascular risk factors did not differ significantly across blood groups. Similarly, Sari found in a Turkish cohort study that ABO blood type did not appear to substantially influence the onset of myocardial infarction, which is consistent with the findings of this study [33].

Conclusion

The results of this study reveal no statistically significant differences in the levels of total cholesterol, triglycerides, HDL, and LDL across the various ABO blood groups. These findings suggest that the lipid profiles of individuals with blood groups A, B, AB, and O remain similar, with no detectable correlation between ABO blood type and lipid parameters such as total cholesterol, triglycerides, HDL, and LDL. Given the lack of significant variations observed in this study, further investigation is warranted to explore potential underlying factors that could explain the observed lipid profile trends and to better understand the relationship, if any, between ABO blood groups and lipid metabolism.

Conflict of Interest: The authors declare no conflicts of interest. The authors alone are responsible for the content and the writing of the paper.

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References

1. Fahy E, Subramaniam S, Murphy RC, Nishijima M, Raetz CR, et al. (2009) Update of the LIPID MAPS comprehensive classification system for lipids. Journal of Lipid Research 50: 9-14.
2. Sidhu D, Naugler C (2012) Fasting Time and Lipid Levels in a Community-Based Population. Archives of Internal Medicine pp: 1-4.
3. Ugonabo MC, Onwuamaeze IC, Okafor EN, Ezeoke AC (2007) Plasma cholesterol of sickle cell anaemia patients in Enugu Nigeria. Bio Res 5: 241-243.
4. Nnodim JK, Opara HU, Ibeaja OA (2012) Plasma Lipid Profile in Sickle Cell Disease Patients in Owerri, Nigeria. Pakistan Journal of Nutrition 11: 64-65.
5. Festus OO, Ebaluegbeifoh LO, Iyevhobu LOK, Dada FL, Iweka FK (2016) Assessment of Lipid Profile in Automobile Mechanics in Ekpoma, Edo State. European Journal of Biomedical and Pharmaceutical Sciences 9: 100-107.
6. Maton KI (1993) A bridge between cultures: Linked ethnographic-empirical methodology for culture anchored research. American Journal of Community Psychology 21: 747-773.
7. Tunbridge WMG, DC Evered, R Hall, D Appleton, M Brewis, et al. (1977) The spectrum of thyroid disease in a community: the Whickham surve. Clinical Endocrinology 6: 481-493.
8. Pucci B, Kasten M, Giordano A (2000) Cell cycle and apoptosis. Neoplasia 4: 291-299.
9. Omolumen LE, Obodo BN, Iyevhobu KO, Okereke NP, Idara IU, et al. (2020). Evaluation of Lipid Profile of Human Immuno Deficiency Virus (HIV) and Tuberculosis (TB) Positive Pregnant Women. European Journal of Biomedical and Pharmaceutical Sciences 5: 693-699.
10. Armelle P, Pierre Yves Boelle R, Dominique W, Raoul P, Annie R (2006) Association between ABO blood group and fibrosis severity in chronic hepatitis C infection. Dig Dis Sci 51: 1633-1636.
11. Adamo PD (2010) Intestinal Alkaline Phosphates Enzyme. Retrieved 6: 32-323.
12. Omolumen LE, Iyevhobu KO, Usiobeigbe OS, Airhomwanbor KO, Ebaluegbeifoh LO, et al. (2024) Lipid Profile and Nutritional Indices in Pregnant and Post-Menopausal Women. Scholastic Medical Sciences 2: 01-08.
13. Aird I, Bentall HH, Roberts JAF (1953) A relationship between cancer of stomach and the ABO blood groups. British Medical Journal 1: 799-801.
14. Clarke CA, Cowan WK, Edwards JW, Howel Evans AW, McConnell RB, et al. (1955) The relationship of the ABO blood groups to duodenal and gastric ulceration. British Medical Journal 4940: 643-646.
15. Glass RI, Holmgren J, Haley CE, Khan MR, Svennerholm AM, et al. (1985) Predisposition for cholera individuals with "0" blood group. Possible evolutionary significance. American Journal of Epidemiology 6: 791-796.
16. Bates MM (1971) Venous thromboembolic disease and ABO blood type. The Lancet 7692: 1-239.
17. Cronenwett JL, Davis JT, Garrett EH (1983) ABO blood group and serum lipids in female atherosclerosis. Journal of Cardiovascular Surgery 24: 658-661.
18. Erikssen J, Thaulow E, Stormorken H, Brendemoen O, Hellem A (1980) Blood groups and coronary heart disease. Thrombosis and Haemostasis 2: 137-140.
19. Platt D, Muhlberg W, Kiehl L, Schmitt Ruth R (1985) ABO blood group system, age, sex, risk factors and cardiac infarction. Arch Gerontol Geriatr 4: 241-249.
20. Whincup PH, Cook DG, Phillips AN, Shaper AG (1990) ABO blood group and ischaemic heart disease in British men. Archive of British Medical Journal 300: 1679-1682.
21. World Gazetteer (2007) Population of Cities, news, divisions. Retrived from: https://whgazetteer.org/.
22. Cheesbrough M (2006) Antimicrobial sensitivity testing, in District ¬Laboratory Practice in Tropical Countries. Cambridge, UK: Cambridge University Press.
23. Richmond W (1973) Preparation and properties of a cholesterol oxidase from Nocardia sp. and its application to the enzymatic assay of total cholesterol in serum. Clinical Chemistry 19: 1350-1356.
24. Trinder P (1969) Cholesterol and Triglycerides estimation. Annals of Clinical Biochemistry 6: 24-26.
25. Lopes-Virella MF (1977) High Density Lipoprotein estimation, Precipitation method. Clinical Chemistry 18: 449.
26. Friedewald WT, Levy RI, Fredrickson DS (1972) Estimation of the concentration of low- density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clinical Chemistry 18: 499-502.
27. Suchandra B, Utpal K (2011) Relationship of ABO Blood Groups with Ischaemic Heart Disease West Bengal University of Health Sciences, Kolkata. Indian Medical Gazette pp: 431-433.
28. Garrison RJ, Havlik RJ, Harris RB, Feinleib M, Kannel WB, et al. (1976) ABO blood group and cardiovascular disease the Framingham study. Atherosclerosis 25: 311-318.
29. Stakishaitis DV, Ivashkiavichene LI, Narvilene AM (1991) Atherosclerosis of the coronary arteries and the blood group in the population of Lithuania. Vrach Delo 1: 55-57.
30. Akhund IA, Alvi IA, Ansari AK, Mughal MA, Akhund AA (2001) A study of relationship of ABO blood groups with myocardial infarction and angina pectoris. J Ayub Med Coll Abbottabad 13: 25-26.
31. Stakisaitis D, Maksvytis A, Benetis R, Viikmaa M (2002) Coronary atherosclerosis and blood groups of ABO system in women. Medicina (Kaunas) 38: 230-235.
32. Amirzadegan A, Salarifar M, Sadeghian S, Davoodi G, Darabian C, et al. (2006) Correlation between ABO blood groups, major risk factors and coronary heart disease. Intern J Cardiol 110: 256-258.
33. Sari IA, Ozer OB, Davutoglu VA, Gorgulu SB, Eren MB, et al. (2008) ABO blood group distribution and major cardiovascular risk factors in patients with acute myocardial infarction. Blood coagulation and Fibrinolysis 19: 231-234.
34. Iyamu OA, Onyeneke EC, Nwarunma EO, Iyevhobu KO (2023). Effects of Allium cepa on Lipid Profile and Cardiac Enzyme Marker in Wistar Rats Fed with High-Fat Diet. African Scientist 24: 436-444.
35. Bhattacharya K, Ghosh R, Ghosh M, Singha Mahapatra A, et al. (1991) ABO and risk of artheriosclerosis. Indian J Physiol Allied Sci 45: 34-40.
36. Banerjee S, Datta UK (2008) A Study of the distribution of ABO & Rh (D) blood groups amongst Sikkimese. JIMA 106: 506-507.
37. Airhomwanbor KO, Iyevhobu KO, Omolumen LE, Idehen CI, Akhere PI, et al. (2023) Assessment of Lipid Profile on Patients with Pulmonary Tuberculosis. Acta Scientific Medical Sciences 7: 198-207.
38. Chaudhary R, Shukla JS (1999) Association of Lewis blood group with ischemic heart disease. Indian J Med Res 109: 103-104.
39. Hein H, Suadicani P, Gyntelberg F (2001) Lewis phenotypes, leisure time physical activity, and risk of disease: an 11 year follow up in the Copenhagen male study. Heart 85: 159-164.
40. Cakir B, Heiss G, Pankow JS, Salomaa V, Sharrett AR, et al.

(2004) Association of Lewis genotype with cardiovascular risk factors and subclinical carotid atherosclerosis: the Atherosclerosis Risk in Communities (ARIC) study. J Intern Med 255: 40-51.

41. Suadicani PA, Hein HO, Gyntelberg FA (2002) Airborne occupational exposure, ABO phenotype and risk of ischaemic heart disese in the Copenhagen Male Study. J Cardiovascular Risk 9: 191-198.
42. Nydegger UE, Wuillemin WA, Julmy F, Meyer BJ, Carrel TP (2003) Association of ABO histo-blood group B allele with myocardial Infarction. Eur J Immunogenet 30: 201-206.
43. von Beckerath N, Koch W, Wemer M, Mehilli J, Gorchakova O, et al. (2004) ABO locus O1 allele and risk of myocardial infarction. Blood coagulation and Fibrinolysis 15: 61-67.