

Assessment of Apo Lipoprotein A-I and High-Density Lipoprotein Levels in Patients with Prostate Cancer

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Abstract

Background: Apolipoprotein A-I (ApoA-I) and high-density lipoprotein (HDL) are key regulators of lipid transport and anti-inflammatory processes. Dysregulation of their levels has been implicated in cancer progression, particularly in prostate cancer, one of the most common malignancies among men worldwide. **Objective:** The current study aims to evaluate the levels of Apo lipoprotein A-I (ApoA-I) and high-density lipoprotein (HDL) in men with prostate cancer. **Materials and Methods:** One hundred eighty male patients were used in this study between December 2021 and June 2022. They were divided into four groups. The first group served as a control group. The second group included the new diagnosed patients with malignant prostate cancer. The third group involved the patient who received treatment and the last group involved the benign prostate hyperplasia. The male patients with prostate cancer were divided into three stages and grades (scores). The stages are II, III, and IV and the scores are (5), (6), and (7, 8, and 9). This study included the studying of ApoA-I and HDL in the serum of suspected patients. **Results:** The results revealed a significant decrease in both ApoA-I and HDL in prostate cancer patients compared to benign, tumor treated, and control groups. In addition, ApoA-I and HDL recorded a significant decrease in both new diagnosed and tumor treated patients at grade IV compared to grades III and II as well as 7, 8, and 9 compared to 5 and 6 in both new diagnosed and tumor treated patients. **Conclusion:** This study showed that low level ApoA-I and HDL has an important role in the increased risk of prostate cancer with the possibility of using this marker for the initial detection of cancer.

Keywords: ApoA-I, HDL, Prostate Cancer

INTRODUCTION

The lipoprotein known as high-density lipoprotein (HDL) transports cholesterol and fats in the blood. Because it assists in removing extra cholesterol from the blood and delivering it to the liver for elimination from the body, it is frequently referred to as “good cholesterol.”^[1]

HDL may control innate and adaptive immune responses and may have anti-oxidative, anti-apoptotic, and anti-inflammatory properties.^[2,3]

HDL is usually associated with another important protein component known as Apolipoprotein A-I (ApoA-I). This component is known for its role in cholesterol trafficking, cardiovascular protection, and inflammatory and immune responses modulation.^[4]

ApoA-I has a significant role in building HDL by facilitating the entry of cellular phospholipids and free cholesterol to the HDL particles, and then enhances its assembly.^[5]

Cancer cells are known to exhibit abnormal lipid metabolism. Lipids are essential for cell membrane biosynthesis, signal transduction, intracellular trafficking, cell polarization, and migration in addition to serving as an energy source. These processes are also crucial for the emergence and spread of cancer. As a result, one of the characteristics of cancer

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is changes in the regulation of lipid metabolism.^[6] HDL cholesterol variation has been linked to lung, endometrial, and colorectal cancer risks, prostate cancer.^[7-9]

Prostate cancer is the second most common cancer diagnosis in men and the fifth greatest cause of death globally.^[10] It affects the prostate, with an annual incidence rate of around 1.3 million, and its risk factors are strongly linked to increasing age and family history.^[11]

The prostate is tiny gland in the male reproductive system with an approximately 3 cm in length, similar to the size of walnut, with an average weight of 20 g. It participates in the production of about one third of the total seminal fluid.^[12]

Surgery, radiotherapy, and hormonal therapy are all still choices of treatment for doctors, taking into account the stage of the cancer and the health status of the patients.^[13,14] This study aims to find whether ApoA-I and HDL will rise or decline in prostate cancer patients and whether they are associated with the development of prostate cancer.

MATERIALS AND METHODS

Subject

The present study included patients of Iraqi men diagnosed with prostate cancer.

The male patients with prostate cancer were divided into three stages and grades. The stages are II, III, and IV and the grades are 5, 6, 7, 8, and 9 which were located as individual groups for scores 5 and 6 and united group for scores 7, 8, and 9. The patients were also further subdivided according to their ages into two groups (50–59 years) and (60–69 years).

Patients

The samples of newly diagnosed and tumor treated patients were drawn from the patients during their regular visits to Al-Forat Al-Awsat Center in Al-Najaf city in a period from December 2021 to June 2022. In contrast, the samples of benign prostate hyperplasia patients and controls were collected from private laboratories in Al-Najaf Al-Ashraf when the suspected patients visited nephrologists clinics.

The study was approved by institutional review board (IRB) of university of Kufa (#643, February 2022). The patients or their immediate first-degree members have obtained written informed consent based on the protocol described by the IRB.

Experimental design

Total number of subjects in this study were 180 consisting of 40 new diagnosed patients, 40 control groups, 50 tumor treated, and 50 benign prostate hyperplasia. The patients are subdivided into subgroups as explained in Table 1.

Blood collection

Prostate cancer patients and healthy controls were venipunctured to draw 5 mL of venous blood. Blood was collected in gel serum tubes and centrifuged at 3000 rpm for 15 min to isolate the serum. Serum was transferred into eppendorf tubes and stored at –20°C.

Estimation of serum high-density lipoproteinL

Determination of HDL levels was evaluated, using enzyme linked immune sorbent assay (ELISA) method, according to the procedures provided by the manufacturer's instructions (SUBN, China, SU-BN10627).

Estimation of serum Apolipoprotein A-I

Determination of ApoA-I levels was evaluated, using ELISA method, according to the procedures provided by the manufacturer's instructions (Solarbio, Beijing, China, SEKH-0093).

Statistical analysis

Present study data were analyzed using SPSS (Microsoft, Redmond, Washington, United States) Version 26.01, 2019 (ANOVA was used to find statistically significant mean differences between men groups). The least significant difference test determined significant variances. Microsoft Excel 2016 was used to create all figures (descriptive statistics, correlation coefficients, *P* value).

Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with patients verbal and

Table 1: Experimental study design

Sub group	Stages			Grade		
	II	III	IV	5	6	7,8,9
Control	–	–	–	–	–	–
New diagnosed	10	15	15	10	15	15
Tumor treated	10	15	25	10	15	25
Benign	–	–	–	–	–	–

analytical approval before sample was taken. The study protocol and the subject information and consent form were reviewed and approved by a local ethics committee according to the document number 121 (including the number and the date in October 12, 2021) to get this approval.

RESULTS

Estimation of serum high-density lipoprotein and Apo lipoprotein A-I in prostate cancer patients and control group

HDL

The result in Figure 1 indicated a significant decrease ($P < 0.05$) in serum levels of HDL in new diagnosed (6.76 ± 0.74), tumor treated (15.19 ± 1.2), and benign (9.80 ± 0.77) compared with control group (84.92 ± 1.93). The results also showed significant increase ($P < 0.05$) in tumor treated compared with new diagnosed group, whereas no significant difference was recorded between benign and new diagnosed groups. The results also showed

a significant decrease ($P < 0.05$) in benign compared with tumor treated group.

Apo lipoprotein A1

The results shown in Figure 2 indicate a significant decrease ($P < 0.05$) in new diagnosed (34.45 ± 1.89), tumor treated (44.36 ± 1.24), and benign (40.7 ± 2.38) groups in comparing with control group (174.76 ± 1.23). The tumor treated and benign groups had a significant increase ($P < 0.05$) compared with new diagnosed group, whereas no significant change was recorded between benign and tumor treated groups.

Levels of high-density lipoprotein and Apo lipoprotein A-I in patients with prostate cancer and their relationship with cancer stages

HDL [Figure 3] indicates a significant increase ($P < 0.05$) in the level of HDL in tumor treated patients at stage II (29.11 ± 1.95), stage (III) (17.27 ± 0.95), and stage (IV) (8.43 ± 0.39), compared with new diagnosed patients (13.86 ± 1.82) at stage (II), (5.41 ± 0.21) at stage (III), and (3.37 ± 0.9) at stage (IV) respectively.

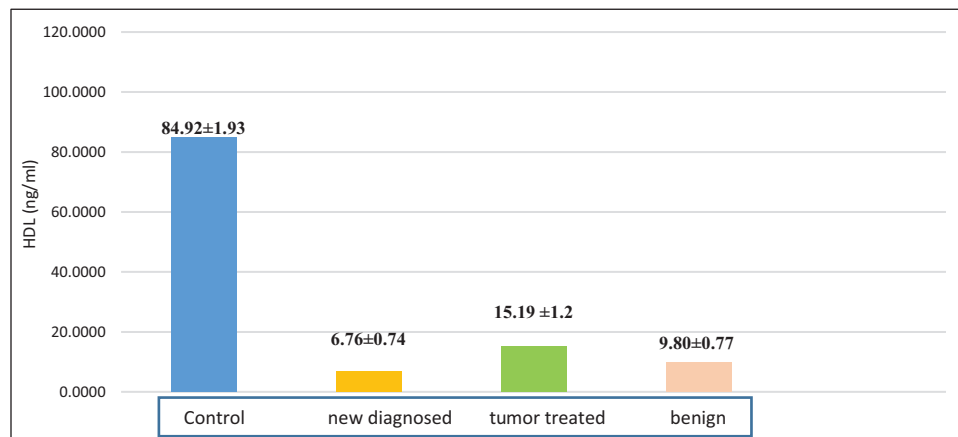


Figure 1: Comparison of serum HDL level in new diagnosed, tumor treated and benign prostate cancer patients compared with control. P value < 0.05 is considered significant. $N = (40)$ Cont., $N = (40)$ New, $N = (50)$ Tumor 9, $N = (50)$ Benign

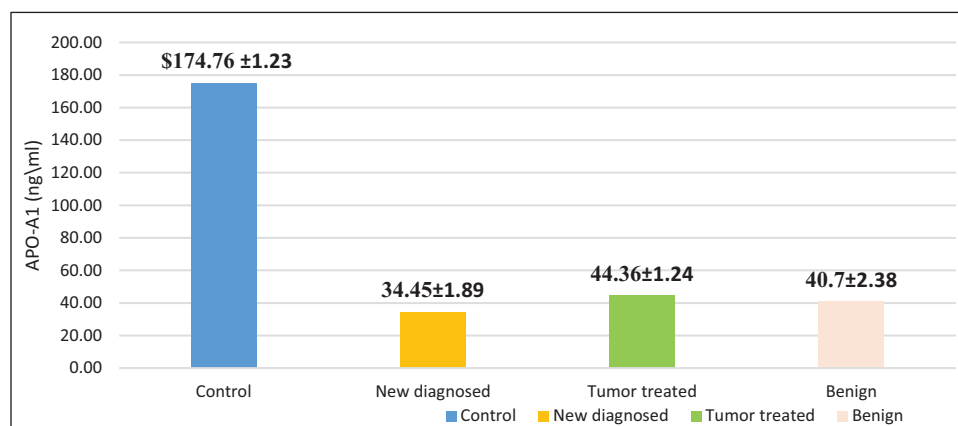


Figure 2: Comparison of serum Apo lipoprotein A1 level in new diagnosed, tumor treated, and benign prostate cancer patients compared with control. P value < 0.05 is considered significant. $N = (40)$ Cont., $N = (40)$ New, $N = (50)$ Tumor 9, $N = (50)$ Benign

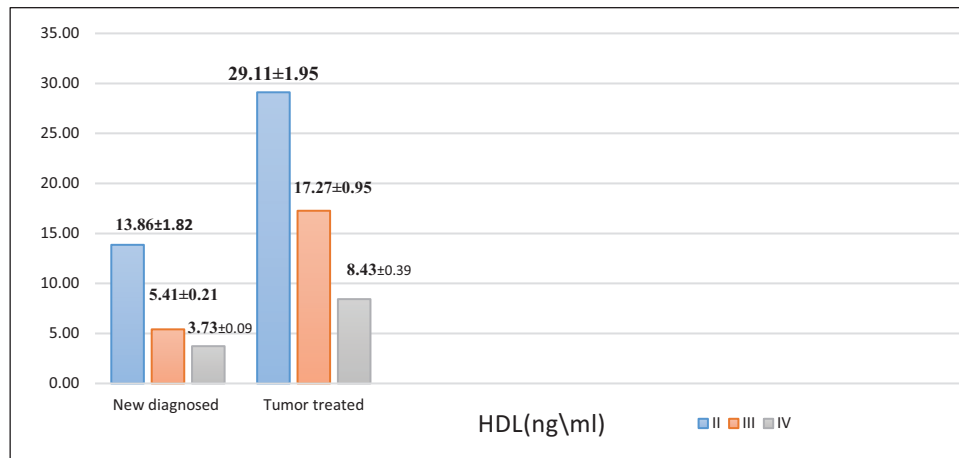


Figure 3: Comparison of serum HDL level in in new diagnosed, tumor treated compared with cancer stages. P value < 0.05 is considered significant. $N = (40)$ New, $N = (50)$ Tumor treated

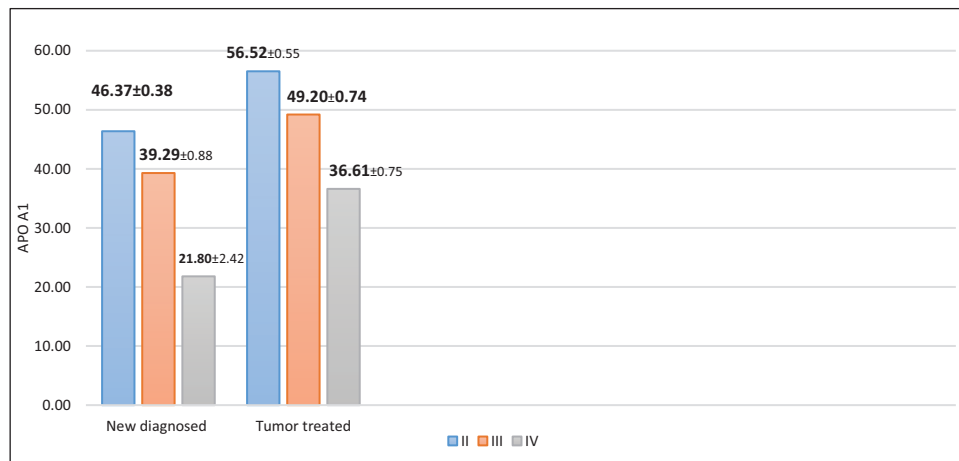


Figure 4: Comparison of serum ApoA-I level in in new diagnosed, tumor treated compared with cancer stages. P value < 0.05 is considered significant. $N = (40)$ New, $N = (50)$ Tumor treated

The result of present study revolved that the new diagnosed patients who were at stage (II) showed a significant increase ($P < 0.05$) compared with stages (III) and (IV) of the same group (new diagnosed), whereas there was no significant change among stages (III) and (IV) of the same group as well. Moreover, the results of the tumor treated patients who were at stage (II) showed a significant increase ($P < 0.05$) compared with stages (III) and (IV) of the same group (tumor treated). Also, the tumor treated at stage (III) reordered a significant increase ($P < 0.05$) compared with stage (IV) of the tumor treated group as well.

Apo lipoprotein A-I

Figure 4 showed a significant increase ($P < 0.05$) in the level of ApoA-I in tumor treated patients at stage II (56.52 ± 0.55), stage (III) (49.20 ± 0.74), and stage (IV) (36.61 ± 0.75), compared with new diagnosed patients (46.37 ± 0.38) at stage (II), (39.29 ± 0.88) at stage (III), and (21.80 ± 2.42) at stage (IV), respectively.

The result of present study revolved that the new diagnosed patients who were at stage (II) showed a significant increase ($P < 0.05$) compared with stages (III) and (IV) of the same group (new diagnosed). Also, the tumor treated at stage (III) reordered a significant increase ($P < 0.05$) compared with stage (IV) of the tumor treated group as well. Moreover, the results of the tumor treated patients who were at stage (II) showed a significant increase ($P < 0.05$) compared with stages (III) and (IV) of the same group (tumor treated). Also, the tumor treated at stage (III) reordered a significant increase ($P < 0.05$) compared with stage (IV) of the tumor treated group as well.

Levels of high-density lipoprotein and Apo lipoprotein A-I in patients with prostate cancer and their relationship with cancer grades

High-density lipoprotein

Figure 5 shows a significant increase ($P < 0.05$) in the level of HDL in tumor treated patients at score (5)

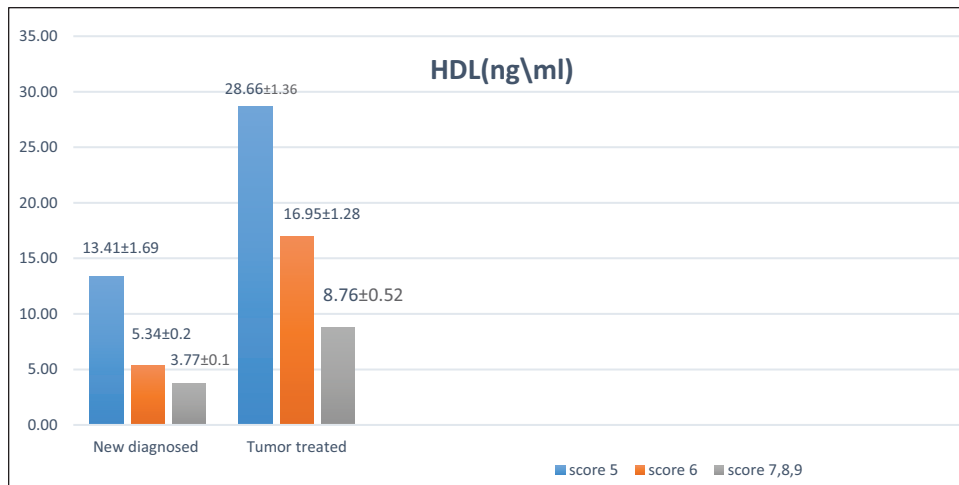


Figure 5: Comparison of serum HDL level in in new diagnosed, tumor treated compared with cancer grades. P value < 0.05 is considered significant. $N = (40)$ New, $N = (50)$ Tumor treated

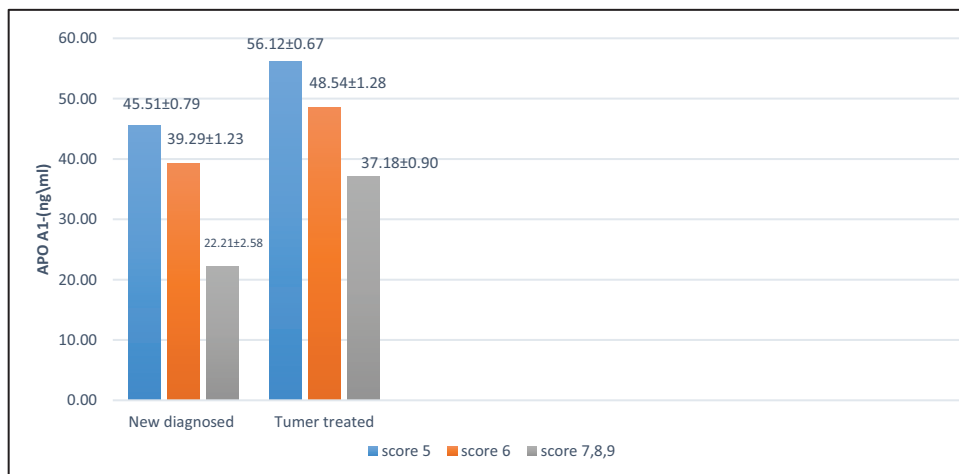


Figure 6: Comparison of serum Apo lipoprotein A1 level in in new diagnosed, tumor treated compared with cancer grades. P value < 0.05 is considered significant. $N = (40)$ New, $N = (50)$ Tumor treated

(28.66 ± 1.36), score (6) (16.95 ± 1.28), and scores (7, 8, and 9) (8.76 ± 0.52), compared with new diagnosed patients at score (5) (13.41 ± 1.69), score (6) (5.34 ± 0.2) and scores (7, 8, and 9) (0.49 ± 0.05), respectively.

The result of present study revolved that the new diagnosed patients who were at score (5) showed a significant increase ($P < 0.05$) compared with score (6) and scores (7, 8, and 9) of the same group (new diagnosed). Also, the tumor treated at score (6) reordered a significant increase ($P < 0.05$) compared with scores (7, 8, and 9) of the tumor treated group as well. Moreover, the results of the tumor treated patients who were at score (5) showed a significant increase ($P < 0.05$) compared with score (6) and scores (7, 8, and 9) of the same group (tumor treated). Also, the tumor treated at score (6) reordered a significant increase ($P < 0.05$) compared with scores (7, 8, and 9) of the tumor treated group as well.

Apo lipoprotein A-I

Figure 6 shows a significant increase ($P < 0.05$) in the level of ApoA-I in tumor treated patients at score (5) (56.12 ± 0.67), score (6) (48.54 ± 1.28), and scores (7, 8, and 9) (37.18 ± 0.90) in comparison with new diagnosed patients at score (5) (45.51 ± 0.79), score (6) (39.29 ± 1.23) and scores (7, 8, and 9) (22.21 ± 2.58), respectively.

The result of present study revolved that the new diagnosed patients who were at score (5) showed a significant increase ($P < 0.05$) compared with score (6) and scores (7, 8, and 9) of the same group (new diagnosed). Also, the tumor treated at score (6) reordered a significant increase ($P < 0.05$) compared with scores (7, 8, and 9) of the tumor treated group as well. Moreover, the results of the tumor treated patients who were at score (5) showed a significant increase ($P < 0.05$) compared with score (6) and scores (7, 8, and 9) of the same group (tumor treated). Also, the tumor treated at score (6) reordered a significant increase

($P < 0.05$) compared with scores (7, 8, and 9) of the tumor treated group as well.

DISCUSSION

The results that were explained in Figures 1 and 2 indicate a significant decrease in the level of HDL and ApoA-1 in new diagnosed prostate cancer compared with tumor treated, benign prostate hyperplasia and control group. The relationship of HDL/ApoA-1 with most types of cancers such as colorectal, lung, and prostate is still controversial.^[15,16] ApoA-1 plays an important role as an antitumor, where angiogenesis is reduced and cholesterol efflux is enhanced with reverse cholesterol transport. All these effects will inhibit proliferation, growth, and development of cancer.^[17,18] Our results agreed with some previous studies where the decrease in ApoA-1 in prostate cancer patients, will maximize the influx of reverse cholesterol to cancer cells on the behalf of efflux reverse cholesterol leading to its accumulation in cells and enhancement of cancer cell proliferation.^[19] ApoA-1 in cancer has been shown to have a major role in trafficking cholesterol which in turn will regulate and modulate inflammation and immune response.^[20,21] Moreover, ApoA-1 and HDL have not only a role in prostate cancer but also in other types of cancers such as hepatocellular carcinoma.^[22,23] In contrast, HDL/ApoA-1 may be considered a potential marker for early prediction of cancers such as prostate, gastrointestinal tract, adenocarcinoma of stomach and colon cancer.^[24] HDL/ApoA-1 have been found by many studies to have a protective role against all types of cancers and those studies concluded that ApoA-1 can be used for the detection and better prevention of cancer. In addition, during cancer therapy, high serum ApoA-1 has been detected in breast cancer patients after taking chemotherapy.^[25] ApoA-1 has also a protective property against all types of cancers by having an anti-inflammatory property. At a decreased level of ApoA-1, many inflammatory factors may be activated such as Toll-like receptor-4, nuclear factor-kappa B, IL-6, and various oxide lipids and enzymes that participate in inflammation such as cyclooxygenase 2.^[26-29]

Figures 3 and 4 indicate a significant decrease in HDL and ApoA-1, respectively, where low levels detected at high stage (IV) compared with stages III and III for both new diagnosed and tumor treated. Figures 5 and 6 show low level of HDL and ApoA-1, respectively, where low levels detected at high score (7, 8, and 9) compared with scores 6 and 5 for both new diagnosed and tumor treated. The aggressiveness of prostate cancer (indicated by high stage (IV) and high grades at scores (7, 8, and 9)) has been found by many other studies to be strongly related with HDL and ApoA-1 due to its role in the development of cancer. They also revealed a positive correlation between low HDL level and incidence and progression of prostate cancer.^[30,31]

CONCLUSION

ApoA-I and HDL levels have been studied in relation to prostate cancer in men. Studies have suggested that men

with higher levels of ApoA-I and HDL may have a lower risk of developing prostate cancer. Additionally, some research has suggested that men with prostate cancer may have lower levels of ApoA-I and HDL compared to healthy men. However, more research is needed to confirm these findings and to determine the potential mechanisms by which ApoA-I and HDL levels may influence the development and progression of prostate cancer.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Acuña-Aravena M, Cohen DE. Lipoprotein metabolism and cholesterol balance. In: The Liver: Biology and Pathobiology. 6th ed. Hoboken, NJ: Wiley-Blackwell; 2020. p. 255-67.
2. Catapano AL, Pirillo A, Bonacina F, Norata GD. HDL in innate and adaptive immunity. *Cardiovasc Res* 2014;103:372-83.
3. Ko SH, Kim HS. Menopause-associated lipid metabolic disorders and foods beneficial for postmenopausal women. *Nutrients* 2020;12:202.
4. Abdul Hasan AM, Ewadh MJ, Aljubawii AAA. Assessment of Serum Cathepsin k and Lipid Profile in Chronic Coronary Syndrome Patients. *Med J Babylon* 2024;21:280-4.
5. Duong PT, Weibel GL, Lund-Katz S, Rothblat GH, Phillips MC. Characterization and properties of pre β -HDL particles formed by ABCA1-mediated cellular lipid efflux to apoA-I. *J Lipid Res* 2008;49:1006-14.
6. Bian X, Liu R, Meng Y, Xing D, Xu D, Lu Z. Lipid metabolism and cancer. *J Exp Med* 2021;218:e20201606.
7. Angelantonio D. Major lipids, apolipoproteins, and risk of vascular disease. *JAMA* 2009;302:1993-2000.
8. Pedersen KM, Çolak Y, Bojesen SE, Nordestgaard, BG. Low high-density lipoprotein and increased risk of several cancers: 2 population-based cohort studies including 116,728 individuals. *J Hematol Oncol* 2020;13:1-11.
9. Van Hemelrijck M, Walldius G, Jungner I, Hammar N, Garmo H, Binda E, *et al.* Low levels of apolipoprotein AI and HDL are associated with risk of prostate cancer in the Swedish AMORIS study. *Cancer Causes Control* 2011;22:1011-9.
10. Deaibil HB, Al-Dujaili AN. Immunological Study of Scavenger Receptor Class B Type I Associated with Prostate Cancer Aggressiveness and Development in Iraqi Provinces. *Med J Babylon* 2024;21(Suppl 1):S111-5.
11. Rawla P. Epidemiology of prostate cancer. *World J Oncol* 2019;10:63-89.
12. Toivanen R, Shen MM. Prostate organogenesis: Tissue induction, hormonal regulation and cell type specification. *Development* 2017;144:1382-98.
13. Mohammed SH, Al-Dujaili AN, El Katib WA. High mobility group box protein-1 (HMGB1) level in gallstones patients. *AIP Conf Proc* 2022;2398:040014-1-040014-13.
14. Shen WJ, Azhar S, Kraemer FB. SR-B1: A unique multifunctional receptor for cholesterol influx and efflux. *Annu Rev Physiol* 2018;80:95-116.
15. Jafri H, Alsheikh-Ali AA, Karas RH. Baseline and on-treatment high-density lipoprotein cholesterol and the risk of cancer in

- randomized controlled trials of lipid-altering therapy. *J Am Coll Cardiol* 2010;55:2846-54.
16. Pirro M, Ricciuti B, Rader DJ, Catapano AL, Sahebkar A, Banach M. High density lipoprotein cholesterol and cancer: Marker or causative? *Progr Lipid Res* 2018;71:54-69.
 17. Cruz PM, Mo H, McConathy WJ, Sabnis N, Lacko AG. The role of cholesterol metabolism and cholesterol transport in carcinogenesis: A review of scientific findings, relevant to future cancer therapeutics. *Front Pharmacol* 2013;4:119.
 18. Zamanian-Daryoush M, Lindner D, Tallant TC, Wang Z, Buffa J, Klipfell E, *et al.* The cardioprotective protein apolipoprotein A1 promotes potent anti-tumorigenic effects. *J Biol Chem* 2013;288:21237-52.
 19. Zamanian-Daryoush M, DiDonato J. Apolipoprotein AI and cancer. *Front Pharmacol* 2015;6:265.
 20. Barker G, Winer JR, Guirgis FW, Reddy S. HDL and persistent inflammation immunosuppression and catabolism syndrome. *Curr Opin Lipidol* 2021;32:315-22.
 21. Chen W, Wu Y, Lu Q, Wang S, Xing D. Endogenous ApoA-I expression in macrophages: A potential target for protection against atherosclerosis. *Clin Chim Acta* 2020;505: 55-9.
 22. Jiang Y, Sun A, Zhao Y, Ying W, Sun H, Yang X, *et al.* Proteomics identifies new therapeutic targets of early-stage hepatocellular carcinoma. *Nature* 2019;567:257-61.
 23. Mustafa MG, Petersen JR, Ju H, Cicalese L, Snyder N, Haidacher SJ, *et al.* Biomarker discovery for early detection of hepatocellular carcinoma in hepatitis C-infected patients. *Mol Cell Proteomics* 2013;12:3640-52.
 24. Muntoni S, Atzori L, Mereu R, Satta G, Macis MD, Congia M, *et al.* Serum lipoproteins and cancer. *Nutr Metab Cardiovasc Dis* 2009;19:218-25.
 25. Zablocka-Słowińska K, Placzowska S, Skórska K, Prescha A, Pawelczyk K, Porębska I, *et al.* Oxidative stress in lung cancer patients is associated with altered serum markers of lipid metabolism. *PLoS ONE* 2019;14:e0215246.
 26. Aguirre-Portolés C, Feliu J, Reglero G, Ramírez de Molina A. ABCA1 overexpression worsens colorectal cancer prognosis by facilitating tumour growth and caveolin-1-dependent invasiveness, and these effects can be ameliorated using the BET inhibitor apabetalone. *Mol Oncol* 2018;12:1735-52.
 27. Al-Fatlawi NAG, Al-Dujaili AN, Kammona TH, Al-Dujaili HH. Assessment toll_likereceptor4(TLR4)in thrombocytopenia patients in Holy-Najaf. *AIP Conf Proc* 2022;2386:020038-1–020038-13.
 28. Gao F, Chattopadhyay A, Navab M, Grijalva V, Su F, Fogelman AM, *et al.* Apolipoprotein AI mimetic peptides inhibit expression and activity of hypoxia-inducible factor-1 α in human ovarian cancer cell lines and a mouse ovarian cancer model. *J Pharmacol Exp Ther* 2012;342:255-62.
 29. Gkouskou K, Ioannou M, Pavlopoulos G, Georgila K, Sigano A, Nikolaidis G. Apolipoprotein AI inhibits experimental colitis and colitis-propelled carcinogenesis. *Oncogene* 2016;35:2496-505.
 30. Salam S, Al-Dujaili AN. Growth differentiation factor-15 level in ischemic heart disease patients. *AIP Conf Proc* 2022;2547:020039-1–020039-7.
 31. McGrowder D, Riley C, Morrison E, Gordon L. The role of high-density lipoproteins in reducing the risk of vascular diseases, neurogenerative disorders, and cancer. *Cholesterol* 2011.