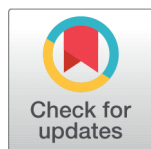


The relationship between thyroid hormones and lipid profile in subclinical hypothyroidism female patients

Kazheen H. Jawzal¹ , Mohammed A. Hami¹ , Lina Y. Mohammed² and Aveen A. Ibrahim¹

¹Department of Chemistry, Faculty of Science, University of Zakho, Zakho, Iraq

²Department of Biomedical Science, College of Medicine, University of Zakho, Zakho, Iraq



Received 26-07-2022

Revised 28-08-2022

Accepted 30-08-2022

Published 16-09-2022

Corresponding Author

Mohammed A. Hami
mohammed.hami@uoz.edu.krd

Department of Chemistry, Faculty of Science, University of Zakho, Zakho, Iraq

DOI <https://doi.org/10.47419/bjbabs.v3i03.137>

Pages: 200-209

Distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 International (CC-BY-NC 4.0), which permits use for any non-commercial purpose, distribution, and reproduction in any medium, provided that the original work is properly cited.

Copyright: © 2022 Kazheen H. Jawzal, Mohammed A. Hami, Lina Y. Mohammed, Aveen A. Ibrahim

OPEN ACCESS

ABSTRACT

Background and objectives: Subclinical thyroid disorder is a most common subclinical disease among many medical conditions such as cardiovascular disease and subclinical Lyme disease. The aim of this study was to determine the relationship between lipid profile results, lipid ratios, and anthropometric parameters and thyroid hormones.

Methods: One hundred patients (ages 20 to 50 years) with subclinical hypothyroidism (SHT group) and 50 healthy subjects (control group), who are age-matched with patients, were included in the current study. Thyroid function tests (TSH, T₃ and T₄) were determined by immunodiagnostic assay system (VIDAS) for all participants. The serum glucose and lipid profile tests parameters were evaluated by Biolis 24i Premium chemistry analyser. In addition, systolic and diastolic blood pressure were measured for each individual in the study using mercury sphygmomanometer.

Results: The levels of high-density lipoprotein-cholesterol (HDL-c) were significantly lower in the SHT group when compared with control group. Whereas, the level of low-density lipoprotein-cholesterol (LDL-c), the ratios total cholesterol (TC)/HDL-c and LDL-c/HDL-c were significantly higher in SHT group than in controls. Moreover, TSH was negatively correlated with diastolic blood pressure.

Conclusions: Both LDL-c and HDL-c are altered in subclinical hypothyroidism patients. And, there is a negative association between TSH and diastolic blood pressure.

Keywords dyslipidaemia, lipid profile, subclinical hypothyroidism, thyroid hormones, TSH

INTRODUCTION

The term “subclinical” refers to the presence of a disease without clear signs, i.e. the disease maybe at an early stage.¹ This term has been used for many medical conditions such as subclinical cardiovascular disease and subclinical Lyme disease, but subclinical thyroid

disorder is probably the most common one. Therefore, subclinical thyroid disease is known as a biochemically diagnosed disorder with abnormal serum thyrotropin (TSH) with both T₄ and T₃ are within normal range. Subclinical thyroid disease is characterized as subclinical hyperthyroidism and subclinical hypothyroidism (SHT).² The former one occurs when serum TSH concentrations are low while T₄ and T₃ concentrations are normal, and subclinical hypothyroidism occurs when serum TSH concentrations are elevated but serum thyroid hormones concentrations are normal.³

The incidence of thyroid dysfunctions in the general population could be due to many reasons, such as ethnic, age and geographical factors as well as iodine intake. The prevalence of SHT and hyperthyroidism affects 4 -10% and 1-2% of people, respectively.⁴

Thyroid hormones play an important role in regulating a variety of metabolic processes, including lipid synthesis, mobilization, and degradation.⁵ These hormones stimulate the synthesis of cholesterol which is catalysed by 3-hydroxy-3-methyl-glutaryl coenzyme A reductase in the liver. Thyroid hormones affect the activity of lipoprotein lipase which stimulates the hydrolysis of triglycerides in chylomicrons converting it to its components, fatty acids and glycerol.⁶ SHT is linked with lipid profile alterations mainly total cholesterol (TC) and low-density lipoprotein-cholesterol (LDL-c).⁷

It has been shown that the prevalence of hypothyroidism is higher among type II diabetes mellitus.⁸ Thyroid hormones have a significant role in glucose homeostasis by exerting antagonistic and agonistic effects on insulin actions in many organs. Thus, any alteration in the level of thyroid hormones would lead to defects in glucose metabolism.⁹ For example, hypothyroidism inactivate gluconeogenesis pathway leading to decrease in glucose levels.¹⁰

The main objective of this study was to determine the relationship between lipid profile tests [TC, triglyceride (TGs), high-density lipoprotein cholesterol (HDL-c), LDL-c, very low-density lipoprotein-cholesterol (VLDL-c)] and lipid ratios (TC/HDL-c and LDL-c/HDL-c), on one hand; and thyroid function tests (TFTs: T3, T4 and TSH), on the other hand. It was sought to assess the association with anthropometric parameters such as age, blood pressure (systolic and diastolic), and BMI as well.

MATERIALS AND METHODS

Study type, participants and blood sampling

This is a cross-sectional study, extended from September 2020 to December 2020 among the female population in Zakho city, Kurdistan region of Iraq. A total of 100 female patients with SHT (2.91–8.61 mIU/ml was used as a cut-off value for TSH) and age range 20 to 50 years (32.17±13.69), and 50 age-matched healthy females were recruited in the study.¹¹ Blood pressure and body mass index (BMI) were recorded in all participants. And, after an overnight fasting, 10 ml of venous blood samples using disposable syringe were collected from all participants.

Participants who are smoking, diabetic and alcoholics were excluded from the study. In addition, subjects who were taking supplements or people with elevated blood pressure

were also excluded from this study.

Biochemical analyses

Serum TSH, T₄ and T₃ were evaluated by VIDAS (BioMérieux, France), in which the principle is Enzyme Linked Fluorescent Assay (ELFA).¹² Serum glucose, and lipid profile tests were performed by Biolis 24i Premium chemistry analyser (Tokyo Boeki, Japan) using enzymatic methods¹³ on the same day in the laboratory of General Zakho Hospital, Kurdistan Region, Iraq.

The ethical approval of this protocol was obtained from the local health ethics committee. The subjects voluntarily participated in the study and the written informed consent was taken from all the subjects. All the ethical guidelines of the Declaration of Helsinki was followed.¹⁴

Statistical analysis

The levels of TSH, T₄, T₃, blood glucose and lipid profile parameters as well as BMI and blood pressure were expressed in mean and standard deviation. The general comparisons of these parameters among the group were examined using the student *t*-test and the Pearson's correlation co-efficient was used to assess the association between the parameters. A *p*-value of less than or equal to 0.05 was considered statistically significant. The statistical evaluations were performed using the Statistical Package for Social Sciences version 25 (IBM SPSS Statistics for Windows, v. 25.0. Armonk, NY).

RESULTS

The study included 100 females diagnosed with SHT (their age of 32.17±13.69 years). Blood samples were taken from the volunteers after overnight fasting and blood pressure was taken after resting the patient for 10 minutes. The results of the study relied on the analysis of the blood samples obtained from volunteers who participated in this study.

The effects of subclinical thyroid disorder on the BMI, blood pressure, serum glucose, T₄ and T₃ are shown in Table 1. The results indicate that, the SHT lowered BMI, serum glucose, systolic and diastolic blood pressure; however, the effects were non-significant as compared to control group.

The effect of SHT on serum lipid profile is presented in Table 2. In SHT, serum levels of TC, TGs and VLDL-c, were increased when compared to control group but with not significant difference. However, HDL-c, LDL-c, the ratios TC/HDL-c and LDL-c/HDL-c were significantly elevated in SHT in comparison with control group.

Table 3 shows the correlation between TSH and the studied parameters in subclinical thyroid disorder. In SHT, the TSH levels was negatively associated to diastolic blood pressure ($r = -0.482$, $p = 0.043$). While, non-significant negative correlation was found with sys-

Table 1 Anthropometric and biochemical parameters in control and SHT groups.

Variable	Control (n=50)	SHT patients (n=100)	P-value
BMI (kg/m ²)	27.44±4.27	25.78±5.12	0.29
Systolic blood pressure (mmHg)	122.81±13.65	114.47±15.42	0.09
Diastolic blood pressure (mmHg)	80.00±10.29	81.36±12.44	0.66
TSH (mIU/ml)	1.48±0.86	3.72±29.03	<0.001*
T ₄ (μg/dL)	8.59±1.18	7.72±6.03	0.55
T ₃ (ng/ml)	1.94±0.68	1.76±0.81	0.54
Serum glucose (mg/dL)	99.27±10.22	98.42±11.77	0.81

Data presented as mean±SD. *= statistically significant (unpaired t-test).

Table 2 Lipid profile parameters of control and SHT groups.

Variables	Control (n=50)	SHT (n=50)	P-value
TC (mg/dl)	162.38±28.43	182.90±47.05	0.12
Triglycerides (mg/dl)	92.61±39.23	116.59±49.94	0.11
HDL-c (mg/dl)	51.67±11.46	43.83±9.30	0.03*
LDL-c (mg/dl)	92.19±24.6	115.75±44.17	0.05*
VLDL-c (mg/dl)	18.52±7.85	23.32±9.99	0.11
TC/HDL-c	3.25±0.82	4.33±1.41	<0.001*
LDL-c/HDL-c	1.87±0.68	2.76±1.26	0.01*

Data presented as mean±SD. *= statistically significant (unpaired t-test).

tolic blood pressure, T₃, serum glucose, HDL-c and VLDL-c. And, TSH levels were positively correlated to BMI, TC, TGs and T₄ but their correlations were not significant.

Table 3 Correlation between TSH and other parameters in SHT.

Variables	r	P-value (2-tailed)
BMI (kg/m ²)	0.33	0.18
Systolic blood pressure (mmHg)	-0.34	0.17
Diastolic blood pressure (mmHg)	-0.48	0.04*
T ₄ (μg/dL)	0.13	0.61
T ₃ (ng/mL)	-0.28	0.26
Serum glucose (g/dL)	-0.22	0.36
TC (g/dL)	0.28	0.26
Triglyceride (g/dL)	0.02	0.94
HDL-c (g/dL)	-0.05	0.84
LDL-c (g/dL)	0.31	0.21
VLDL-c (g/dL)	-0.02	0.94

* denotes significant correlation (Pearson's correlation) at p<0.05.

DISCUSSION

Thyroid hormones play an important role in the regulation of cell metabolism, maintaining phospholipids levels in the cell membranes as well as the fatty acid composition of lipids. The impact of thyroid hormones is well-known in all aspects of metabolism in particular lipid metabolism including synthesis, mobilization and degradation.^{15,16} Therefore, this study aimed to assess the role of lipid profile in SHT patients.

There are so many conflicts in the results of lipid profile in the SHT patients in the literature. This is mainly due to poor control and not adjusting for the confounding factors. In our study, TC and triglyceride did not show any significant differences between control and SHT groups despite the higher levels of these two parameters in the SHT group. These two parameters did not correlate with TSH. Our results in this regard are in comparable to those of Staub et al. (1992).¹⁷ However, other studies found significant elevation in the levels of TC and triglyceride in SHT group,^{18,19} but the mean age in these studies (42 ± 13 years) were elder than our study (32.17 ± 13.69 years). Age do affect the levels of TC and triglyceride, in which these two parameters are increased directly with age.²⁰ This higher levels of TC and triglyceride indicates that SHT could be a risk factor for cardiovascular diseases.²¹ It seems geographical location and ethnic groups also show variation in the results. For example, a study in the United States²² showed significant elevations of TC and triglyceride but a European study²³ did not show significant difference. The mechanism behind the increase in TC is the direct effect of thyroid hormone on the Niemann-Pick C1-like 1 protein in the gut which leads to increase in cholesterol absorption.²⁴

Significantly lower levels of HDL-c and significantly higher levels of LDL-c in this study indicates that thyroid hormones have widespread effects on lipid profile and also indicates that SHT can be a risk factor for atherosclerosis.²⁵ These results agree with those of Wang et al. and Zha et al. However, our results do not agree with those of Vierhapper et al. who found significant difference between control and overt hypothyroidism but not control and SHT.²³ Thyroid hormones contribute in the expression of LDL-c receptor. Thus, in SHT, the level of LDL-c receptors is decreased which in turn leads to decrease in the catabolism of LDL-c.²⁴ In addition, the lower levels of HDL-c could be due to the effects of thyroid hormone on the HDL-c binding site.²⁶

Both systolic blood pressure and diastolic blood pressure is elevated in SHT but the elevation is not significant. In addition, TSH is negatively significantly associated with diastolic blood pressure. This because of T_3 is considered to be vasodilator, by having a direct effect on the vascular smooth muscle cells through affecting catecholamine and catecholamine receptor.²⁷ Our results is parallel with those of Tseng et al.²⁸

BMI did not show any significant difference between control and SHT. Our results are in comparable with those of Caraccino et al.²⁹ and Ito et al.³⁰ However, a study by Knudsen et al. found a small change in TSH would significantly affect BMI.³¹

Serum glucose levels did not affect by the change in the level of TSH despite being a little bit lower in the SHT. This difference might be more significant in the overt hypothyroidism because of the fact that thyroid hormones maintain glucose haemostasis so any alteration in the levels of thyroid hormones will obviously have an effect on glucose levels.¹⁰ In similarity

to our study, Ganie et al. and Cheserek et al. found no significant difference in serum glucose in both control and SHT groups.^{32,33} Our results are not in agreement with those of Vyakaranam et al. who found significant rise in the levels of serum glucose in the SHT.³⁴

One of the limitations of this study is the quite small number of participants. In addition, performing the analysis after one month can also be considered a limitation. For future studies, it would be more interesting to subdivide the SHT group into 2-3 grades based on the level of TSH.

CONCLUSIONS

In conclusion, thyroid hormones do affect the levels of some lipid profile parameters in SHT that can be seen by significant rise in the level of LDL-c and significant fall in the level of HDL-c. This association can be further studied by subdividing the subclinical group into mild and sever subclinical groups and studying the lipid profiles in them.

ACKNOWLEDGEMENTS

The authors would like to offer their special thanks to the Head of Chemistry Department, University of Zakho for the support and help.

DECLARATIONS

Authors' contributions

The authors have equally contributed to this work. They reviewed and approved the final draft before publication.

Conflict of interest

The authors declare no conflict of interest.

Ethical approval and consent to participate

The ethical approval of this protocol was obtained from the local health ethics committee. The subjects voluntarily participated in the study and the written informed consent was taken from all the participants. All the ethical guidelines of the Declaration of Helsinki were followed.

Data availability

The data that support the findings of this study is available from the corresponding author, upon reasonable request.

Funding resources

This work was funded by the University of Zakho.

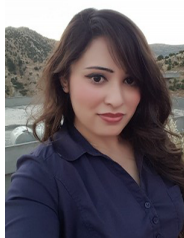
REFERENCES

1. Stedman TL. *Stedman's Medical Dictionary for the Health Professions and Nursing*. Philadelphia, USA, Lippincott Williams & Wilkins; 2005.
2. Hashimoto K. Update on subclinical thyroid dysfunction. *Endocr J*. 2022;69(7):725–738. Available from: [10.1507/endocrj.EJ22-0182](https://doi.org/10.1507/endocrj.EJ22-0182).
3. Toth PP. Subclinical atherosclerosis: what it is, what it means and what we can do about it. *Int J Clin Pract*. 2008;62(8):1246–54. Available from: [10.1111/j.1742-1241.2008.01804.x](https://doi.org/10.1111/j.1742-1241.2008.01804.x).
4. Taylor PN, Albrecht D, Scholz A. Global epidemiology of hyperthyroidism and hypothyroidism. *Nat Rev Endocrinol*. 2018;14(5):301–316. Available from: [10.1038/nrendo.2018.18](https://doi.org/10.1038/nrendo.2018.18).
5. Duntas LH, Brenta G. A renewed focus on the association between thyroid hormones and lipid metabolism. *Front Endocrinol*. 2018;9:511–511. Available from: [10.3389/fendo.2018.00511](https://doi.org/10.3389/fendo.2018.00511).
6. Loh K, Tam S, Murray-Segal L, et al. Inhibition of adenosine monophosphate-activated protein kinase-3-hydroxy-3-methylglutaryl coenzyme A reductase signaling leads to hypercholesterolemia and promotes hepatic steatosis and insulin resistance. *Hepatol Commun*. 2018;3(1):84–98. Available from: [10.1002/hep4.1279](https://doi.org/10.1002/hep4.1279).
7. Liu H, Peng D. Update on dyslipidemia in hypothyroidism: the mechanism of dyslipidemia in hypothyroidism. *Endocr Connect*. 2022;11:e210002–e210002. Available from: [10.1530/EC-21-0002](https://doi.org/10.1530/EC-21-0002).
8. Huang X, Zhang X, Zhou X, et al. Prevalence of thyroid dysfunction in a Chinese population with different glucose intolerance status: A community-based cross-sectional study. *Diabetes Metab Syndr Obes*. 2020;13:4361–4368. Available from: [10.2147/DMSO.S271328](https://doi.org/10.2147/DMSO.S271328).
9. Brenta G. Why can insulin resistance be a natural consequence of thyroid dysfunction. *J Thyroid Res*. 2011;p. 152850–152850. Available from: [10.4061/2011/152850](https://doi.org/10.4061/2011/152850).
10. Khan SH, Fazal N, Ijaz A, et al. Insulin resistance and glucose levels in subjects with subclinical hypothyroidism. *J Coll Physicians Surg Pak*. 2017;27(6):329–333.
11. Delitala AP, Fanciulli G, Maioli M, Delitala G. Subclinical hypothyroidism, lipid metabolism and cardiovascular disease. *Eur J Intern Med*. 2017;38:17–24. Available from: [10.1016/j.ejim.2016.12.015](https://doi.org/10.1016/j.ejim.2016.12.015).
12. Sukhu K, Beavis J, Baker PM, Keeling DM. Comparison of an immunoturbidometric method (STalia D-DI) with an established enzyme linked fluorescent assay (VIDAS) D-dimer for the exclusion of venous thromboembolism. *Int J Lab Hematol*. 2008;30(3):200–204. Available from: [10.1111/j.1751-553X.2007.00942.x](https://doi.org/10.1111/j.1751-553X.2007.00942.x).
13. Kaplan Ç, Erdoğan F. Effect of dietary propolis on growth, body composition, and serum biochemistry of juvenile sea bream (*Sparus aurata*). *Aquacult Int*.

- 2021;29:553–563. Available from: [10.1007/s10499-020-00642-w](https://doi.org/10.1007/s10499-020-00642-w).
14. General Assembly of the World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. *J Am Coll Dent*. 2014;81(3):14–18.
 15. Peppas M, Betsis G, Dimitriadis G. Lipid abnormalities and cardiometabolic risk in patients with overt and subclinical thyroid disease. *J Lipids*. 2011;p. 575840–575840. Available from: [10.1155/2011/575840](https://doi.org/10.1155/2011/575840).
 16. Pucci E, Chiovato L, Pinchera A. Thyroid and lipid metabolism. *Int J Obes*. 2000;24(2):109–112. Available from: [10.1038/sj.ijo.0801292](https://doi.org/10.1038/sj.ijo.0801292).
 17. Staub JJ, Althaus BU, Engler H, et al. Spectrum of subclinical and overt hypothyroidism: effect on thyrotropin, prolactin, and thyroid reserve, and metabolic impact on peripheral target tissues. *Am J Med*. 1992;92(6):631–642. Available from: [10.1016/0002-9343\(92\)90782-7](https://doi.org/10.1016/0002-9343(92)90782-7).
 18. Kvetny J, Heldgaard PE, Bladbjerg EM, Gram J. Subclinical hypothyroidism is associated with a low-grade inflammation, increased triglyceride levels and predicts cardiovascular disease in males below 50 years. *Clin Endocrinol (Oxf)*. 2004;61(2):232–238. Available from: [10.1111/j.1365-2265.2004.02088.x](https://doi.org/10.1111/j.1365-2265.2004.02088.x).
 19. Efsthadiadou Z, Bitsis S, Milionis HJ, et al. Lipid profile in subclinical hypothyroidism: is L-thyroxine substitution beneficial? *Eur J Endocrinol*. 2001;145(6):705–710. Available from: [10.1530/eje.0.1450705](https://doi.org/10.1530/eje.0.1450705).
 20. Hami MA, Al-Tamer YA, Al-Hebib OM. The association between selected elements and body mass index and age in male subjects. *Trace Elem Electroly*. 2020;37(3):139–144. Available from: [10.5414/TEX01613](https://doi.org/10.5414/TEX01613).
 21. Maleki N, Kazerouni F, Hedayati M, Rahimpour A, Maleki H. Assessment of cardiovascular risk factors in patients with subclinical hypothyroidism. *Acta Cardiol*. 2016;71(6):691–697. Available from: [10.2143/AC.71.6.3178188](https://doi.org/10.2143/AC.71.6.3178188).
 22. Canaris GJ, Manowitz NR, Mayor G, Ridgway EC. The Colorado thyroid disease prevalence study. *Arch Intern Med*. 2000;160(4):526–534. Available from: [10.1001/archinte.160.4.526](https://doi.org/10.1001/archinte.160.4.526).
 23. Vierhapper H, Nardi A, Grösser P, Raber W, Gessl A. Low-density lipoprotein cholesterol in subclinical hypothyroidism. *Thyroid*. 2000;10(11):981–984.
 24. Pearce EN. Update in lipid alterations in subclinical hypothyroidism. *J Clin Endocrinol Metab*. 2012;97(2):326–333. Available from: [10.1210/jc.2011-2532](https://doi.org/10.1210/jc.2011-2532).
 25. Zha K, Zuo C, Wang A. LDL in patients with subclinical hypothyroidism shows increased lipid peroxidation. *Lipids Health Dis*. 2015;14:95–95. Available from: [10.1186/s12944-015-0092-4](https://doi.org/10.1186/s12944-015-0092-4).
 26. Karthick N, Dillara K, Poornima KN, Subhasini AS. Dyslipidaemic changes in women with subclinical hypothyroidism. *J Clin Diagn Res*. 2013;7(10):2122–2125. Available from: [10.7860/JCDR/2013/5777.3448](https://doi.org/10.7860/JCDR/2013/5777.3448).
 27. Pesic MM, Radojkovic D, Antic S, Kocic R, Stankovic-Djordjevic D. Subclinical hypothyroidism: association with cardiovascular risk factors and components of metabolic syndrome. *Biotechnol Biotechnol Equip*. 2015;29(1):157–163. Available

- from: [10.1080/13102818.2014.991136](https://doi.org/10.1080/13102818.2014.991136).
28. Tseng FY, Lin WY, Lin CC. Subclinical hypothyroidism is associated with increased risk for all-cause and cardiovascular mortality in adults. *J Am Coll Cardiol*. 2012;60(8):730–737. Available from: [10.1016/j.jacc.2012.03.047](https://doi.org/10.1016/j.jacc.2012.03.047).
 29. Caraccio N, Ferrannini E, Monzani F. Lipoprotein profile in subclinical hypothyroidism: response to levothyroxine replacement, a randomized placebo-controlled study. *J Clin Endocrinol Metab*. 2002;87(4):1533–1538. Available from: [10.1210/jcem.87.4.8378](https://doi.org/10.1210/jcem.87.4.8378).
 30. Ito M, Arishima T, Kudo T. Effect of levo-thyroxine replacement on non-high-density lipoprotein cholesterol in hypothyroid patients. *J Clin Endocrinol Metab*. 2007;92(2):608–611. Available from: [10.1210/jc.2006-1605](https://doi.org/10.1210/jc.2006-1605).
 31. Knudsen N, Laurberg P, Rasmussen LB. Small differences in thyroid function may be important for body mass index and the occurrence of obesity in the population. *J Clin Endocrinol Metab*. 2005;90(7):4019–4024. Available from: [10.1210/jc.2004-2225](https://doi.org/10.1210/jc.2004-2225).
 32. Ganie MA, Laway BA, Wani TA. Association of subclinical hypothyroidism and phenotype, insulin resistance, and lipid parameters in young women with polycystic ovary syndrome. *Fertil Steril*. 2011;95(6):2039–2043. Available from: [10.1016/j.fertnstert.2011.01.149](https://doi.org/10.1016/j.fertnstert.2011.01.149).
 33. Cheserek M, Wu G, Shen L, Shi Y, Le G. Evaluation of the relationship between subclinical hypothyroidism and metabolic syndrome components among workers. *Int J Occup Med Environ Health*. 2014;27(2):175–187. Available from: [10.2478/s13382-014-0240-5](https://doi.org/10.2478/s13382-014-0240-5).
 34. Vyakaranam S, Vanaparthi S, Nori S, Palarapu S, Bhongir AV. Study of Insulin Resistance in Subclinical Hypothyroidism. *Int J Health Sci Res*. 2014;4(9):147–153. 25580384.

AUTHOR BIOGRAPHY



Kazheen H. Jawzal is an assistant lecturer of Clinical biochemistry at the Department of Chemistry, Faculty of Science, University of Zakho and the coordinator of the Chemistry Department and coordinator of Quality Assurance (Zakho, Iraq). She got her B.Sc. in Chemistry from the Department of Chemistry University of Duhok (Duhok, Iraq) in 2011; and her M.Sc. in Clinical biochemistry from the Department of Chemistry, University of Zakho (Zakho, Iraq) in 2016. Her main research interests include: clinical lipidology, atherogenic indices, hormones, and enzymes.



Mohammed A. Hami is a lecturer at the Chemistry Department of University of Zakho and also the Manager of Journals of University of Zakho. He received his B.Sc. in Chemistry from the University of Duhok (Duhok, Iraq) in 2010, and his master's degree in Clinical biochemistry from Keele University (United Kingdom) in 2013. He got a Ph.D. in Clinical biochemistry from the University of Zakho (Duhok, Iraq) in 2019.

His research interests are: diabetes, obesity, oxidative stress, and trace elements.



Lina Y. Mohammed is a lecturer of Biochemistry at the Department of Biomedical Sciences, College of Medicine, University of Zakho, and Director of the Postgraduate Unit at the College of Medicine (Zakho, Iraq). She got her B.Sc. in Chemistry from the Department of Chemistry, University of Al-Mustansiriyah (Baghdad, Iraq) in 1996 and her M.Sc. in Biochemistry from the Department of Chemistry, University of Al-Mustansiriyah in 1999. Her Ph.D. degree in Biochemistry was received in 2018 from the University of Bristol, School of Chemistry (Bristol, United Kingdom). Lina's main research interests include: protein chemistry, protein mass spectrometry, clinical biochemistry, and nanotechnology.



Aven A. Ibrahiem is a lecturer of Biochemistry at the Department of Chemistry, Faculty of Science, University of Zakho (Duhok, Iraq). She got her B.Sc in Chemistry from the Department of Chemistry, University of Mosul (Mosul, Iraq) in 1997; and her M.Sc. in Biochemistry from the Department of Chemistry, University of Dohuk (Dohuk, Iraq) in 2007. Her main research interests include: biochemistry, and natural products.