sciendo

Original Paper

Biomedical Human Kinetics, 16, 278–284, 2024
DOI: 10.2478/bhk-2024-0029

# Body fat, lipid profile and selected hormones in active male Polish students characterized by lower and higher normal thyroid-stimulating hormone levels

Marzena Malara, Patrycja Widłak

Department of Human Biology, Faculty of Physical Education, Józef Piłsudski University of Physical Education, Warsaw, Poland

## Abstract

*Study aim*: To evaluate the relationship between body fat, lipid profile and selected hormones in the context of lower and higher normal thyroid-stimulating hormone (TSH) levels in active male students.
*Material and methods*: A total of 112 male students of the Faculty of Physical Education volunteered to participate in the study. Body weight, height and waist circumference were measured using standard medical equipment. The percentage of body fat was determined from the sum of the thickness of four skinfolds. Circulating lipids, TSH, glucose and insulin level were measured.
*Results*: Biochemical characteristics revealed that the circulating total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triacylglycerol (TG) levels were significantly lower in students with lower normal TSH levels vs. students with higher normal TSH levels. The analysis of distorted metabolic variables indicated that for students with higher normal TSH levels the frequency of disturbances was significantly higher than in students with lower normal TSH levels. Additionally, this group was characterized by a significantly higher percentage of fat in the body.
*Conclusion*: In young, physically active men, certain metabolic disorders related to plasma lipid profiles may be observed despite normal TSH levels.

Key words: Body fat – Lipid profile – Hormones – Physical activity

## Introduction

The endocrine system is one of the most crucial systems in the human body, playing an important role in maintaining homeostasis. It significantly influences the survival of the organism by managing the functions of all systems and organs, affecting the body's immune response and its ability to combat diseases. Disruptions in its functioning can lead to the improper operation of other systems and affect various bodily processes, including metabolic ones [1, 25].

Presently, numerous scientific studies in databases indicate that abnormal thyroid hormone levels negatively affect the body's homeostasis, including energy and lipid metabolism. This observation is critical as lipid metabolism disorders can predispose individuals to cardiovascular diseases, dyslipidemia, and atherosclerosis. There is evidence linking increased TSH levels to higher total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) levels. It is also worth noting that cortisol affects thyroid function, as high cortisol levels can reduce the production of TSH, potentially leading to hypothyroidism [3, 9, 10, 36].

However, it is important to note that even borderline TSH values, both at the upper and lower limits of normal, impact lipid levels in the body. Studies have identified a relationship among euthyroid subjects between elevated TSH levels and higher TG levels, and between lower TSH levels and decreased TC and LDL-C levels in subjects with TSH in mid-range and low normal values [7, 39].

Additionally, subclinical hypothyroidism has been observed to affect blood lipid levels and reduce the size of LDL-C fractions, which may contribute to atherosclerosis and heart diseases [4, 9, 17]. Subclinical hypothyroidism may also contribute to the development of metabolic syndrome [24]. Studies have noted that subclinical hypothyroidism is associated with lower levels of small high-density lipoprotein cholesterol (HDL-C) subfractions that exhibit vascular protective effects [16, 26].

Author's address Patrycja Widłak, Department of Human Biology, Faculty of Physical Education, Józef Piłsudski University of Physical Education, Marymoncka 34 Street, 00-968 Warsaw, Poland patrycja.widlak@awf.edu.pl

Conversely, subclinical hyperthyroidism has been linked to lower levels of large HDL-C subfractions and higher average HDL-C subfractions [17]. However, this issue has been studied mainly in middle-aged and older adults due to the severity of thyroid disease [18]. It is worth emphasizing that there are few reports on the metabolic effects of variability in normal circulating TSH levels in young adults, and their results are interpreted jointly for male and female participants [28].

These findings prompted researchers to investigate whether there is a relationship between TSH concentration and the lipid profile in the studied group of young men.

## Materials and methods

### Subjects

A total of 112 male students of the Faculty of Physical Education volunteered to participate in the study. The participants were recruited on the basis of word of mouth and advertisements in student dormitories. All volunteers reported having no health problems, did not smoke and were not taking supplements on a regular basis. All participants were participating in different types of regular physical activity according to the study program (martial arts, swimming, games). The average age of the research participants was 21 years, body weight was 79 kg, and body height was 182 cm. Before the study all subjects provided their written consent for participation in all procedures. The study protocol was accepted by the local ethics commission at the University of Physical Education (ethics committee approval number SKE 01-01/2015).

### Anthropometric measurements

In all participants after all outer clothing and shoes had been removed body mass and height were measured using standard medical equipment. Body mass was measured to the nearest 0.1 kg and body height to the nearest 0.5 cm. Body mass index (BMI) was calculated as body mass (kg) divided by height (m) squared. Waist circumference was measured to the nearest 0.1 cm using a non-stretchable tape. The percentage of body fat was determined from the sum of the thickness of four skin folds (biceps, triceps, suprascapular and subscapular), which was measured using a Harpenden Skinfold Caliper (British Indicators, Burgess Hill, UK) and calculated according to Durnin and Womersley [12]. Each measurement was repeated twice, and in the event of discrepancies, it was repeated again.

### Biochemical analyses

Participants were required to report for blood sampling at least 12 hours after their last meal. Blood was collected between 7:30 and 9:00 a.m. from the antecubital vein under aseptic conditions into plastic tubes with an anticoagulant, and then centrifuged for 15 min/4000 rpm at 40°C to obtain plasma. Plasma was stored at –70°C until assays were performed. Triacylglycerols (TG), total cholesterol (TC) and HDL cholesterol (HDL-C) were determined using colorimetric methods and appropriate kits (Randox Laboratories, UK). Glucose was determined in plasma using the GOD-PAP method. The coefficients of variation for all parameters did not exceed 5. The concentration of LDL cholesterol (LDL-C) in plasma was calculated using the Friedewald formula [13]. Plasma insulin, cortisol and TSH levels were determined using radioimmunoassay methods and commercial BioSource kits (Belgium). Inter – and intra-assay coefficients of variation for hormones did not exceed 7%. All measurements were done in duplicate. The Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) index was calculated according to the following formula [20]:

$$\text{HOMA-IR} = [\text{glucose (mmol/L)} \times \text{insulin (}\mu\text{IU/ml)}] /22.5.$$

Concentrations of plasma lipoproteins were classified according to the recommendations of the National Institute of Health (HDL-C <40 mg/dl, ≥60 mg/dl; LDL-C <100 mg/dl, ≥100 mg/dl; TG <150 mg/dl, ≥150 mg/dl; TC <200 mg/dl, ≥200 mg/dl), glucose 70–99 mg/dl, insulin 2.6–24.9 µIU/ml, cortisol 4.82–19.5 µg/dL [23]. The cut-off value of HOMA-IR was established at the level of the 75$^{th}$ percentile (2.188 for males) [15, 33]. The results of circulating TSH were used to identify subjects with normal circulating hormone TSH levels established in the range 0.4–4.0 µIU/ml according to Ruderich [30].

### Energy expenditure

The Seven-Day Physical Activity Recall Questionnaire (SDPAR) was used in the study. It is a reliable and widely used questionnaire that assesses levels of physical activity [8, 31].

Data were obtained from the participants regarding the duration, intensity and frequency of activity, as well as sleep. The different types are expressed as MET 1.5, 4, 6 and 10 and then presented in kcal/day. In addition, the total daily energy expenditure (DEE, kcal/day), being the sum of basal metabolic rate, energy expended on physical activity and the thermal effect of food, was estimated.

### Energy intake

Energy intake (EI) was analyzed on the basis of 24-hour nutritional interviews collected during the 4 days preceding blood collection, including 2 weekdays and 2 days off. Daily food intake was assessed using a Photo Album of Products and Dishes presenting the types and sizes of food in grams [35], and then analyzed using a computer program and the Dieta 5.0 computer program purchased at the Institute of Food and Nutrition in Warsaw (Poland).

**Statistical analysis**

All data were tested for normality using the Shapiro-Wilk test. Statistical significance was tested using the Mann-Whitney test. The Pearson chi-square test was used to analyze the differences between the frequency of abnormal lipid levels and the TSH level. The Spearman rank correlation coefficients between TSH levels and other circulating variables were calculated. The frequency of optimal and higher than optimal circulating lipoprotein and HOMA-IR were calculated. Data are presented as mean ± SD. The value of $P < 0.05$ was accepted as significant. All calculations were carried out using Statistica v.12 (StatSoft, Illinois, USA).

## Results

Subjects with lower and higher normal TSH levels did not differ with respect to age, weight, height, BMI, WC, DEE or EI. However, a significant difference was noted in the FAT % ($P < 0.02$) (Table 1).

Biochemical characteristics revealed that the circulating TC, LDL-C, and TG were significantly lower in subjects with lower normal TSH levels vs. subjects with higher normal TSH levels ($P < 0.02$ for TSH, LDL-C and TG, $P < 0.001$ for TC). However, circulating glucose, HDL-C, cortisol, insulin and HOMA-IR did not differ significantly between the groups (Table 2).

**Table 1.** Characteristics of participants with lower and higher normal TSH levels (mean ± SD)

| Variables | Lower normal TSH levels (0.4–2.49 μIU/ml) (n = 60) | Higher normal TSH levels (2.5–4.0 μIU/ml) (n = 52) |
|---|---|---|
| Age [years] | 21.12 ± 2.15 | 20.93 ± 1.55 |
| Weight [kg] | 77.18 ± 9.38 | 80.83 ± 11.59 |
| Height [cm] | 181.90 ± 6.87 | 182.38 ± 6.35 |
| Fat [%] | 10.80 ± 4.40 | 12.95 ± 5.16 [a] |
| BMI | 23.32 ± 2.78 | 24.27 ± 3.07 |
| WC [cm] | 77.43 ± 4.76 | 78.98 ± 8.27 |
| DEE [kcal/day] | 2466 ± 368 | 2505 ± 318 |
| EI [kcal/day] | 2844 ± 760 | 2887 ± 940 |

BMI – body mass index; Fat (%) – body fat percentage; WC – Waist circumference; DEE – daily energy expenditure; EI – energy intake; [a]$p < 0.02$ – significantly different vs. students with lower normal TSH levels.

**Table 2.** Biochemical characteristics of students with lower and higher normal TSH levels (mean ± SD)

| Variables | Lower normal TSH levels (0.4-2.49 μIU/ml) (n = 60) | Higher normal TSH levels (2.5-4.0 μIU/ml) (n = 52) |
|---|---|---|
| TSH [μIU/ml] | 1.75 ± 0.35 | 3.50 ± 1.00 [b] |
| Glucose [mg/dl] | 85.21 ± 6.92 | 84.81 ± 5.31 |
| TC [mg/dl] | 155.67 ± 31.76 | 202.48 ± 21.99 [a] |
| HDL-C [mg/dl] | 57.24 ± 11.40 | 55.69 ± 12.04 |
| LDL-C [mg/dl] | 84.11 ± 25.56 | 90.77 ± 24.73 [b] |
| TG [mg/dl] | 71.55 ± 31.40 | 87.77 ± 32.47 [b] |
| Cortisol [μg/dl] | 15.23 ± 3.55 | 15.87 ± 3.37 |
| Insulin [μIU/ml] | 5.08 ± 2.20 | 5.57 ± 2.49 |
| HOMA-IR | 1.07 ± 0.48 | 1.17 ± 0.54 |

TC – total cholesterol; HDL-C – high-density lipoprotein cholesterol; LDL-C – low-density lipoprotein cholesterol; TG – triacylglycerols; TSH – thyroid-stimulating hormone; HOMA-IR – Homeostatic Model Assessment for Insulin Resistance; [a]$p < 0.001$ – significantly different vs. students with lower normal TSH levels; [b]$p < 0.02$ – significantly different vs. students with lower normal TSH levels.

The analysis of distorted metabolic variables indicated that for male higher normal TSH levels frequency of disturbances was significantly higher than in male lower normal TSH levels (11.5% vs.1.7% for HDL-C, 30.8% vs.16.7% for LDL-C). On the contrary, the frequency of disturbed plasma glucose, TC, TG, cortisol and HOMA-IR did not differ in both groups (Table 3).

Classification of concentrations of lipids in subjects with lower and higher normal TSH levels revealed that similar percentages of both groups were characterized by optimal and above optimal TC levels (86.67% vs. 86.54% for optimal levels, 13.34% vs. 13.47% for above optimal) and TG levels (96.67% vs. 94.23% for optimal levels, 3.34% vs. 5.77% for above optimal). In students with lower normal TSH levels 1.67% had low and 33.34% had high HDL-C levels. However, in students with higher normal TSH levels 11.54% had low and 30.77% had high HDL-C levels. Optimal LDL-C levels were found in 83.34% of students with lower normal TSH levels and 69.23% of students with higher normal TSH levels, but above optimal LDL-C levels were found in 16.67% of students with lower normal TSH levels and 30.76% of students with higher normal TSH levels (Table 4).

**Table 3.** Frequency distribution of unhealthy levels of biochemical variables in physically active male students with lower normal and higher normal circulating TSH

| Variables | Lower normal TSH levels (0.4–2.49 μIU/ml) (n = 60) | Higher normal TSH levels (2.5–4.0 μIU/ml) (n = 52) |
|---|---|---|
| Glucose [mg/dl] | none | none |
| TC [mg/dl] | | |
| >200 mg/dl] | 3.3^ (2)* | 5.8 (3) |
| HDL-C [mg/dl] | | |
| <40 mg/dl | 1.7 (1) | 11.5 (6) [a] |
| LDL-C [mg/dl] | | |
| >100 mg/dl | 16.7 (10) | 30.8 (16) [b] |
| TG [mg/dl] | | |
| >150 mg/dl | 3.3 (2) | 5.8 (3) |
| Cortisol [μg/dl] | none | none |
| HOMA-IR | | |
| >2.188 | 1.7 (1) | 5.8 (3) |

For abbreviations see Table 2. ^ – percentage of participants; * – number of subjects; [a]$p < 0.02$ [b]$p < 0.05$.

In both groups of men, there was a weak positive correlation between TSH and some lipid parameters, glucose and cortisol. Conversely, there was a weak negative correlation between TSH and other lipid parameters and insulin, suggesting some associations between TSH levels and markers of metabolism and lipid profile (Table 5).

**Table 5.** Spearman rank correlation coefficients between TSH levels and other circulating variables in subjects with lower and higher normal TSH levels

| | Lower normal TSH levels (0.4–2.49 μIU/ml) (n = 60) | Higher normal TSH levels (2.5–4.0 μIU/ml) (n = 52) |
|---|---|---|
| TG [mmol/L] | –0.035 | 0.057 |
| TC [mmol/L] | 0.020 | 0.157 |
| HDL-C [mmol/L] | 0.140 | –0.026 |
| LDL-C [mmol/L] | –0.042 | 0.164 |
| Fat [%] | 0.147 | –0.092 |
| Glucose [mg/dl] | 0.141 | 0.035 |
| Cortisol [μg/dl] | 0.136 | 0.091 |
| Insulin [μIU/ml] | –0.132 | –0.182 |

For abbreviations see Table 2.

**Table 4.** Classification of concentrations of TC, HDL-C, LDL-C and TG in subjects with lower and higher normal TSH levels

| | Lower normal TSH levels (0.4–2.49 μIU/ml) (n = 60) | Higher normal TSH levels (2.5–4.0 μIU/ml) (n = 52) | $Chi^2$ (p) 3.0720 (0.3807) |
|---|---|---|---|
| **TC** | | | |
| Optimal (<200 mg/dl) | 86.67^ (52)* | 86.54 (45) | |
| Above optimal (≥200 mg/dl) | 13.34 (8) | 13.47 (7) | |
| **HDL-C** | | | |
| Low (<40 mg/dl) | 1.67 (1) | 11.54 (6) | |
| High (≥60 mg/dl) | 33.34 (20) | 30.77 (16) | |
| **LDL-C** | | | |
| Optimal (<100 mg/dl) | 83.34 (50) | 69.23 (36) | |
| Above optimal (≥100 mg/dl) | 16.67 (10) | 30.76 (16) | |
| **TG** | | | |
| Optimal (<150 mg/dl) | 96.67 (58) | 94.23 (49) | |
| Above optimal (≥150 mg/dl) | 3.34 (2) | 5.77 (3) | |

TC – total cholesterol; HDL-C – high-density lipoprotein cholesterol; LDL-C – low-density lipoprotein cholesterol; TG – triacylglycerols; ^ – percentage of participants; * – number of subjects.

A higher percentage of men with higher normal TSH levels have HOMA-IR values above the specified cut-off value (2.188) compared to men with lower normal TSH levels (Table 6).

**Table 6.** Percentage of participants with higher value HOMA-IR (cut-off > 2.188) in subjects with lower and higher normal TSH levels

| | Lower normal TSH levels (0.4–2.49 μIU/ml) (n = 60) | Higher normal TSH levels (2.5–4.0 μIU/ml) (n = 52) |
|---|---|---|
| HOMA-IR >2.188 | 1.67^ (1)* | 5.76 (3) |

HOMA-IR – Homeostatic Model Assessment for Insulin Resistance; ^ – percentage of the group; * – number of subjects.

## Discussion

Diseases of the thyroid gland are among the most common endocrine disorders globally, second only to diabetes mellitus [11, 29]. It is widely known that thyroid dysfunction, particularly hypothyroidism, is associated with dyslipidemia, increasing the risk of endothelial dysfunction, hypertension, and cardiovascular diseases [6, 27]. The most significant conclusion from our study is that even TSH concentrations at the upper limit of the norm influence the occurrence of some metabolic disorders in young, physically active men. These men had significantly higher levels of adipose tissue and serum TC, LDL-C and TG levels compared to men whose TSH levels were at the lower end of the reference range. This is consistent with other data indicating a direct influence of thyroid hormones on lipid and lipoprotein metabolism [2, 19]. It is known that hypothyroidism is associated with hyperlipidemia through modifications in lipid synthesis, absorption, circulation, and metabolism [21, 34]. A significant decrease in TC and LDL-C levels has been demonstrated after administering thyroxine in a small group of patients with hypercholesterolemia, where the baseline TSH levels were within the upper range of normal values. In our study, the frequency of unhealthy levels, especially LDL-C and HDL-C, is particularly noteworthy in men with higher normal TSH levels. Literature evidence suggests that hypothyroidism leads to an increase in gastrointestinal cholesterol absorption and a decrease in cell-surface LDL-C receptors, resulting in reduced plasma LDL-C clearance. Decreased clearance of LDL-C implies that the body does not efficiently remove LDL-C from the bloodstream, posing a risk factor for the development of non-alcoholic fatty liver disease [5, 14].

In our study, a higher percentage of participants with higher values of HOMA-IR was also observed in males with higher normal TSH levels. These results are consistent with reports from other authors, as HOMA-IR was increased in patients with hypothyroidism and subclinical hypothyroidism, potentially increasing the risk for atherogenesis and cardiovascular disease due to increased insulin resistance [37, 38].

It is important to underline that this study has limitations due to the small number of participants. Moreover, this is a cross-sectional study. Our data indicate that significant metabolic disorders may affect young, slim, and physically active men with higher plasma TSH concentrations, even within the reference range. However, in many studies, such disturbances are observed only in the subclinical form of hypothyroidism, where TSH levels in the serum exceed the norm [9]. Simultaneously, it cannot be ruled out that the cause of metabolic disorders is an unfavorable change in lifestyle or diet. It has been shown that the fast pace of life among students, who are often both studying and working, does not support making healthy dietary choices. Students are particularly vulnerable to dietary irregularities not only due to their irregular schedules but also due to frequent financial or organizational challenges in meal preparation [22, 32].

**Conflict of interest: Authors state no conflict of interest.**

## References

1. Anbazhagan R., Kavarthapu R., Mathur P.P., Kostic T.S., Prakash H. (2021) Editorial: Systemic Regulation of Organ Homeostasis and Implications of Hormones and Immunity. *Front. Endocrinol.* (Lausanne), 12: 740835. DOI: 10.3389/fendo.2021.740835.
2. Asvold B.O., Vatten L.J., Nilsen T.I., Bjøro T. (2007) The association between TSH within the reference range and serum lipid concentrations in a population-based study. The HUNT Study. *Eur. J. Endocrinol*., 2007 156(2): 181-6. DOI: 10.1530/eje.1.02333. Erratum in: *Eur. J. Endocrinol.,*156(6): 707.
3. Atrooz O.M., Hiresh M.N., Dlewan A.R., Atrooz M.O., Hiresh G.N., Alasoufi A.M., Atrooz I.O. (2023) Prevalence of dyslipidemia and the association with levels of TSH and T4 hormones among patients in south region of Jordan. *J. Med. Biochem.,* 42(4): 706-713. DOI: 10.5937/jomb0-40504.
4. Beukhof C.M., Massolt E.T., Visser T.J., Korevaar T.I.M., Medici M., de Herder W.W. et al. (2018) Effects of thyrotropin on peripheral thyroid hormone metabolism and serum lipids. *Thyroid,* 28: 168-174. DOI: 10.1089/thy.2017.0330.

5. Buzzetti E., Pinzani M., Tsochatzis E.A. (2016) The multiple-hit pathogenesis of non-alcoholic fatty liver disease (NAFLD). *Metabolism,* 65(8): 1038-1048. DOI: 10.1016/j.metabol.2015.12.012.
6. Chen Y., Chen Y., Wang N., Chen C., Nie X., Li Q., Han B., Lu Y. (2018) Thyroid Stimulating Hormone Within the Reference Range is Associated with Visceral Adiposity Index and Lipid Accumulation Product: A Population-Based Study of SPECT-China. *Horm. Metab. Res.,* 50(1): 29-36. DOI: 10.1055/s-0043-122235.
7. Chin K.Y., Ima-Nirwana S., Mohamed I.N., Aminuddin A., Johari M.H., Ngah W.Z. (2014) The relationships between thyroid hormones and thyroid-stimulating hormone with lipid profile in euthyroid men. *Int. J. Med. Sci.*, 11(4): 349-355. DOI: 10.7150/ijms.7104.
8. Czeczelewska E., Czeczelewski J., Wasiluk A., Saczuk J. (2016) Evaluation of the Usability of Selected Questionnaires Assessing Physical Activity in the Prophylaxis of Cardiovascular Diseases. *Adv. Clin. Exp. Med.,* 25(1): 59-67. DOI: 10.17219/acem/39157.
9. Delitala A.P., Fanciulli G., Maioli M., Delitala G. (2017) Subclinical hypothyroidism, lipid metabolism and cardiovascular disease. *Eur. J. Intern. Med.*, 38: 17-24. DOI: 10.1016/j.ejim.2016.12.015.
10. Delitala A.P., Scuteri A., Maioli M., Mangatia P., Vilardi L., Erre G.L. (2019) Subclinical hypothyroidism and cardiovascular risk factors. *Minerva Med.,* 110(6): 530-545. DOI: 10.23736/S0026-4806.19.06292-X.
11. Deschampheleire M., Luyckx F.H., Scheen A.J. (1999) Dysthyroïdies et dyslipidémies [Thyroid disorders and dyslipidemias]. *Rev. Med. Liege.,* 54(9): 746-750. [in French].
12. Durnin J.V., Womersley J. (1974) Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *Br. J. Nutr.,* 32(1): 77-97. DOI: 10.1079/bjn19740060.
13. Friedewald W.T., Levy R.I., Fredrickson D.S. (1972) Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin. Chem.,* 18(6): 499-502.
14. Gälman C., Bonde Y., Matasconi M., Angelin B., Rudling M. (2008) Dramatically increased intestinal absorption of cholesterol following hypophysectomy is normalized by thyroid hormone. *Gastroenterology,* 134(4): 1127-1136. DOI: 10.1053/j.gastro.2008.01.032.
15. Hedblad B., Nilsson P., Janzon L., Berglund G. (2000) Relation between insulin resistance and carotid intima-media thickness and stenosis in non-diabetic subjects. Results from a cross-sectional study in Malmö, Sweden. *Diabet. Med.,* 17(4): 299-307. DOI: 10.1046/j.1464-5491.2000.00280.x.
16. Janovsky C.C.P.S., Generoso G., Goulart A.C., Santos R.D., Blaha M.J., Jones S., Toth P.P., Lotufo P.A. et al. (2020) Differences in HDL particle size in the presence of subclinical thyroid dysfunctions: The ELSA-Brasil study. *Atherosclerosis,* 312: 60-65. DOI: 10.1016/j.atherosclerosis.2020.08.021.
17. Janovsky C.C.P.S., Goulart A.C., Generoso G., Santos R.D., Blaha M.J., Jones S., Toth P.P., Lotufo A.A. et al. (2022) Substantially elevated TSH, not traditional clinical subclinical thyroid disorder groupings, are associated with smaller LDL-P mean size: ELSA-Brasil. *J. Clin. Lipidol.,* 16(3): 335-344. DOI: 10.1016/j.jacl.2022.03.010.
18. Lo Sasso B., Vidali M., Scazzone C., Agnello L., Ciaccio M. (2019) Reference interval by the indirect approach of serum thyrotropin (TSH) in a Mediterranean adult population and the association with age and gender. *Clin. Chem. Lab. Med.,* 57(10): 1587-1594. DOI: 10.1515/cclm-2018-0957.
19. Luxia L., Jingfang L., Songbo F., Xulei T., Lihua M., Weiming S., Ying N., Gaojing J. et al. (2021) Correlation Between Serum TSH Levels Within Normal Range and Serum Lipid Profile. *Horm. Metab. Res.,* 53(1): 32-40. DOI: 10.1055/a-1191-7953.
20. Matthews D.R., Hosker J.P., Rudenski A.S., Naylor B.A., Treacher D.F., Turner R.C. (1985) Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia,* 28(7): 412-9. DOI: 10.1007/BF00280883.
21. Mavromati M., Jornayvaz F.R. (2021) Hypothyroidism-Associated Dyslipidemia: Potential Molecular Mechanisms Leading to NAFLD. *Int. J. Mol. Sci.,* 22(23): 12797. DOI: 10.3390/ijms222312797.
22. Moszak M., Szulińska M., Bogdański P. (2020) You Are What You Eat – The Relationship between Diet, Microbiota, and Metabolic Disorders – A Review. *Nutrients,* 12(4): 1096. DOI: 10.3390/nu12041096.
23. National Institutes of Health; National Heart Lung, and Blood Institute; 2002 National Cholesterol Education Program. Third report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III): Final Report. NIH Publication No. 02-5215. September 2002.
24. Peng P., Wang Q., Zhou Y., Hao Y., Chen S., Wu Q., Li M., Wang Y. et al. (2023) Association of subclinical hypothyroidism with metabolic syndrome and its components among outpatients with first-episode drug-naïve major depressive disorder: a large-scale cross-sectional study. *Eur. Arch. Psychiatry Clin. Neurosci.,* DOI: 10.1007/s00406-023-01588-9.
25. Pidugu V.K., Kostic T.S., Andric S.A., Tharappel A.M., Koshimizu T.A. (2023) Editorial: Systemic regulation of organ homeostasis and implications of hormones and immunity, Volume II. *Front. Endocrinol.* (Lausanne), 14: 1235274. DOI: 10.3389/fendo.2023.1235274.
26. Post A., Garcia E., Gruppen E.G., Kremer D., Connelly M.A., Bakker S.J.L., Dullaart R.P.F. (2022) Higher

Free Triiodothyronine Is Associated With Higher HDL Particle Concentration and Smaller HDL Particle Size. *J. Clin. Endocrinol. Metab.,* 107(5): e1807-e1815. DOI: 10.1210/clinem/dgac044. PMID: 35106588.
27. Pucci E., Chiovato L., Pinchera A. (2000) Thyroid and lipid metabolism. *Int. J. Obes. Relat. Metab. Disord.,* 24 Suppl 2: S109-12. DOI: 10.1038/sj.ijo.0801292.
28. Rahbar A.R., Kalantarhormozi M., Izadi F., Arkia E., Rashidi M., Pourbehi F., Daneshifard F., Rahbar A. (2017) Relationship between Body Mass Index, Waist-to-Hip Ratio, and Serum Lipid Concentrations and Thyroid-Stimulating Hormone in the Euthyroid Adult Population. *Iran. J. Med. Sci.,* 42(3): 301-305.
29. Regmi A., Shah B., Rai B.R., Pandeya A. (2010) Serum lipid profile in patients with thyroid disorders in central Nepal. *Nepal Med. Coll. J.,* 12(4): 253-256.
30. Ruderich F., Feldkamp J. (2022) Subklinische Hypothyreose [Subclinical hypothyroidism]. *Dtsch. Med. Wochenschr.,* 147(6): 289-294. [in German]. DOI: 10.1055/a-1612-4816.
31. Sallis J.F., Buono M.J., Roby J.J.., Micale FG., Nelson J.A. (1993) Seven-day recall and other physical activity self-reports in children and adolescents. *Med. Sci. Sports Exerc.,* 25(1): 99-108. DOI: 10.1249/00005768-199301000-00014.
32. Schoeler M., Caesar R. (2019) Dietary lipids, gut microbiota and lipid metabolism. *Rev. Endocr. Metab. Disord.,* 20(4): 461-472. DOI: 10.1007/s11154-019-09512-0.
33. Shashaj B., Luciano R., Contoli B., Morino G.S., Spreghini M.R., Rustico C., Sforza R.W., Dallapiccola B. et al. (2016) Reference ranges of HOMA-IR in normal-weight and obese young Caucasians. *Acta Diabetol.,* 53(2): 251-260. DOI: 10.1007/s00592-015-0782-4.
34. Shrestha N. (2011) Thyroid dysfunction and its effect in serum lipids. *J. Nepal Health Res. Counc.,* 9(1): 33-37.
35. Szponar L., Wolnicka K., Rychlik E. (2000). Album of photographs of food products and dishes. Warsaw: National Food and Nutrition Institute.
36. Wang J.J., Zhuang Z.H., Shao C.L., Yu C.Q., Wang W.Y., Zhang K., Meng X.B., Gao J. et al. (2021) Assessment of causal association between thyroid function and lipid metabolism: a Mendelian randomization study. *Chin. Med. J.* (Engl)., 134(9): 1064-1069. DOI: 10.1097/CM9.0000000000001505.
37. Yang N., Yao Z., Miao L., Liu J., Gao X., Fan H., Hu Y., Zhang H. et al. (2015) Novel Clinical Evidence of an Association between Homocysteine and Insulin Resistance in Patients with Hypothyroidism or Subclinical Hypothyroidism. *PLoS One,* 10(5): e0125922. DOI: 10.1371/journal.pone.0125922.
38. Yang W., Jin C., Wang H., Lai Y., Li J., Shan Z. (2023) Subclinical hypothyroidism increases insulin resistance in normoglycemic people. *Front. Endocrinol.* (Lausanne), 14: 1106968. DOI: 10.3389/fendo.2023.1106968.
39. Zhang Y., Lu P., Zhang L., Xiao X. (2015) Association between lipids profile and thyroid parameters in euthyroid diabetic subjects: a cross-sectional study. BMC *Endocr. Disord.,* 15: 12. DOI: 10.1186/s12902-015-0008-3.

**Received 26.02.2024**
**Accepted 06.08.2024**



**Acknowledgements**

**This study was supported by the grants DS-230 from the Józef Piłsudski University of Physical Education, Warsaw, Poland.**