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EFFECT OF ELECTRONIC SMOKING (VAPING) ON THYROID HORMONES LEVEL AND LIPID PROFILE IN MEN

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In the last period, the market quickly became saturated with vaping devices available in many flavors and forms appealing to youth. Both traditional cigarette and e-cigarette smoking are known to potentially alter metabolic processes, including hormone production and to increase the risk of lung, heart and kidney diseases. The aim of the study was to estimate the level of thyroid hormones and lipids in the blood of young men who smoked traditional or e-cigarettes. A case-control study involved 200 men aged 24-25 years who smoked 5-7 h per day, divided into two groups (100 e-cigarette smokers and 100 cigarette smokers) and 50 healthy men who did not smoke. The levels of thyroid-stimulating hormone (TSH), free thyroxine (FT4), and free triiodothyronine (FT3) were measured using COBAS E411. The levels of total cholesterol, TG, LDL and HDL were estimated spectrophotometrically. No significant changes were found in thyroid hormone levels or lipid profiles, except for an increased TG content in the group of traditional cigarette smokers compared to the control group. Whereas in the blood of e-smokers, the increase in the level of FT3 and FT4 and a significant decrease in that of TSH, as well as the elevated content of total cholesterol, TG, and LDL, were detected compared to the control group. The results obtained indicate that e-smoking affects the function of the thyroid gland and lipid metabolism.

K e y w o r d: vaping, thyroid hormones, lipid profile, vaping, blood samples.

Many adolescents are fond of vaping devices, which have become the predominant method for teenagers in the United States to consume nicotine. Some research suggests that many children are unaware that vape cartridges include nicotine, mistakenly believing that pods only contain flavoring [1]. This demographic is attracted to these gadgets due to their accessibility, attractive marketing campaigns, diverse range of options, and perceived superiority over traditional cigarettes. They are discreet and can be easily concealed from parents and instructors due to their lack of cigarette smell and their resemblance to flash drives. 25% of high school students surveyed reported using e-cigarettes for dripping, a practice involving inhaling vapors created by placing e-liquid droplets directly into heated atomizer coils [1, 2]. Preliminary research suggests that vaping may serve as a gateway substance for preteens and adolescents who then move on to using other nicotine-containing products, such as cigarettes, which are well-known for their association with disease and premature death. A study found that students who had used e-cigarettes before starting ninth grade were more inclined to start using cigarettes and other smokable tobacco products within the next year [2, 3]. Further research confirms that high school students who did not use e-cigarettes in the past month were about seven times more likely to acknowledge smoking cigarettes when asked six months later compared to those who had used e-cigarettes in the previous month [3, 4]. Findings on European adult smokers revealed that

individuals who used nicotine vaping were less inclined to have successfully stopped smoking compared to those who did not vape. Individuals who used e-cigarettes consumed a greater quantity of cigarettes compared to those who did not. More than 800 individuals in a separate study reported in European Union countries using vaping as a method to aid in quitting traditional cigarette smoking. Just 9% of the individuals reported successfully quitting [5, 6]. In 2016, the U.S. Food and Drug Administration (FDA) started regulating e-cigarettes as novel tobacco products. The market quickly became saturated with throwaway devices available in many flavors, forms, and colors appealing to youth. In 2019, an epidemic of e-cigarette or vaping use-associated lung injury (EVALI) resulted in 2,807 hospitalizations and 68 deaths [7, 8]. EVALI is a severe and potentially dangerous lung condition associated with the use of e-cigarettes or vaping products. The outbreak was ultimately linked to a chemical that was added to certain vaping devices [9]. In 2020, the FDA implemented a partial prohibition on certain flavors due to their attractiveness to young people [10]. Thyroid function problems are common in the general population. Smoking it is increasingly acknowledged that variations in thyroid function, indicated by levels of thyroid stimulating hormone (TSH), free thyroxine (FT4), and free triiodothyronine (FT3) within normal ranges, can impact various health issues, such as vulnerability to atherosclerosis [11, 12]. Subtle fluctuations in thyroid activity within the normal range may influence life span; however, this has not been conclusively documented. Hence, it is crucial to gain a deeper understanding of how different lifestyle factors impact thyroid function [13, 14]. Cigarette or electronic smoking is known to potentially impact thyroid function, either due to exposure to harmful byproducts, increased sympathetic nervous system activity, or influencing autoimmune reactions targeting the thyroid. During the 1980s, it was reported that quitting smoking led to an increase in TSH levels and a drop in T4 levels [15-17]. Recent data indicates a correlation between smoking cigarettes and decreased TSH levels and increased FT4 levels, with T3 and FT3 levels showing an increase or remaining stable. Cotinine levels in serum or urine were utilized to gauge nicotine intake, according to S. Kim et al., 2019 [17]. Additionally, individuals who smoke exhibit reduced TSH levels, according to Gruppen et al., 2020, and Lee et al., 2021 [18, 19]. The aim of this research is to assess the effect of chemicals, including flavorings and glycerin, on thyroid function and lipids levels and the relationship between them.

Materials and Methods

The study design. A case-control study involved 200 blood samples from men who smoked (100 e-cigarette smokers, 100 cigarette smokers) and 50 samples from healthy men who did not smoke. The samples were age-matched. Samples were collected from subjects who visited Al-Habbobi Teaching Hospital,Thi-Qar, Iraq to conduct routine confirmatory examinations. Some of them were accompanied for the period between 10/1/2024 and 29/3/2024, and complete information was taken from the patients, including age. A 5 ml blood sample was drawn from each participant, placed in a gel tube, left for 30 min at room temperature until clotting, and quickly separated using a centrifuge [20]. The blood serum was separated using a centrifuge at 3000 rpm for 15 min and frozen at -20°C until use. The level of the lipid profile was quantified using a spectrophotometer [21]. The levels of thyroid hormones were assessed using the COBAS E411. Ages less than 18 years were excluded; women and thyroid diseases were excluded.

Human free triiodothyronine, free-T3 COBAS E411 Kit. Free T3 is measured within 18 min using electrochemical immunoluminescence (ECLIA) devices. The procedure begins by mixing 15 µl of a sample with anti-T3 antibodies bound to the ruthenium complex. In the second step, magnetic particles coated with biotinylated T3 and streptavidin bind to empty binding sites on antibodies, forming an antibody-haptin complex. Particles are held inside the measuring cells by a magnet and unbound material is removed, and the photochemical release is detected by a photodetector. The device is calibrated at two points, and the barcode reading is used to obtain the standard curve for the device. The working group contains streptavidin-coated particles, ruthenium-conjugated anti-T3 antibodies, and biotinylated T3. Microparticles are added to the solution automatically before use, and codes are entered to adjust test variables. The device automatically determines the concentration for each sample, with conversion factors for different units of measurement [22].

Human free thyroxine, free-T4 COBAS E411 Kit. Free thyroxine is detected in human plasma and serum using an electrochemiluminescence immunoassay (ECLIA). The test involves two steps. In the first step, 15 µl of the sample is incubated with T4-

specific antibodies bound to the ruthenium complex, and in the second step, T4-coated particles bind to the empty binding sites, forming a stable complex. When the mixture is drawn into the measuring cell, the magnetic particles are collected and the unbound material is cleaned. The photochemical release is detected by a photodetector. In order to obtain the results, the master curve is read from the barcode of the chemicals, and the device is calibrated at two points. Particles are automatically resuspended in water before use. The chemical barcode contains the specific criteria for the test, and data can be entered manually if the barcode cannot be read. The device automatically calculates the concentration for each sample in pmol/l, ng/dl, or ng/l with conversion factors between these units [22].

Human thyroid stimulating hormone, TSH COBAS E411 Kit. TSH is estimated in human serum and plasma using the "sandwich" technique of the electrochemiluminescence assay. A 50-µl sample is used to make a complex with TSH-specific antibodies bound to both biotin and the ruthenium complex. Streptavidin-coated particles are then added in the second incubation, which bind to the biotin and form a complex on the solid phase. The magnetic particles in the measuring cell are attracted using a magnetic field, and the unbound material is eliminated before the photochemical release is detected via the photodetector when a voltage is applied to the electrode. Test results depend on the calibration curve of the device and the e-barcode. Particles are automatically resuspended before use and the barcode can be entered manually. The temperature of chemicals, opening and closing of bottles are automatically controlled, and the device automatically determines the sample concentration in IU/ml or mIU/ml [22].

Measurement of total cholesterol and high-density lipoprotein (HDL-cholesterol) concentrations. Measurement and assessments of total cholesterol and HDL-cholesterol concentrations were performed according to the manufacturer's instructions (Biolabo, France). The manufacturer catalog number was 66005, the REF for R1 was 80106 and 66007, the REF for kits was 95506, respectively.

Measurement of low-density lipoprotein (LDL-cholesterol) and triglyceride concentration. Measurement and assessments of LDL-cholesterol and triglyceride concentrations were performed according to the manufacturer's instructions (Biolabo, France). The manufacturer catalog number was 66006, the REF for kits was K1416 and 66008, the REF for kits was LP80519, respectively.

Ethical approval. Before the samples were taken, all patients who participated in this study were properly informed. The Committee on Publication Ethics at the Thi-Qar Health Directorate, Al Habbobi Teaching Hospital, gave its approval to the study (Protocol No 3345 in 8/1/2024).

Statistical analysis. In this study, all data are presented as mean ± SD. The statistical analyses were performed using SPSS (version 26) and dependent t-test (two-tailed) and independent t-test (two-tailed) for normally distributed variables. The Mann-Whitney U and Wilcoxon test was used for those variables that were not normally distributed. $P < 0.05$ was considered statistically significant.

Results and Discussion

The socio-demographic characteristics among the study groups. The average age of the control group was 25.0 ± 5.4 compared to 24.7 ± 5.3 for the case group. Analyzing smoking duration in the smoking group, we observed that smoking duration was distributed between 89.0% for those who smoked between 5 and 7 h per day and 11.0% for those who smoked between 2 and 4 hours per day. According to the study conducted by Hussain et al., 2021 [23], the percentage of smokers in Iraq was 35.0%. In contrast, the study conducted by Oudah et al., 2020 [24], showed that the percentage of smokers in Thi Qar Governorate was 19.4%. For smokers who use e-cigarettes in Thi Qar, the percentage was 10.4%. As for the percentage of smokers of traditional cigarettes in Thi Qar, according to the same study, it was recorded at 9.0% the *P* value of less than <0.001

Thyroid hormone concentration differences between the control and vaping groups. In light of the study conducted to compare thyroid hormone levels between the control group and the case group, the following results were presented: FT3 hormone levels were analyzed, and it was found that the average concentration in the control group reached 3.94 ± 0.26, while in the case group, it reached 4.60 ± 1.00. Thus, the value of ($P < 0.001$) indicates a statistically significant difference between the two groups, suggesting a significant increase in FT3 hormone levels in the case group. Second, looking at FT4 hormone levels, it was observed that the average concentration was 16.12 ± 0.42 in the control group versus 35.97 ± 5.26 in the case group. This data indicates a large and strong statistically significant ($P < 0.001$) change between the two groups in FT4

hormone levels. Finally, upon statistical analysis of TSH levels, an average concentration of 1.47 ± 0.51 was recorded in the control group, compared to a low concentration of 0.14 ± 0.04 in the case group. Also here, we found that the value of ($P < 0.001$) reinforces these results as statistically significant, indicating a sharp decrease in TSH levels in the case group (Table 1).

Thyroid hormone concentration differences between the control group and the cigarette smoking group. Thyroid function parameters were evaluated between the control and case groups. The control group included 50 people, while the case group included 100 people. The results showed the average levels of FT3 in the control group was 3.94 with a standard deviation of ±0.26, while in the case group, it was 3.68 with a standard deviation of ±1.07, with a statistical significance value of 0.75. While FT4, the average measurement in the control group was 16.12 with a standard deviation of ±0.42 compared to 16.04 with a standard deviation of ±0.95 in the case group, and the statistical value was 0.48. Regarding TSH levels, it was found that the average in the control group was 1.47 with a standard deviation of ±0.51, while the case group recorded 1.18 with a standard deviation of ±0.25, and the statistical value was 0.22. On a statistical level, the study did not show any significant differences between the two groups in the levels of FT3, FT4, and TSH, as all statistical values were higher than 0.05 (Table 2).

Differences in the lipid profile levels between the control and the vaping group. First, average TC levels were recorded at 216.93 ± 24.58 mg/dl in the control group, compared to 246.40 ± 39.13 mg/dl in the case group. The value of $P < 0.01$ indicates a statistically significant difference between the two groups, with an increase in the level of total cholesterol in the case group. As for TG levels, average measurements of 130.77 ± 25.61 mg/dl were observed in the control group compared to 174.43 ± 32.87 mg/dl in the case group, and this increase was limited with a P value of 0.02, which means that there is a significant statistical difference, but to a lesser extent than total cholesterol. Considering the levels of HDL cholesterol, no statistically significant difference was recorded between the two groups, as it was found that the average level in the control group was 34.09 ± 2.22 mg/dl compared to 33.99 ± 3.57 mg/dl in the case group, with $P = 0.89$NS (not statistically significant). Regarding LDL cholesterol, a noticeable increase appeared in the case group, as the average

Table 1. Thyroid hormone concentration differences between the control and vaping groups

Parameters	Control group, $n = 50$	Case group, $n = 100$
FT3, pg/ml	3.94 ± 0.26	4.60 ± 1.00
FT4, pmol/l	16.12 ± 0.42	35.97 ± 5.26
TSH, μIU/ml	1.47 ± 0.51	0.14 ± 0.04

Note. Mean ± SD, $P < 0.001$.

Table 2. Thyroid hormone concentration differences between the control group and the cigarette smoking group

Parameters	Control group, $n = 50$	Case group, $n = 100$	*P.* value
FT3, pg/ml	3.94 ± 0.26	3.68 ± 1.07	0.75
FT4, pmol/l	16.12 ± 0.42	16.04 ± 0.95	0.48
TSH, μIU/ml	1.47 ± 0.51	1.18 ± 0.25	0.22

Note. Mean ± SD.

level reached 129.88 ± 8.71 mg/dl compared to 117.15 ± 2.74 mg/dl in the control group. This result is supported by strong statistical significance ($P < 0.001$), which reflects a significant difference between the two groups in LDL level (Table 3).

Differences in the lipid profile levels between the control and the cigarette smoking group. There were no significant differences in TC levels between the two groups (control mean = 216.93 ± 24.58 vs. case mean = 220.76 ± 25.88) with a P value of 0.62. Triglycerides: a statistically significant difference was found (control mean = 130.77 ± 25.61 vs. case mean = 153.65 ± 28.94) with a P value of 0.001. High-density cholesterol: there was no statistically significant difference (control mean = 34.09 ± 2.22 vs. case mean = 32.44 ± 2.87) with a P value of 0.88. Low-density cholesterol: there was no statistically significant difference (control mean = 117.15 ± 2.74 vs. case mean = 119.34 ± 5.23) with a P value of 0.17. It is clear from these data that there is a noticeable difference in triglyceride levels between the two groups, while the levels of total cholesterol, HDL, and LDL remain without statistically significant differences (Table 4).

Comparison of thyroid hormone levels and lipid profile levels between cigarette smokers and electronic-cigarette smokers. Free triiodothyronine: a statistically significant increase appeared in e-ciga-

rette smokers with an average of 4.60 ± 1.00 compared to 3.68 ± 1.07 for traditional cigarette smokers ($P = 0.01$). Free thyroxine: a significant increase was recorded for e-cigarette smokers with an average of 35.97 ± 5.26 compared to 16.04 ± 0.95 for traditional smokers ($P < 0.001$). Thyroid stimulating hormone: a sharp decrease in TSH levels was found in e-cigarette smokers with an average of 0.14 ± 0.04 compared to 1.18 ± 0.25 for the first group ($P < 0.001$). Total cholesterol: e-cigarette smokers showed higher cholesterol with an average of 246.40 ± 39.13 compared to 220.76 ± 25.88 for traditional smokers ($P < 0.01$). Triglycerides: it was found to be higher in e-cigarette smokers with an average of 174.43 ± 32.87 compared to 153.65 ± 28.94 for traditional smokers ($P < 0.001$). High-density lipoprotein cholesterol: there is a statistically significant difference, as e-cigarette smokers recorded a higher average value of 33.99 ± 3.57 compared to 32.44 ± 2.87 for traditional smokers ($P = 0.01$). Low-density lipoprotein cholesterol: LDL levels in e-cigarette smokers increased statistically significantly with an average of 129.88 ± 8.71 compared to 119.34 ± 5.23 for traditional smokers ($P < 0.001$). These results indicate that there are statistically significant differences in most physiological indicators of thyroid function and blood lipid levels between smokers of traditional cigarettes and smokers of e-cigarettes (Table 5).

Smoking can alter various metabolic and biological processes, including hormone production and

Table 3. Differences in lipid profile between the control group and the vaping group

Parameters, mg/dl	Control group, *n* = 50	Case group, *n* = 100	*P.* value
TC	216.93 ± 24.58	246.40 ± 39.13	<0.01
TG	130.77 ± 25.61	174.43± 32.87	<0.001
HDL	34.09 ± 2.22	33.99 ± 3.57	0.89^{NS}
LDL	117.15 ± 2.74	129.88 ± 8.71	<0.01

Note. Mean ± SD.

Table 4. Differences in Lipid profile between the control and the cigarette smoking group

Parameters, mg/dl	Control group, *n* = 50	Case group, *n* = 100	*P.* value
TC	216.93 ± 24.58	220.76 ± 25.88	0.62
TG	130.77 ± 25.61	153.65± 28.94	0.001
HDL	34.09 ± 2.22	32.44 ± 2.87	0.88^{NS}
LDL	117.15 ± 2.74	119.34 ± 5.23	0.17

Note. Mean ± SD.

Table 5. Thyroid hormone levels and lipid profile levels between cigarette smokers and electronic- cigarette smokers

Parameters	Cigarette smokers, *n* = 100	e-cigarette smokers, *n* = 100	*P.* value
FT3, pg/ml	3.68 ± 1.07	4.60 ± 1.00	0.01
FT4, pmol/l	16.04 ± 0.95	35.97 ± 5.26	<0.001
TSH, μIU/ml	1.18 ± 0.25	0.14 ± 0.04	<0.001
TC, mg/dl	220.76 ± 25.88	246.40 ± 39.13	<0.01
TG, mg/dl	153.65± 28.94	174.43± 32.87	<0.001
HDL, mg/dl	32.44 ± 2.87	33.99 ± 3.57	0.01
LDL, mg/dl	119.34 ± 5.23	129.88 ± 8.71	<0.001

Note. Mean ± SD.

release. It can disrupt the release, binding, transport, storage, and clearance of thyroid hormones, leading to negative effects on the thyroid gland and impacting hormone levels in the bloodstream [25]. Some individuals believe that tobacco smoking may potentially induce thyroid inflammation. Extensive evidence indicates that smoking significantly impacts Graves' hyperthyroidism, particularly Graves' orbitopathy. Smoking's impact on Hashimoto's thyroiditis is less evident compared to Graves' illness. Some studies showed an inverse correlation between urine cotinine levels and TSH levels in both genders, and a positive correlation with TPO Ab levels in males [26, 27]. There are limited investigations on how smoking status affects thyroid function, as measured by serum cotinine level. In a study by Soldin et al., 237 non-pregnant women were classified into active smokers, latent smokers, and non-smokers according to their serum cotinine levels. Smokers showed decreased levels of TSH and T4 [28]. However, the researchers did not examine the dose-response relationship between cigarette intake and thyroid function. Belin et al. examined the correlation between thyroid function and serum cotinine level as a continuous variable in 15592 participants from the Third National Health and Nutrition Examination Survey III conducted in the United States from 1988 to 1994. Some studies have focused on the relationship between smoking and thyroid diseases for several years between 2004, 2009 and 2021 [28-30]. After considering age, gender, race, and urinary iodine status, researchers discovered that the likelihood of having decreased TSH levels by 1.4% with every 10 ng/ml rise in blood cotinine. The correlation between serum TSH and cotinine levels varied according to the individual's smoking status (active, passive, or non-smoker). We found that the level of cotinine in the urine was inversely correlated with the level of TSH, similar to other research findings. Even after considering factors such as age, height, weight, health habits (such as alcohol consumption and exercise), and urinary iodine levels, this remained true. This is the initial study to demonstrate the correlation between urine cotinine levels and TSH levels. There are several potential underlying reasons. Smoking may activate the thyroid gland by increasing blood levels of thyroxine-binding globulin and T3, while decreasing serum levels of TSH [31, 32]. Changes in iodide transport provide an additional mechanism. Tobacco use is associated with changes in thyroid autoimmunity, suggesting that smoking interferes with the arrangement and movement of iodide [33]. Using tobacco or e-cigarettes has been linked to several inflammatory disorders, such as rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, Graves' disease, and primary biliary cirrhosis [34]. So yet no one has completely understood the impact of burning cigarettes or electronic cigarettes on that hormone [35]. Several studies have identified a correlation between electronic smoking and reduced levels of TPO Ab. However, Cho et al. could not find a statistically significant association between these variables. Previous research primarily utilized questionnaires to identify smokers and various thresholds of TPO Ab or thyroid hormone assay to indicate the existence of thyroid autoimmunity or dysfunction [36, 37]. The study indicates that there are varying levels of thyroid hormones between smokers and the control group. T3 and T4 levels are markedly elevated. TSH levels decreased considerably. This instance demonstrates the impact of electronic smoking on thyroid function. Increased levels of thyroid hormones in smokers are associated with the impact of chemicals in cigarette smoke on the thyroid gland. Thiocyanate is one of these compounds. It is a substance that alters the thyroid gland's ability to absorb iodine, essential for producing thyroid hormones. In addition, smoking can trigger a response from the adrenal gland to release cortisol, which in turn can affect levels of thyroid hormones. Smoking also increases the levels of adrenaline and norepinephrine in the body, which can negatively affect the thyroid gland. Moreover, there is an association between smoking and an increased risk of developing autoimmune thyroid diseases such as Graves' disease and Hashimoto's thyroiditis, and these diseases may result in a change in the levels of thyroid hormones [37]. The comparative health impacts of combustible cigarettes and electronic cigarettes remain significant inquiries, as individuals often transition between tobacco products in an attempt to safeguard their well-being. This study examined significant cardiometabolic health indicators in two groups: tobacco users and non-tobacco users [38]. Our investigation found elevated levels of triglycerides and cholesterol in those who exclusively used e-cigarettes. Simultaneous use of traditional and electronic cigarettes was associated with elevated levels of triglycerides and LDL, but not with HDL. A connection between smoking and poor glucose metabolism as well as abnormal lipid profiles has been proven by previous

research through the use of smoking [38]. Higher pack years and daily cigarette consumption are both linked to cigarette use intensity, which is associated with increased levels of glucose and cholesterol. These relationships have not been extensively studied in respect to electronic cigarettes [38, 39]. A recent study investigated glucose tolerance in mouse models and a nationwide cohort, finding no links between insulin resistance, abnormal glucose levels, and the use of electronic cigarettes. The study focused on a limited sample of individuals who exclusively used electronic cigarettes or both electronic and traditional cigarettes, in contrast to those who did not use either [39]. A study comparing the lipidome profiles of long-term smokers of traditional cigarettes and electronic cigarettes showed no discernible alterations as compared to the control group. This indicates that neither of them alters the lipid makeup. They found differences in the lipid species between male and female smokers [40]. Conversely, research on the Korean general population indicated that individuals who use electronic cigarettes have a higher probability of having metabolic syndrome, elevated triglyceride levels, and reduced HDL cholesterol levels [41]. The individuals who utilized electronic cigarettes throughout the initial enrollment period were predominantly older users who had been consistently using these items for a minimum of three months. We identified unfavorable lipid ratios in e-cigarette users by the same analytical techniques previously employed with combustible cigarette users. Smoking is associated with the release of catecholamines, which leads to an increase in free fatty acids in the blood and alters cholesterol levels. The exact mechanism by which smoking affects lipid variables is not fully understood [42, 43]. These alterations have also been associated with nicotine, the addictive substance found in tobacco products. Nicotine present in e-cigarettes may play a role in altering the use of fats in the body. These variations may also stem from prolonged use of traditional cigarettes prior to transitioning to e-cigarettes [44]. We examined the lipid profiles of those who consumed pods. We observed no adverse distinctions between individuals who only utilized pod-based devices and those who employed them in conjunction with tobacco products. Early-generation products may have differed from pod-based electronic cigarettes in terms of voltage, flavorings, and nicotine content, which could explain the discrepancy. Pod-based electronic cigarettes contain nicotine salts that can have varying impacts on health. They also possess a fixed quantity of vehicle components, including polyethylene glycol and vegetable glycerol. Some early electronic cigarette devices allowed users to create their own e-liquids, enabling the creation of intricate blends. Increasing evidence indicates that specific components of electronic cigarettes can impact heart health [44]. A study on animals using electronic cigarettes found that the chemicals in the cigarettes significantly influenced the body's fat metabolism [45]. The vehicle quantities in the e-cigarette group may be associated with lipid levels, but not in the pod group. Further investigation using animal models could elucidate the reasons for the adverse effects of certain components of electronic cigarettes on lipids. The disparities observed between e-cigarette and pod users may also stem from the evolving demographics of electronic cigarette consumers. Most e-cigarette users were former smokers, while many pod users had no prior experience with flammable substances. Additionally, individuals who utilized pods tended to be younger, potentially resulting in lower total exposure to various tobacco products [45].

Conclusion. The levels of thyroid hormones differed between the two groups and were significantly higher in smokers. This indicates the effects that electronic smoking has on thyroid gland dysfunction. While lipid profile levels were also significantly different in the two groups, this indicates the effect of smoking on the cellular metabolism of fats in the body. E-smoking affects the function of the thyroid gland and also affects cellular metabolism of fats due to chemicals including glycerin and flavorings that have a toxic effect on the body.

Conflict of interest. The authors have completed the Unified Conflicts of Interest form at http://ukrbiochemjournal.org/wp-content/uploads/2018/12/coi_disclosure.pdf and declare no conflict of interest.

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ВПЛИВ ПАЛІННЯ ЕЛЕКТРОННИХ СИГАРЕТ (ВЕЙПІНГ) НА РІВЕНЬ ГОРМОНІВ ЩИТОПОДІБНОЇ ЗАЛОЗИ ТА ЛІПІДНИЙ ПРОФІЛЬ У ЧОЛОВІКІВ

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Останнім часом ринок швидко насичується привабливими для молоді пристроями для вейпінгу, які мають різноманітні смаки і форми. Відомо, що паління традиційних і електронних сигарет впливає на метаболічні процеси, а саме порушує продукування гормонів підвищує ризики розвитку захворювань легенів, серця та нирок. Мета дослідження – оцінити рівень гормонів щитоподібної залози та ліпідів у крові молодих чоловіків, які палили традиційні або електронні сигарети. У дослідженні випадок-контроль брали участь 200 чоловіків у віці 24-25 років, які палили 5-7 годин на день та 50 здорових чоловіків, які не палили (контрольна група). Чоловіків, які палили було розподілено на дві групи (100 осіб, які палили електронні сигарети, та 100 осіб, які палили традиційні сигарети). Рівні тиреоїд-стимулюючого гормону (TSH), вільного тироксину (FT4) і вільного трийодтироніну (FT3) вимірювали за допомогою COBAS E411. Загальний холестерол, ТГ, ЛПНЩ і ЛПВЩ визначали спектрофотометрично. Нами не було виявлено значних змін у рівні гормонів щитоподібної залози та ліпідних профілях, за винятком підвищеного вмісту ТГ у групі курців традиційних сигарет порівняно з контрольною групою. У той же час, у крові курців електронних сигарет було встановлено підвищення FT3 і FT4 та значне зниження рівня TSH, а також підвищений вміст загального холестерину, ТГ і ЛПНЩ порівняно з контрольною групою. Зроблено висновок, що паління електронних сигарет впливає на функцію щитоподібної залози та ліпідний обмін.

Ключові слова: вейпінг, гормони щитоподібної залози, ліпідний профіль, зразки крові.

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