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Original Article

Relationship between thyroid function tests and bioelectrical impedance measurement parameters according to degree of obesity

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ABSTRACT

Aims: This study aimed to investigate the relationship between thyroid function tests and bioelectrical impedance measurement parameters showing body composition in groups of patients with varying degrees of obesity as determined by body mass index (BMI).

Methods: A total of 120 people between the ages of 18 and 65 years were enrolled in the study, including 20 individuals with stage 1 obesity, 20 with Stage 2 obesity, 20 with Stage 3 obesity, and 40 with normal body weights. In the collection of data, a case report form prepared by the researcher for gathering information about the participants and the results of blood tests and bioelectrical impedance measurements made with a Tanita device were used. Blood samples were taken from the antecubital vein between 09:00 and 10:00 in the morning after at least 8 hours of fasting. HOMA-IR values were calculated by measuring fasting plasma glucose and insulin.

Results: No significant correlation was found between median BMI and TSH, fT3 and fT4 levels in the whole population. While a negative correlation was found between median TSH and BMI in the group with normal body weight (r=-0.430, p=0.006), a positive correlation was found in the Stage 1 obese group (r=0.553, p=0.011). This relationship was not significant in the Stage 2 and morbidly obese groups. There was no significant correlation between BMI and fT4 and fT3 in all groups. A positive correlation was found between fat percentage and TSH (r=0.391, p=0.014) and fT3 (r=0.333, p=0.038) levels in the group with normal body weight, while no correlation was found with T4 levels.

Conclusion: No statistically significant differenceswere found in terms of thyroid functions in different obesity classes as determined by BMI. Elevations of thyroid-stimulating hormone in obese patients are thought to be a result of obesity, not the cause.

Keywords: Obesity, morbid obesity, thyroid, free T3, free T4, thyroid-stimulating hormone

INTRODUCTION

Although the morbidities caused by obesity have been known since 2500 BC, its prevalence hasbeen gradually increasing throughout history.[1] Obesity, a chronic disease, is an epidemic public health problem. Its prevalence is regularly monitored in most countries due to its increasing incidence. Its prevalence in the United States was stated to be between 20.2% and 36.2%.[2] In another study conducted in the United States, it was reported that it had increased from 22.9% to 34.9% between 1988 and 2012.[3] The findings in Turkey are similar, as the prevalence of obesity was found to be approximately 32% in the TURDEP II study, in which 26,499 individuals were evaluated.[4]

Considering that obesity causes many complications such as atherosclerosis, malignancy, cardiovascular diseases, metabolic syndrome, respiratory failure, and diabetes, its impact on public health is significant and it constitutes the basis of serious health expenditures.[5,6]

Various methods are used in the evaluation of obesity. Anthropometric measurements, body mass index (BMI), and body components evaluated by assistive devices are frequently preferred because they are easy and fast to apply and they do not entail radiation. In addition, measurements of body fat or water percentage by computed tomography, magnetic resonance imaging, ordual-energy X-ray absorptiometry (DXA) may also be used.

Thyroid hormones regulate the metabolic processes necessary for normal growth and development.[7] It is known that thyroid hormone levels are closely related to body weight and energy expenditure. Since a hypermetabolic state occurs in hyperthyroidism, defined asexcessive levels of thyroid hormones, resting energy expenditures increase, weight loss is observed, cholesterol levels decrease, and lipolysis and gluconeogenesis increase.[7] On the contrary, in hypothyroidism, which is defined as a decrease in thyroid

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hormones, resting energy expenditures decrease, weight gain is observed, cholesterol levels increase, and lipolysis and gluconeogenesis decrease.[10] It has been stated that even slight variations from the reference range in thyroid functions can cause weight gain and the development of localized obesity.[11] Thyroid-stimulating hormone (TSH) levels in obese children, adolescents, and adults have been shown to be at the upper end of the normal range or slightly elevated.[12]

Bioelectrical impedance measurements, which are frequently utilized for both practical and research purposes, can be used to evaluate total body water, total fat percentage, and the difference between them. Body fat percentage is thought to correlate better with obesity-related health risks compared to BMI.[5,6] The present study was undertaken to investigate the relationships between thyroid function tests and bioelectrical impedance measurement parameters showing body composition among patients with varying degrees of obesity as determined by BMI.

METHODS

Ethics

The study was carried out with the permission of Kırıkkale University Faculty of Medicine Clinical Researches Ethics Committee (Date: 29.11.2016, Decision No: 23/04). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Purpose and Type of Research

This study aimed to evaluate the effect of thyroid hormones on the differences in bioelectrical impedance measurement parameters among different obesity groups.

Place and Time of Research

Our research was conducted on patients who applied to the diabetes-obesity outpatient clinic of Kırıkkale University Faculty of Medicine between 29.11.2016 and 30.12.2016.

Population and Sample of the Research

A total of 120 people between the ages of 18 and 65 years were enrolled in the study, including 20 individuals with stage 1 obesity, 20 with stage 2 obesity, 20 with stage 3 obesity, and 40 with normal body weight. Obesity was evaluated with BMI, a common screening tool calculated with the formula of body weight (kg)/height (m^2). BMI values of <18.5 kg/m^2 reflect underweight, 18.5-24.9 kg/m^2 normal weight, 25-29.9 kg/m^2 overweight, 30-34.9 kg/m^2 class 1 obesity, 35-39.9 kg/m^2 class 2 obesity, and ≥40 class 3 or morbid obesity.[13]

Inclusion Criteria for Research

1. Those between the ages of 18-65
2. BMI of ≥25 kg/m^2 (being overweight or obese) for patients and BMI of 18-24.9 kg/m^2 for the control group
3. Not using steroids or hormonal therapy for any reason
4. Not having an eating disorder such as bulimia or anorexia nervosa
5. Volunteer by signing the informed consent form

Exclusion Criteria

1. Those who are not between the ages of 18-65
2. Refusing to participate in the study
3. Having a communication difficulty
4. Presence of Diabetes mellitus
5. Using steroids or hormonal therapy for any reason
6. Presence of chronic liver or kidney disease
7. Use of orlistat or a similar drug that inhibits fat absorption
8. Having thyroid disease and using a thyroid hormone or anti-thyroid agent, or having a history of thyroid hormone or anti-thyroid agent use, or a history of antidepressant or antipsychotic drug use
9. Pregnancy
10. Malignancy
11. Engaging in another study

Data Collection Tools

In the collection of data, a case report form prepared by the researcher for gathering information about the participants and the results of blood tests (TSH, free T3 [fT3], free T4 [fT4], insulin, HOMA-IR, fasting blood glucose [FBG], low-density lipoprotein cholesterol [LDL]) and bioelectrical impedance measurements made with a Tanita device (Tanita BC-420 MA) were used. Blood samples were taken from the antecubital vein between 09:00 and 10:00 in the morning after at least 8 hours of fasting. HOMA-IR values were calculated by measuring fasting plasma glucose and insulin. The formula was as follows: HOMA-IR=fasting plasma glucose (mmol/L) × fasting plasma insulin (mUmL)/22.5.

Case Report Form for Personal and Disease Characteristics

The demographic characteristics (sex and age), laboratory findings (TSH, fT3, fT4, FBG, insulin, HOMA-IR, and LDL) and bioelectrical impedance measurements (height [cm], weight [kg], body mass index [kg/m^2], fat mass [kg], muscle mass [kg], bone mass [kg], fat percentage [%], basal metabolic rate [kcal]) of the participants were recorded in the case report form.

Bioelectrical Impedance Measurements

Bioelectrical impedance analysis was performed with the Tanita-BC 420 MA device. This device has a frequency of 50-60Hz and current of 1.5 A. Its use is based on the difference of the electrical permeability of different tissues. Participants were required to not eat anything for at least 4 hours before the measurementsor drink anything containing caffeine, not enter a sauna or bath or consume alcohol 24 hours before the measurements, and not engage in sports on the day of the measurements. While the measurements were being taken, individuals were asked to stand on the metal surface of the device with bare feet and position their arms parallel to the body. The measurements took approximately 1-2 minutes for each subject and the values of weight, body fat mass, bone mass, fat percentage, and basal metabolic rate determined by the bioelectrical impedance analyzer were output from the device and recorded on the case report forms.

Statistical Analysis

SPSS version 20 package software was used for statistical analysis. Descriptive statistics were presented as numbers, percentages, mean/median and standard deviation. The conformity of the variables to the normal distribution was examined using visual (histogram and probability graphs) and analytical methods (Kolmogorov-Smirnov, Shapiro-Wilk tests). Numerical variables that did not show normal

distribution were compared using the Kruskal Wallis Test. Correlation between variables was evaluated with Pearson or Spearmen Tests according to normality distribution. Categorical data were compared with the Chi-square test. Confidence interval was set as 95%. P<0.05 was considered statistical significance.

RESULTS

The median age did not differ significantly between the groups (p=0.18). Demographic characteristics are presented in **Table 1**.

Table 1. Demographic characteristics of the obesity groups and the normal weight group

	Group 1 (n=40) (BMI normal)	Group 2 (n=20) (BMI 30-34.9 kg/m²)	Group 3 (n=20) (BMI 35-39.9 kg/m²)	Group 4(n=20) (BMI ≥40 kg/m²)	P value
Gender, n (%)					0.052
Male	13(32.5)	4(20)	1(5)	2(10)	
Female	27(67.5)	16(80)	19(95)	18(90)	
Age, years	38.35±10.4	37.45±11.04	42.60±10.26	46.15±12.23	0.180

The median TSH, median fT3 and median fT4 did not differ significantly between the groups. While the Median insulin level was higher in group 4, the lowest level was found in group 1. The median insulin level did not differ significantly between group 2 and group 3 (**Table 2**, **Figure 1**). The median HOMA-IR, median FBG and median LDL levels differed significantly between the groups (p<0.05) (**Table 2**).

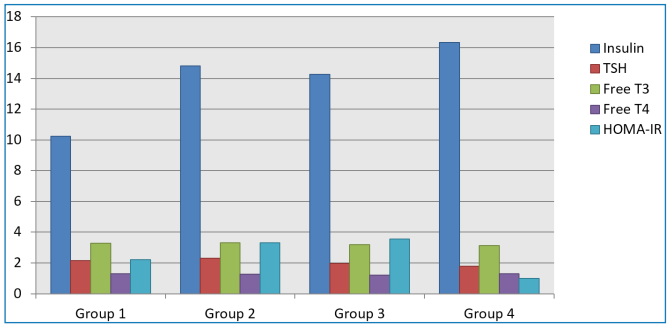

Figure 1. Insulin, HOMA-IR, TSH, free T3 and free T4 values of the groups

The medianfat mass level was significantly different between all groups (p<0.001), and the fat mass linearly increased as increased the degree of obesity. While medianmuscle mass and the median bone mass levels were significantly different in Group 1 and Group 4 compared to other groups, these parameters were higher in group 4 compared to Group 1 (p<0.05). The medianfat percentages was significantly different between all groups (p<0.001), and the fat percentages linearly increased as increased the degree of obesity. The medianbasal metabolic rate was lower in Group 1 compared to Group 2 (p=0.025) and Group 4 (p<0.001), while it was higher in Group 4 compared to Group 3 (p=0.043).

No significant correlation was found between BMI and TSH, fT3 and fT4 levels in the whole population. There was a negative correlation between BMI and TSH in the group with normal body weight (r=-0.430, p=0.006), while no correlation was found between BMI and fT3 (r=0.064, p=0.696) and fT4 (r=0.003, p=0.985). While there was a positive correlation between TSH and BMI in the stage 1 obese group (r=0.553, p=0.011), no significant correlation was found between BMI and T4 (r=-0.193, p=0.415) and T3 (r=0.008, p=0.974). There was no significant correlation between BMI and TSH (r=0.147, p=0.537), T4 (r=-0.346, p=0.136) and T3 (r=-0.267, p=0.256) in the stage 2 obese group. There was no significant correlation between BMI and TSH (r=-0.267, p=0.256), T4 (r=-0.267, p=0.256) and T3 (r=0.060, p=0.801) levels in the morbidly obese group.

While there was a positive correlation between fat percentage and TSH (r=0.391, p=0.014) and T3 (r=0.333, p=0.038) levels in the group with normal body weight, no significant correlation was found with T4 (r=0.037, p=0.821). In the stage 1 obese group, there was a positive correlation between fat percentage and T3 (r=0.521, p=0.018) levels, but no significant correlation was found between fat percentage and TSH (r=0.129, p=0.589) and T4 (r=-0.345, p=0.136) levels. There was no significant correlation between fat percentage and TSH (r=0.326, p=0.161), T4 (r=0.059, p=0.806) and T3 (r=-0.336, p=0.113) levels in the stage 2 obese group. There was no significant correlation between fat percentage and TSH (r=-0.117, p=0.456), T4 (r=0.272, p=0.246) and T3 (r=0.242, p=0.304) levels in the morbid obese group.

There was no significant correlation between HOMA-IR and TSH (r=-0.094, p=0.563), T4 (r=0.189, p=0.242) and T3 (r=0.141, p=0.386) levels in the group with normal body weight. In the stage 1 obese group, there was a positive correlation between HOMA-IR and T3 (r=0.512, p=0.021) levels, but no significant correlation was found between HOMA-IR and TSH (r=0.303, p=0.194) and T4 (r=0.043, p=0.857) levels. There was no significant correlation between HOMA-IR and TSH (r=0.123, p=0.607), T4 (r=-0.114, p=0.631) and T3 (r=0.115, p=0.630) levels in the stage 2 obese group. There was no significant correlation between HOMA-IR and (r=-0.428, p=0.060), T4 (r=0.073, p=0.761) and T3 (r=0.182, p=0.442) levels in the morbid obese group.

There was no significant correlation between LDL and TSH (r=-0.206, p=0.202), T4 (r=0.069 p=0.674) and T3 (r=-0.124, p=0.445) levels in the group with normal body weight. In the stage 1 obese group, there was a positive correlation between LDL and TSH (r=0.469, p=0.037) levels, but no significant correlation was found between LDL and T4 (r=0.005 p=0.983) and T3 (r=0.153, p=0.519) levels. In the stage 2 obese group, there was a positive correlation between

Table 2. TSH, fT3 and fT4, insulin, HOMA-IR, fasting blood glucose and LDL results of the groups

	Group 1 (n=40) (BMI normal)	Group 2 (n=20) (BMI 30-34.9 kg/m²)	Group 3 (n=20) (BMI 35-39.9 kg/m²)	Group 4 (n=20) (BMI ≥40 kg/m²)	P value
TSH, µU/mL	2.14±1.26	2.31±1.08	1.97±1.18	1.79±0.97	0.585
fT3, pg/mL	3.27±0.49	3.32±0.38	3.19±0.46	3.12±0.43	0.274
fT4, ng/dL	1.31±0.15	1.27±0.18	1.22±0.17	1.29±0.20	0.222
Insulin, µU/mL	10.23±4.10	14.80±7.87	14.25±8.20	16.33±6.32	0.002
HOMA-IR	2.22±0.90	3.31±1.64	3.56±2.13	4.06±1.85	<0.001
FBG, mg/dL	88.02±7.78	92.37±11.47	97.55±12.93	100.05±10.64	<0.001
LDL, mg/dL	87.53±29.10	104.90±27.69	107.15±25.67	124±71.57	0.013

LDL and TSH (r=0.621, p=0.003) levels, but no significant correlation was found between LDL and T4 (r=0.113 p=0.634) and T3 (r=0.271, p=0.248) levels. In the morbid obese group, there was a negative correlation between LDL and T4 (r=-0.485, p=0.030) levels, but no significant correlation was found between LDL and TSH (r=-0.047 p=0.845) and T3 (r=-0.167, p=0.480) levels.

In the group with normal body weight, there was a positive correlation between basal metabolic rate and T3 (r=0.414, p=0.009) levels, but no significant correlation was found between basal metabolic rate and TSH (r=-0.050, p=0.764) and T4 (r=0.143, p=0.384) levels. In the stage 1 obese group, there was no significant correlation between basal metabolic rate and TSH (r=0.176, p=0.458), T4 (r=0.173, p=0.466) and T3 (r=0.315, p=0.176) levels. In the stage 2 obese group, there was no significant correlation between basal metabolic rate and TSH (r=0.078, p=0.743), T4 (r=0.111 p=0.643) and T3 (r=-0.032, p=0.895) levels. In the morbid obese group, there was no significant correlation between basal metabolic rate and TSH (r=0.218, p=0.356), T4 (r=-0.201, p=0.396) and T3 (r=-0.032, p=0.895) levels.

In the group with normal body weight, there was a positive correlation between fat mass and TSH (r=0.447, p=0.004) levels, but no significant correlation was found between basal metabolic rate and T4 (r=0.125 p=0.448) and T3 (r=-0.251, p=0.124) levels. In the stage 1 obese group, there was a positive correlation between fat mass and T3 (r=0.543, p=0.013) levels, but no significant correlation was found between basal metabolic rate and TSH (r=0.133, p=0.577) and T4 (r=0.358, p=0.121) levels. In the stage 2 obese group, there was no significant correlation between fat mass and TSH (r=0.323, p=0.165), T4 (r=0.151 p=0.525) and T3 (r=-0.417, p=0.068) levels. In the morbid obese group, there was no significant correlation between fat mass and TSH (r=0.089, p=0.708), T4 (r=0.009, p=0.971) and T3 (r=0.013, p=0.957) levels.

DISCUSSION

When the relationships between thyroid functions and body compositions were evaluated, a positive correlation was found between fT3 and fat percentage among participants with stage 1 obesity(r=0.521, p=0.018), and a positive correlation was also found between fat mass and fT3 (r=0.543, p=0.013). However, this relationship could not be shown in other BMI groups. To our knowledge, there are limited studies evaluating the relationships between thyroid hormones and body compositions in obese patients. In a study conducted with euthyroid men, including obese patients, 1001 individuals were evaluated and a positive correlation was shown between fT3 and fat mass,[14] consistent with our findings. In another study conducted in 2014, it was reported that there was a positive correlation between fT3 and body fat mass in euthyroid overweight and obese participants, regardless of age and gender, but no significant correlation was found for fT4.[15] Two mechanisms are generally held responsible for the relationship between fT3 and fat mass. First, in obese individuals, circulating leptin levelsare increased and peripheral adipocyte amountsare proportionally related to leptin levels.[16] At the same time, leptin accelerates the conversion of T4 to T3. Second, insulin levels are also higher in obese individuals and insulin resistance is higher compared to those with normal BMI. In an animal study, it was reported that deiodinase activity in adipocyte tissues increased with increased insulin.[17] These factors explain the relationship between fT3 and fat mass. As expected, in our study, it was observed that the percentages of fat and fat mass increased as the degree of obesity increased (p<0.001).

In our study, no significant relationship was found between the participants in different obesity classes as evaluated by BMI in terms of thyroid functions (TSH, fT3 and fT4). Similarly, when the obese groups and the control group were compared, no significant differenceswere found between groups in terms of TSH, fT3,or fT4 levels.

Among the BMI groups, there was an inverse relationship between BMI and TSH only in the control group, while a positive correlation was found in patients with stage 1 obesity. No correlation was found between BMI and TSH or T3 in the other body weight groups. The finding of no relationship between BMI and fT4 in our study is confirmed by most other studies in the literature, but contrary to our findings, it has been reported in many studies that BMI and TSH or fT3 levels in obese individuals vary compared to healthy controls.In the study by Knudsen et al.,[11] it was reported that there was no relationship between BMI and fT3, consistent with our study. A study by Manji et al.[18] conducted with 401 euthyroid patients reported that TSH and fT4 levels did not differ between obese and healthy controls, similar to our study. A study by Chomardet al.[19] reported that fT4 levels did not differ between obese and healthy controls. Previous studies by Gusekko et al.[20] and Hak et al.[21] reported that obesity did not affect fT4 values.Another study from 2014 reported similar thyroid hormone levels in overweight and obese participants.[15]

There are also many studies with results opposite to our finding that fT3 levels do not change in obese patients. In a study conducted by Reinehr et al.[22] in 2006, it was reported that TSH and fT3 concentrations increased in obese patients while fT4 levels did not change.In another study in which 6164 participants were evaluated, it was shown that serum TSH levels and BMI were positively correlated.[23] Parallel to these findings, Knudsen et al.[11] evaluated 4649 participants in a cross-sectional study and showed a positive correlation between BMI and serum TSH and a negative correlation between BMI and fT4. There are many studies showing a positive relationship between BMI and TSH.[24-27] The reasons forthis relationship are not fully known; however, some theories have been suggested. Among them, the leading mechanism is proposed to be the increase in deiodinase activity in obese individuals. It has been suggested that the conversion of T4 to T3 in obese individuals createsa defense mechanism against increasing energy needs.[28,29] Another theory is that leptin produced from adipocytes accelerates T3 conversion by increasing deiodinase activity.[30] Finally, it has been suggested that inflammatory cytokines increase in obesity and that increased cytokines inhibit sodium or iodine transporters, resulting in a secondary TSH increase.[31] The small number of participants in our study may have masked the relationship between obesity and fT3.

TSH elevations in obese patients are thought to be a result of obesity, not the cause. In a study conducted with adolescents in which patients were followed for 1 year, it was shown that TSH and fT3 levels decreased to normal ranges in those who reached the target of significant weight loss, but they did not change in those who did not achieve significant

weight loss.[22] Similarly, in the study conducted by Sarı et al.[32] a decrease in TSH values was reported with more than 10% weight loss. Leptin levels correlate directly with the amount of adipose tissue, and leptin has been reported to stimulate TSH biosynthesis in vitro.[33] In addition, synchronization between leptin and TSH secretion has been reported in both adults[34] and children.[35] However, there are also studies showing no relationship between leptin and TSH.[36] On the other hand, although TSH levels are high in obese individuals, TSH receptors are less expressed in adipocytes. Therefore, peripheral thyroid hormone resistance develops and TSH and fT3 levels increase more. This relationship is reversed as adipocytes return to their normal state with weight loss. In our study, leptin levels could not be evaluated due to technical limitations.

As expected in our study, insulin, HOMA-IR as an indicator of insulin resistance, fasting plasma glucose, and LDL levels increased as BMI increased. When the relationships between thyroid functions and LDL concentrations were evaluated, a positive relationship was found between TSH and LDL in the group with stage 1 obesity, and a negative relationship was found between fT4 and LDL in the morbidly obese group. In hypothyroidism, hypercholesterolemia and increased LDL levels are observed due to the decrease in LDL catabolism. In a study conducted with euthyroid patients, it was reported that fT4 levels and LDL levels were negatively correlated, supporting our findings.[37] This relationship was maintained even when adjusted for age and gender. In light of these findings, it can be said that the relationship between thyroid functions and lipid metabolism extends into the euthyroid range. Therefore, low fT4 levels in euthyroid individuals may be associated with cardiovascular risk.

In our study, it was observed that increases in BMI and increases in the basal metabolic rate occurred in parallel. However, when the relationships between thyroid functions and basal metabolic rate were evaluated, no significant relationship was found between basal metabolic rate and TSH, fT4, or fT3 in any of the patient groups. It is known that resting energy expendituresare sensitive to small changes in thyroid functions in patients receiving levothyroxine therapy, and resting energy expenditures are increased in cases of hyperthyroidism and decreased in hypothyroidism. However, this relationship could not be demonstrated in studies conducted with euthyroid individuals. In a study by Tagliaferri et al.[38] similar to our study, it was confirmed that there was no relationship between TSH and resting energy expenditures in obese patients. Resting energy expenditureswere measured by the indirect calorimetric method in these studies. In our study, data were obtained with a formula using lean body mass and age withthe bioelectrical impedance method.

Hypothyroidism and hyperthyroidism are conditions associated with glucose metabolism. It has been reported that insulin resistance is also encountered after T3 administration to healthy volunteers. In a study conducted with euthyroid patients, a negative relationship was shown between HOMA-IR and fT4 while a positive relationship was shown with TSH, and it was also stated that fasting blood glucose and insulin levels were associated with fT4. However, that study did not include obese individuals. In our study, no relationship could be demonstrated between HOMA-IR, insulin,or fasting blood glucose and thyroid functions. This is likely because obesity itself affects glucose metabolism parameters.[39]

Limitations of the study: To our knowledge, the studies conducted to date to evaluate the relationships between thyroid hormones and body compositions in obese patients are very limited. Thyroid functions are affected by factors such as temperature, nutrition, fasting time, and smoking. Although attention was paid to parameters such as hunger and temperature in our study, other possibly related factors were not evaluated. In our study, which was conducted with a total of 120 participants, relationships between obesity and fT3 and TSH levels, frequently shown in the literature, could not be demonstrated. One of the possible reasons for this was the small number of participants. Although the basal metabolic rate is an indirect indicator of resting energy expenditures, considering that calorimetric measurements, which require strict patient preparation, are the gold standard, it may be assumed that evaluations of basal metabolic rate calculations by the bioelectrical impedance method do not fully reflect reality. In calculations of body compositions, methods such as DXA have been used more frequently in the literature and have provided more accurate results. However, it has been reported that bioelectrical impedance results show good correlations with DXA results.[40]

CONCLUSION

In our study, no statistically significant difference was found in terms of thyroid functions (TSH, fT3, and fT4) among participants in different classes of obesity as evaluated by BMI. The small number of participants in our study may have masked the relationship between obesity and fT3. TSH elevations in obese patients are thought to be a result of obesity, not the cause. More extensive studies on this issue are needed.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Kırıkkale University Faculty of Medicine Clinical Researches Ethics Committee (Date: 29.11.2016, Decision No: 23/04).

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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