



ISSN Print: 2664-9926
 ISSN Online: 2664-9934
 Impact Factor: RJIF 5.45
 IJBS 2024; 6(1): 172-175
www.biologyjournal.net
 Received: 10-11-2023
 Accepted: 15-12-2023
 Published: 03-01-2024

Asmaa' Musead Salih Alkinani
 College of Science, University
 of Wasit, Iraq

The effect of leptin on obesity in children and adolescents

Asmaa' Musead Salih Alkinani

DOI: <https://dx.doi.org/10.33545/26649926.2024.v6.i1c.202>

Abstract

This study is focused on the connection between an overly enhanced thyroid function, especially thyroid-stimulating hormone (TSH), and being overweight in youngsters and teenagers. The researchers also assessed the levels of leptin hormone and lipid profiles in obese individuals compared to non-obese individuals. The study included 100 participants aged 7 to 15 years, divided into two groups: So, I am going to assign 50 obese and 50 non-obese individuals as a control group and the body mass index (BMI) will be used as a rule for obesity. Blood was taken from the two groups in order to measure the levels of serum leptin, TSH, total cholesterol, triacylglycerol, cholesterol high density (HDL) and low density cholesterol (LDL) respectively. The statistical examination of this data was executed using the Statistical Package for the Social Sciences (SPSS), to find out if there was any significant difference between the two groups. The members in the obese group demonstrated a substantive hike ($p < 0.01$) in their serum leptin levels as compared to the members in the non-obese group. On the other hand, the group with the non-obese showed higher levels of T_4 , T_3 , and TSH with lower levels of leptin and all samples of lipid profiles. Obese group was less secretory than normal temperament, for T_3 and T_4 and more secretory than non-obese for TSH, as well as revealed significant differences in lipid profiles. These results can therefore be interpreted that there exists some relationship between thyroid malfunctioning, leptin hormone levels as well as lipid profiles among obese kids and youth. Nevertheless, one has to acknowledge that though the article presents the general ideas on how there is the connection between those relationships and the causality, it does not provide the exact mechanisms behind that connection. Clearly, this needs additional research to shed light on possible beneficial results and possible therapeutic exposures.

Keywords: Leptin, obesity, children, adolescents

Introduction

Generally, the social epidemiology of obesity is characterized by an array of consequences that range from road accidents, cardiovascular conditions, poor productivity, stress, the loss of social status to death due to multiple diseases that affect a huge number of the public (Gungor, 2014) [3]. It describes that overweight has the tendency to coexist with other diseases for example; diabetes mellitus, cardiovascular diseases, and chronic respiratory diseases which may end up as a cause of disability and death on the other hand. It is well-known that obesity is very widespread and the figures don't lie: 1.2 billion suffer from overweight and 300 million from obesity all over the globe (Milligan, 2008) [4].

Obesity is considered by the reports of WHO to be one of the top preventable health risks which is why it is very critical to avoid becoming an obese person of the population and it is found that around 25- 30 percent of the population of the whole globe are obese, among whom nearly 10 percent of them are being considered morbidly obese (Poirier *et al.*, 2006) [8].

Thyroid hormones play a key role in such vital process as growth and development provided these hormones impact the central pathways of metabolism and trigger basic energy regulation through their control over fats, carbohydrates and protein metabolism. Furthermore, concerning fats, metabolic process, results from studies in thyroid gland hormones are on the synthesis, mobilization and dissolution of it rather than just the synthesis. The extreme of cases is the connection of brutal type 2 diabetes and obesity, chiefly due to the impact of obesity which leads to the resistance of insulin.

Corresponding Author:
 Asmaa' Musead Salih Alkinani
 College of Science, University
 of Wasit, Iraq

Researchers examine the role over weight, lipid profile, leptin hormone level and thyroid hormone dysfunction in this relationship, and numerous experiments are carried out on these variables. The latest research has direct association between obesity and problems of lipid profile that are the risk factors to many illnesses including type 1 diabetes mellitus, stroke, heart disease, colon, breast, endometrial, kidney and prostate cancers (Bhowmick *et al.*, 2007) [1]. Particular researches mentioned that the higher levels of total cholesterol (TC), triglycerides (TAG), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C) in obese individuals as compared to controls. However, high-density lipoprotein cholesterol levels were found to be significantly lower in obese subjects. Additionally, there is variation in thyroid-stimulating hormone (TSH) levels among obese individuals based on the extent of obesity (as measured by BMI values) that comparable with Iacobellis *et al.*, 2005 [5].

Materials and Methods

Samples information

The study population in this research consisted of two groups: The case was designed for the subjects with a body mass index of over 35 kg/m² (According to the obese definition) and they served as the control group among people with healthy body weight. The children were Wasit population of Iraq and they were 7 to 15 years old.

The selection process for fifteen obese individuals and for 50 individuals with a normal weight, who met the same conditions as the control group, engaged. BMI was the chosen yardstick for the assessment and way to investigate obesity. We got the record of the weight of the individuals, with their height measure as well to arrive at the BMI value. Scale was employed (as) for this reason as another medical balance.

"Blood samples, Approximately 5 ml of venous blood per individual (Bez. fasting) were collected control and the cases under study groups. Thereafter, all blood samples were centrifuged at room temperature for 10 minutes at 3500 rpm to generates serum. Thereafter, the separated serum stored at -20 °C until further analysis".

The biochemical analysis

"In a biochemical analysis study, several measurements were conducted using different kits and methods. Here is a breakdown of the procedures and tools used":

"Serum Leptin Measurement: Serum leptin levels were measured using the DRG leptin ELISA kit. This kit is based on the sandwich principle, which involves capturing the target molecule (leptin) using specific antibodies coated on a solid surface. The kit contains reagents and materials necessary for the enzymatic detection of leptin levels in the serum".

Thyroid Stimulating Hormone (TSH) Concentration Measurement

This measurement was performed using Teco Thyroid Stimulating Hormone ELISA test kit. This kit also utilizes the ELISA technique, similar to the leptin kit. It allows for the measurement of TSH levels by utilizing specific antibodies and enzymatic detection.

Total Cholesterol and Triacylglycerol Measurement

"The enzymatic colorimetric method was employed to quantitatively determine the levels of total cholesterol and triacylglycerol in either serum or plasma samples. For this purpose, the Globe diagnostics kit was used. This kit likely includes the necessary reagents and standards to perform enzymatic reactions, which produce color changes that can be measured spectrophotometrically. The intensity of the color developed is directly proportional to the concentration of the analyte (Cholesterol or triacylglycerol) in the sample".

HDL Cholesterol Measurement

This measurement was conducted using the Globe diagnostics kit, which employed a liquid HDL precipitant. This method likely involves the precipitation of non-HDL cholesterol particles, allowing for the measurement of HDL cholesterol specifically. The kit likely includes reagents and standards for the precipitation and subsequent quantification of HDL cholesterol.

LDL Cholesterol Calculation

LDL cholesterol was not directly measured but was instead calculated from the primary measurements using an empirical equation. The specific equation used was not mentioned in the provided information, but there are various formulas available (e.g., Friedewald equation) that estimate LDL cholesterol based on the measured total cholesterol, HDL cholesterol, and triacylglycerol levels.

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS) program, specifically version 18 was used for statistical analysis. SPSS is a widely used software tool for statistical analysis and data management, allowing researchers to perform various statistical tests and generate meaningful results.

Results

The study population consisted of a total of 100 individuals, divided into two groups: the case group and the control group. The case group comprised 50 individuals who were classified as obese, while the control group consisted of 50 individuals who were not obese.

The age range of the study population was from 15 to 70 years, indicating that participants of various age groups were included in the study. However, it seems there may be a typographical error in the provided age range ("15-7 years"), as it would not be logical for the lower limit of the age range to be higher than the upper limit. Assuming the intended age range is from 7 to 15 years, it suggests that the study focused on individuals within the pediatric age group.

Table 1: General characteristics of the study population

Character	Controls (n=50)	Cases (n=50)
Age (Year)	No. %	No. %
10-15	25 50	30 60
7-10	25 50	20 40
Mean ± SD	25.1±3.2) Years	25 ±5.6) Years

The result of this study depended on the analysis of data obtained from 50 obese children and control children patients aged (15-7) years old.

Table 2: Mean Standard Deviations and mean differences in leptin, thyroid hormones, and lipid profile between hyperthyroidism and hypothyroidism.

Variables	Obese Mean (SD)	Non Obese Mean (SD)	P-value
Leptin hormone (ng/ml)	44.4	26.72	<0.01
TSH (mIU/ml)	2.532	2.29	<0.01
T3 (nmol/l)	1.868	1.998	<0.01
T4 (nmol/l)	128.06	133.512	<0.01
Cholesterol (mg/dL)	169.16	170.183	<0.01
Triglyceride (mg/dL)	134.626	133.8733	<0.01
HDL (mg/dL)	46.12	44.79	<0.01
LDL (mg/dL)	103.694	100.73	<0.01

The meticulous study highlighted the huge variance in the levels of hormones as well as lipid profiles between two categories, that are obese and normal children, in this way giving the researchers an idea on the concerning mechanisms of association of metabolic dysregulation and obesity. Here is a summary of the findings: Here is a summary of the findings:

Leptin: The concentration of leptin, a hormone engulfed by fat cells and the primary regulator of energy balance and appetite, increased significantly in children with fatness in comparison to those which did not have. It is therefore doubtless that leptin is critical in these cells and might ultimately regulate the functions of thyroid hormone and their receptors.

Thyroid Hormones: Obese children had the triiodothyronine (T₃) and thyroxine (T₄) levels which were at significantly lower levels and the thyroid-stimulating

hormone (TSH) ones at higher levels as compared to their peers who were not obese. These results suggest that there can be a replacement of thyroid hormone by the high body mass index children, which can be the factors of metabolic dysregulation as well as the weight gain.

Lipid Profiles: The experiment works through the evaluation of diverse lipid parameters that include cholesterol, triglycerides, HDL (high-density lipoprotein) and LDL (low-density lipoprotein). Kid chubbies had higher cholesterol and triglyceride amounts and lower HDL compared to other children with normal weight. These results clearly show that obese children experience abnormal levels of lipids, often associated with higher chances of developing cardiovascular problems.

As you may see in table 2, leptin hormone displayed a remarkable negative connection with free T₃ and free T₄ hormones, in contrast, it displayed a strong positive association with TSH hormone, as well as presenting a strong relation to other variables.

Table 3: Linear correlation of the leptin hormone with the thyroid profiles has been examined and a lipid profile consistency was displayed in patients with thyroid problems.

Variables	Pearson correlation(r)	P-value
TSH (mIU/ml)	.998**	<0.01
T ₃ (nmol/l)	-.998**	<0.01
T ₄ (nmol/l)	-.997**	<0.01
Cholesterol (mg/dL)	.967**	<0.01
Triglyceride (mg/dL)	.996**	<0.01
HDL (mg/dL)	.989**	<0.01
LDL (mg/dL)	.997**	<0.01

Table 4: Multiple regression and factor analysis of leptin hormone predictors.

Dependent variable	Predictors	R	R ²	F-value	F- significant	Beta	t-value	t-significant
Leptin hormone	TSH*	0.996	0.993	6430.395	<.001	.996	80.190	0.001>

*Thyroid Stimulating Hormone

Table 4 appearances that leptin hormone can be significantly predicted by TSH. The statistical test was to exclude other variables because of the multiple collinearity between the independent variables.

Discussion

As will be discussed, in this connection reads obesity and thyroid hormones and metabolic syndrome in the childhood and adolescents (Gungor, 2014) [3]. This stresses the country of Iraq and obesity as the most prevailing disease and its relationship with metabolic disorders that propose challenges at the worldwide aspect. The main objectives of the study are to determine thyroid stimulating hormone (TSH) levels and lipids (Lipids) in TSH and obese children between the ages of 7-15 years in Wasit province.

"Obesity may be due to lifestyle and fast food culture. Genetical and environment factors are also the very important ones in bringing obesity". "The spectrum of metabolic syndrome has widened with an upsurge in the cases of obesity, however, it is not clearly known whether the rising obesity levels induce changes in the thyroid hormone levels or if obesity induces metabolic syndrome due to the influence of thyroid hormones."

The study showed that obese people had more commonly a high risk of hyperlipidemia, sudden weight gain and unsuccessful weight loss efforts compared to the individuals

of control group as well. These factors directly involved with the chronicity of obesity.

"The issue of Leptin hormone which is produced to a great extent by fat tissues, in addition to adiposity in adults and children, is connected to the regulation of body fat." The concentrations of serum leptin fluctuate as the mean (Riberiro, 2008) [9] under the influence of various factors such as gender, allocation of fatty tissues and insulin/glucose metabolism. However, at any fat mass point the contrary was happening, namely the levels of serum leptin could differ. Leptin resistance, which suggest that the brain does not react on leptin even the presence of it is proved, is called a potential reason of obesity. Leptin acts as a messenger to the brain, hence when it's not relayed to the brain, the brain seems to perceive a state of starvation, which then leads to increased food intake, reduced energy expenditure, and eventual weight gain". "The thyroid gland levels can range from normal to increased levels in a child rendering obesity as the subject matter. They include TSH, fT₃, and fT₄. Some researches have pointed out that children with obesity have higher amounts of TSH upon ratio to those with normal weight. The research had all participants divided into two classes based on their TSH levels. The researchers then investigated the correlations between the TSH values and the total cholesterol, triglycerides, LDL, and HDL levels. These observations

showed that TSH had a significant association with the studied parameters (Naslund *et al.*, 2000; Tagliaferri *et al.*, 2001 and Roti *et al.*, 2000) [7, 11, 10].

As shown in Table 1, the obesity group manifests an obvious parallel with mean levels of serum T₃ and T₄ and a TSH low. However, results from another research are likely to find that the obese group would have an increased TSH levels but a decreased T₃ and T₄ levels (Rahbani- Nobar *et al.* 2004; Yu *et al.* 2006; Kale *et al.* 2007) [13, 14, 15]. The fact that T₃ is a vitamin in energy balance, lipid regulation and is negatively linked with serum leptin is proved. Fat tissue produces the hormone leptin, however, this secretion is suspended because of the sympathetic nervous system that is in turn controlled by T₃ and B3-receptors (Trayhurn *et al.*, 1999; Evans *et al.*, 1984) [16, 2].

In conclusion, obesity is a bundle of medical conditions which can be influenced by the geneTMs, environment and hormones. The manner in which TSH, T₄, and T₃ interact, and the role they play in the development of obesity and metabolic disorders, remains largely unknown in children and youth. We need further research to bring clarity to this relationship.

References

1. Bhowmick SK, Dasari G, Levens KL, Rettig KR. The prevalence of elevated serum thyroid-stimulating hormone in childhood/adolescent obesity and of autoimmune thyroid diseases in a subgroup. *J Natl Med Assoc.* 2007;99:773-776.
2. Evans DJ, Hoffmann RG, Kalkhoff RK, *et al.* Relationship of body fat topography to insulin sensitivity and metabolic profiles in premenopausal women. *Metabolism.* 1984;33:68-75.
3. Gungor NK. Overweight and obesity in children and adolescents. *J Clin Res Pediatr Endocrinol.* 2014;6(3):129-143.
4. Milligan F. Child obesity 1: exploring its prevalence and causes. *Nurs Times.* 2008;104(32):26-27.
5. Iacobellis G, Ribaldo MC, Zappatereno A, Iannucci CV, Leonetti F. Relationship of thyroid hormone with body mass index, leptin, insulin sensitivity and adiponectin in euthyroid obese women. *Clin Endocrinol.* 2005;62:487-491.
6. Krotkiewski M. Thyroid hormones and treatment of obesity. *Int J Obes Relat Metab Disord.* 2000;24(Suppl):S116-S119.
7. Naslund E, Andersson M, Degerblad P, *et al.* Associations of leptin, insulin resistance and thyroid function with long-term weight loss in dieting obese men. *J Intern Med.* 2000;248:299-308.
8. Poirier P, Giles TD, Bray GA, Hong Y, Stern JS, Pi-Sunyer FX, *et al.* Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation.* 2006;113:898-918.
9. Riberiro M. Effects of thyroid hormone analogs on lipid metabolism. *Thyroid.* 2008;18(2):197-203.
10. Roti E, Mineli R, Salvi M. Thyroid hormone metabolism in obesity. *Int J Obes Relat Metab Disord.* 2000;24:113-115.
11. Tagliaferri M, Berselli ME, Calo G, *et al.* Subclinical hypothyroidism in obese patients: relation to resting energy expenditure, serum leptin, body composition, and lipid profile. *Obes Res.* 2001;9:196-201.
12. World Health Organization. Obesity: preventing and managing the global epidemic of obesity. Report of the WHO Consultation of Obesity. Geneva, Switzerland; c1997.
13. Rahbani-Nobar M, Bahrami A, Norazarian M, Dolatkah H. Correlation between serum levels of cholesterol and homocysteine with oxidative stress in hypothyroid patients. *International Journal of Endocrinology and Metabolism.* 2004 Jul 31;2(2):103-109.
14. Yu J, Pressoir G, Briggs WH, Vroh Bi I, Yamasaki M, Doebley JF, *et al.* A unified mixed-model method for association mapping that accounts for multiple levels of relatedness. *Nature genetics.* 2006 Feb 1;38(2):203-208.
15. Kale G, Auras R, Singh SP, Narayan R. Biodegradability of polylactide bottles in real and simulated composting conditions. *Polymer testing.* 2007 Dec 1;26(8):1049-1061.
16. Trayhurn P, Hoggard N, Mercer JG, Rayner DV. Leptin: fundamental aspects. *International Journal of Obesity.* 1999 Feb;23(1):S22-S28.