World of Science: Journal on Modern Research Methodologies

Volume 3 Issue 1, Year 2024
ISSN: 2835-3072
https://univerpubl.com/index.php/woscience

The Relationships between Obesity and Increased Serum TSH Level in Euthroid Subjects

Jawad K. Abdulla, Hashim M. Al-Kadhimi
Department of Medicine, Al-Kadhimiya Teaching Hospital, College of Medicine, Al-Nahrain University, Iraq

Article Information

Received: Nov 13, 2023
Accepted: Dec 22, 2023
Published: Jan 06, 2024

Keywords
Obesity, Serum TSH Level, Euthroid Subjects, body mass index BMI, T3, T4.

ABSTRACT

Objectives: *To investigate the relationship between circulating TSH level and adiposity in cohort of obese people who have normal, thyroid function.*

Methods: *Cross sectional analysis was carried out on 110 thyroid obese or overweight female subjects. Twenty male lean and thyroid subjects (BMI < 25Kg/m2), were included in the study group. Thyroid stimulation hormone (TSH) total thyroxin T4 total triiodothyronine T3, body weight, height, body mass index (BMI) was assessed.*

Results: *TSH level were higher the obese than in the lean subjects, in the study group (lean and obese subjects) there was increase serum TSH with increasing degree of BMI. There was no correlation between serum TSH and age. There was no correlation between serum T4 and body mass index (BMI).*

Conclusion: *This study strongly supports existing, but contradictory evidence that seruum TSH levels are positively correlated with the degree of obesity in overweight people with normal thyroid function.*

Introduction

The Hypothalamic-Pituitary-Thyroid (HPT) axis regulates energy expenditure, oxygen consumption, and fuel metabolism. HPT axis disorders impact metabolic rate, thermogenesis, and body weight. Conversely, changes in body weight are accompanied by compensatory changes in energy expenditure (1). Likewise, considerable differences may be seen in thyroid function between populations when estimated by median serum TSH levels. The optimal level for thyroid hormones and TSH in serum to attain physical changes has not been established, but the trend these years is to narrow the range of serum TSH, regarded as optimal.

Detrimental effects on the cardiovascular system Variation in thyroid function are seen individuals have been reported for particularly elevated serum levels of TSH and follow-up studies have shown an increase in risk of development of thyroid dysfunction (2). In a study of elderly subjects, a possible association between mild hypothyroidism and BMI was found among women however with the opposite tendency among men (3). Overweight is a major threat to public health, Lifestyle is undoubtedly of major importance for weight gain in the population, but the interaction with other factors is far from elucidated in detail (4). TSH synthesis and secretion 1s primarily controlled by the stimulatory action of TRH and the negative feedback restraint by thyroid hormones (T4 and T3), whereas other factors, including leptin, dopamine, somatostatin, and serotonin act to modulate release (5). Furthermore, clinical studies have shown that leptin replacement significantly blunts the fasting- induced fall in TSH secretion in healthy lean men and in nonnal- weight. TSH synthesis and secretion are prin1arily controlled by the

stimulatory action in women of reproductive age in the early follicular phase of their menstrual cycle (6). Finally, indirect evidence for a stimulatory impact of leptin on TSH secretion has also been found in various human disease states characterized by low circulating leptin levels, Evidence emerging that thyrotropin induces adipogenesis and adipokine production directly independent of the mediating influence of thyroid hormone on energy balance. Promoting us to reinvestigate the relationship between circulating TSH levels and body weight in a cohort of obese people with normal thyroid function, our results provide circumstantial evidence that the pituitary gland via thyrotropin may indeed contribute to the evolution of obesity independent of any involvement of the thyroid gland (7).

Material and Methods

Cross sectional analysis was carried out on 155 obese or overweight subjects obtained from the outpatient of Al-Kadhimiya teaching hospital in Baghdad between September 2007 and February 2008. of these, 45 patients were excluded for any of the following reasons: History of thyroid disease, Abnormal thyroid hormone level, History of radioiodine treatment, being on treatment with thyroid hormone antithyroid, drugs or any drugs that might affect evaluation of thyroid status, Age below 18 and above 60 years. 20 female subjects were considered eligible for this study The mean age was 39 (4) years and the mean BMI 22 Kg/m2 Among the lean subjects 2 carried the diagnosis of diabetes mellitus and they were on treatment with sulphonylurea , Three subjects carried the diagnosis of hypertension and they were on treatment with ACEI and B-blocker. 2 subjects were smoker with duration of 10 year and cigarette index of 13 years.

Study Design

In this cross sectional study we measured Serum level of TSH, thyroxin T4, triiodothyronin T3 as indicators of thyroid function.

Anthropometrical Measurement

Body weight (Kg) and height (m) were measured while subjects wore light clothing and no shoes. BMI was calculated as Wight divided by squared height (Kg/m2). Age limit was between (18-60 year).

Laboratory Methods

1. TSH (µU/ml) reference range (0.25-5.0), VIDAS automated quantitative test. (immune Enzymatic detection of TSH using the ELFA technique).
2. T4 nmol/L reference range (60-120). T3 nmol/L reference range (0.95-2.5) were determined by VIDAS T4 VIDAS T3 which are automated quantitative tests for use on the VIDAS instruments for the determination of total T4, T3 in human serum and plasma using the ELFA technique (Enzyme Linked Fluorescent Assay).

Statistical Analysis

All statistical analysis was performed using SPSS 9.0 software (SPSS Inc.) data are presented as means± SD correlation between measured parameters was assessed using the analytical method of correlation and was adjusted for age by linear regression analysis.

Results

Clinical characteristics of lean and obese subjects were shown in (table 1) revealed obese pateints significant increase in age and weight than in lean patients in addition the serum TSH level were higher in the obese than in the lean subjects, there was no significant difference was found in serum T4 level between the two groups, also there is no change in serum T4 with increasing BMI (linear correlation) as in (table 2) and (fig.4). When the study groups was stratified according to the degree of adiposity (lean with BMI <25Kg/m2, pre obese with BMI 25

- 30Kg /m2 , class I obesity with BNII 30-35 Kg/m2, class II obesity with BMI 35-40 Kg/m2 and class III morbid obesity with BMI > 40 Kg/m2) (fig. 2), a gradation in serum TSH level became evident when compared to the lean group although it is significant only in class II and class III obesity ,as in (Fig.3) while TSH level does not change with increasing age (linear correlation) as in (fig.1).

Table 1: Anthropometric and clinical characteristics of lean and obese subjects

Parameters	Lean	Obese
Numbers	20	110
Age (years)	39 ± 4 a	42 ± 6 b
Height (m)	1.6 ± 0.3 a	1.6 ± 0.3 a
Weight (Kg)	54 ± 7 a	88 ± 17 b

**Data were given as mean ± SD*

Table 2: Effect of Obesity on BMl (Kg/m2), TSH Level (µIU/ml), T3 (nmol/L) and T4 (nmol/L).

Parameters	**Lean**	**Obese**
BMl (Kg/m2)	22 ± 2 *a*	36 ± 4 *b*
TSH Level (µIU/ml)	1.5 ± 0.4 *a*	2.15±0.5 *b*
T3 (nmol/L)	1.5 ± 0.2 *a*	1.5 ± 0.4 *a*
T4 (nmol/L)	80 ± 15 *a*	80 ± 20 *a*

**Data were given as mean ± SD*

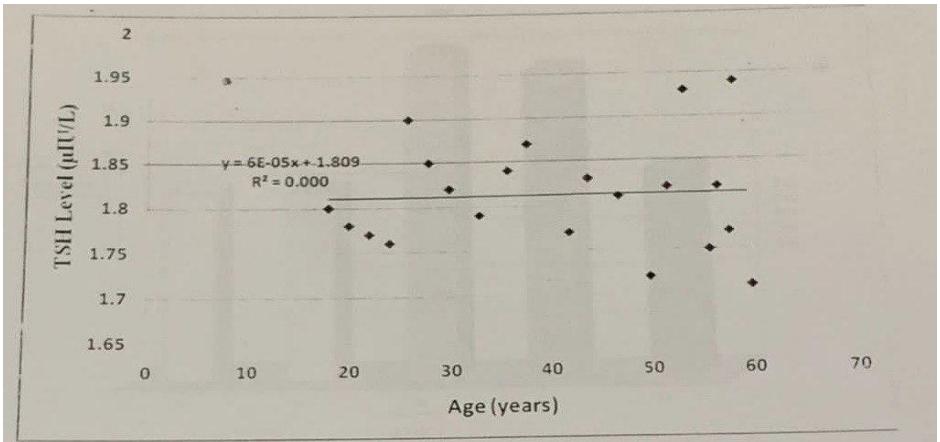

Figure 1: The Relation between TSH Level and Age in the Study Group.

Figure 2: The Number of Subjects according to their BMI in the Study Group.

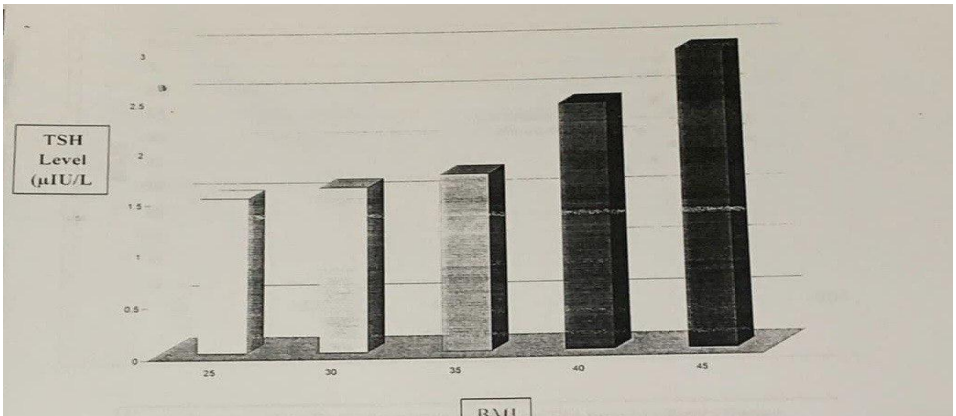

Figure 3: The Relation between TSH Level and BMI in the Study Group.

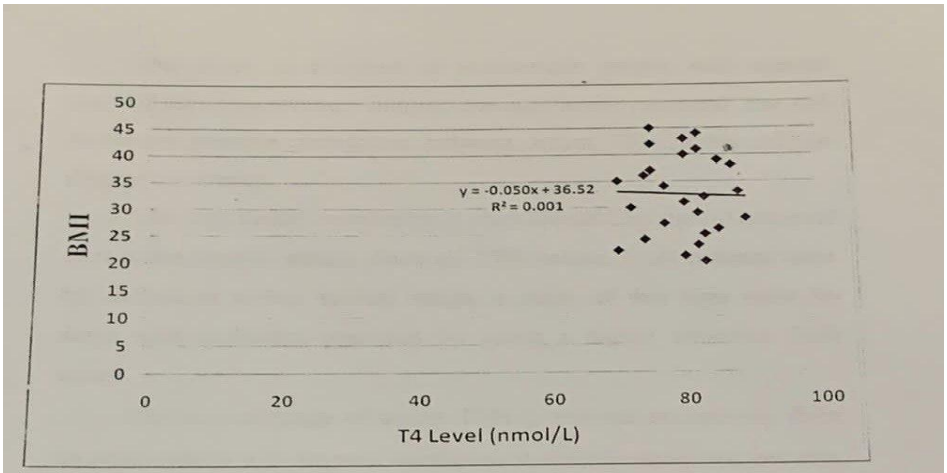

Figure 4: The Relation between BMI and T4 Level in Study Group.

Discussion

Our study in a cohort of overweight people with normal thyroid function strongly support the previously by other studies but still contested positive correlation between serum TSH levels and the degree of obesity, the significant correlation is corroborated by the inclusion of non-obese healthy people since all TSH values in all patients were by definition within normal range, a study of this type must be done with sufficient precision by using a highly sensitive TSH assay.

The normal range of serum TSH is derived exclusively from its relationship with thyroid hormones in strictly euthyroid persons one might assumed that the interaction between TSH and adipose tissue occured on a scale different from that involved in thyroid regulation. Recent studies in man and other mammalian species have been yielded convincing evidence that adipocytes and pre adipocytes possess thyrotropin receptors, the signal generated by thyrotropin in adipocytes is mediated by activation of cAMP deepened on protein kinase (8). Studies in vitro and in vivo demonstrate that the action of thyrotropin via its receptors in fat tissue induced differentiation of pre-adipocytes into adipocytes (adipogenesis). adipose tissues are major endocrine gland protjucing and releasing numerous adipokines which have metabolic or inflammatory effects in other tissues including the liver muscle pancreatic islets B-cells and the brain (9, 10). Some scientists believed that thyrotropin directly induced the synthesis and released of adipokines (11, 12). The positive relationship between serum TSH and adiposity may be attributed to a negative feedback system (13), and hence the positive relation between serum TSH and adiposity could also be interpreted in the reverse lead to increase production and releasing of many of adipokines (14), the adipokines extensive information is available on the inhibition of appetite by leptin dominantly by acting on the hypothalamus, positive correlation has been reported between serum levels of leptin and TSH (12). As expected in a cohort selected for euthyroid state, mean serum T4 level were well within the normal range and very similar among the lean and obese subjects serum. T3 level available only for the obese subject were also well within the normal range, but only serum TSH has a positive correlation with adiposity.

Conclusion

This study strongly supports existing but contradictory evidence that serum TSH level are positively correlated with the degree of obesity in overweigh people with normal thyroid function.

Authors Contribution

All authors contributed equally.

Conflict of interest

The authors have declared no conflict of interest.

References

1. Al Adsani H, Hoffer LJ, Silva JE (1997): Resting energy expenditure is sensitive to small dose changes in patients on chronic thyroid hormone replacement. J Clin Endocrinol Metab., 82:1118-1125.
2. Stokholrn KH, Lindgreen P (1982): Serum free triodothyronine in obesity. Int J Obes., 6:573-578
3. Acheson K, Jequier E, Burger A, Danforth E (1984): Thyroid hormones and thermogenesis: the metabolic costof food and exercise. Metabolism, 33:262-265.
4. Duntas L, Hauner H, Rosenthal J, Pfeiffer EF (1991): Thyrotropin releasing hormone (TRH) immunoreactivity and thyroid function in obesity. Int J Obes., 15:83-87.
5. Morley JE (1981): Neuroendocrine control of thyrotropin secretion. Endocr Rev., 2:396-436.
6. Chan JL, Heist K, DePaoli AM, Veldhuis JD, Mantzoros CS (2003): The role of falling leptin levels in the neuroendocrine and metabolic adaptation to short- term starvation in healthy men. J Clin Invest., 111:1409-1421
7. Naslund E, Andersson M, Degerblad P, et al. (2000); Associations of leptin, insulin resistance thyroid function with long-term weight loss in dieting obese men. J Intern Med., 248:299-308.

8. Roti E, Mineli R, Salvi M. (2000); _Thyroid hormone metabolism in obesity. Int J Obes Relat Metab Disord., 24(2): S113-S115.
9. Cristina M. Rondinone. (2006); Adipocyte derived hormones, cytokines, and mediators. Endocrine, 29: 81-90.
10. Antunes TT, Gagnon A, Chen U, Pacini F, Smith TJ, Sorisky A. (2006); lnterleukin-6 release from human abdominal adipose cells is regulated by thyroid-stimulating hormone: effect of adipocyte differentiation and anatomicdepot. Am J Physiol Endocrinol Metab., 290: E1140-4.
11. Menendez C, Baldelli R, Carnina JP, et al. (2003); TSH stimulates leptin secretion by a direct effect on adipocytes. J Endocrinol., 176: 7-12.
12. Zirnrnerrnann-Belsing T, Brabant G, Holst JJ, Feldt-Rasmussen U. (2003); Circulating leptin and thyroid dysfunction. Eur J Endocrinol., 149: 257-71.
13. Schaffler A, Binart N, Scholmerich J, Buchler C. (2005); Hypothesis paper Brain talks with fat - evidence for a hypothalamic pituitary-adipose axis? Neuropeptides., 39:363-
14. Matthews DR, Hosker JP, Rudenski AS, Naylor BA,Treacher DF, Turner RC. (1985); Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia, 28:412-9.