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Thyroid Metabolic Hormones and its Correlation with BMI and Lipid Profile in Healthy People

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Abstract
Background:Thyroid hormone play an important role in basal lipid metabolism.It is well known that alteration in thyroid function can result in changes in the composition and in the transport of lipoprotein.This study aimed to study the effects of thyroid hormone on serum lipids and evaluted the correlation between thyroid hormone with Body Mass Index(BMI) and lipid profile of healthy people.
Methods: Total of 56 healthy people(28 male:28 female) ,aged 30-60 years included in this study.About 5ml of fasting blood (8-12 h.) was collected from each individual.To determine serum Thyroid stimulating hormone (TSH), Triiodothyronine (T3) and Thyroxine (T4) and the quantitative sandwich enzyme immunoassay technique were used.Lipid profile were measured by an enzymatic colorimetric(GPO-POD) method.
Results: The result of the present study showed that the levels of triglyceride was significantly higher ($p< 0.05$) in males than females ,while the levels of triiodothyronine was significantly higher($p< 0.05$) in females than males of healthy people.Correlation analysis in healthy males showed positive correlation among BMI with lipid profile,while an inverse correlation between TSH and T4,also an inverse correlation between T3 and HDL .In healthy females there was positive correlation among T3 with BMI and WC,also between T4 and LDL ,while an inverse correlation between TSH and VLDL.
Conclusions: Thyroid metabolic hormone especially T3 regulate the resting metabolic rate and the mild thyroid dysfunction was linked to significantly changes in body weight ,lipid profile and likely represents risk factor for healthy and obesity.
Keywords:Thyroid metabolic hormone,lipid profile,BMI ,healthy people.

Introduction

The thyroid is one of the largest endocrine glands of the body. Thyroxine (T4) and triiodothyronine (T3), together referred to as thyroid metabolic hormones, play an important role in basal metabolism and the functioning of almost all tissues and systems in the body . In addition to T4 and T3, thyroid stimulating hormone (TSH) secretion typically is maintained within relatively narrow limits via a sensitive negative feedback loop in which TSH stimulates the synthesis and release of thyroid hormones, that in turn negatively feed back to the hypothalamus and anterior pituitary to limit further TSH release (Walter *et al*.,2012).

In humans 100% of circulating T4 is secreted by the thyroid gland, but only 20% of T3 is derived from this source. The remaining 80% of T3 is generated by peripheral conversion of T4 to T3.The kinetics of T3 metabolism differs from those of T4 because of the 10- to 15-fold lower affinity of T3 for thyroid binding globulin.Thus, for circulating T3 approximately 0.3% is in the free active form and for T4 only 0.02%. The overall production rate of T3 per day is approximately half that of T4(50 vs. 110 nmol), and circulating levels of free T3 (FT3) are approximately 3- to 4-fold less than that for free T4 (FT4) (5 vs. 20 pmol/liter). The half-life of circulating T4 is estimated at 6.7 d and that for T3 of 0.75 d ,but the T3 represent the active form of thyroid hormone(Russell *et al*.,2008).

It is well known that alterations in the thyroid function can result in changes in the composition and in the transport of lipoproteins . Specifically, the thyroid hormone stimulates the hepatic de novo cholesterol synthesis by inducing the HMG-CoA reductase that catalyzes the conversion of HMG-CoA to Mevalonate, which is the first step in the biosynthesis of cholesterol . Additionally, thyroid hormones activate the LDL receptors. The promoter of the LDL receptor gene contains a thyroid hormone responsive element (TRE) which allows T3 to upregulate the gene expression of the LDL receptor. Moreover, thyroid hormones stimulate the cholesteryl ester transfer protein (CETP), an enzyme which transports cholesteryl esters from HDL2 to the very low density lipoproteins (VLDL) and triglycerides in the opposite direction. Finally, thyroid hormones stimulate the lipoprotein lipase (LPL) which catabolizes the triglyceride-rich lipoproteins and the hepatic lipase (HL), which hydrolyzes HDL2 to HDL3 (Mondal *et al*.,2011).

Cholesterol is a fat-like substance made by the body. It is used in the production of bile acids, steroid hormones, Vitamin E, and cell membranes. Dietary cholesterol is found in animal foods such as organ meats, egg yolks, other meats and poultry. Cholesterol levels can have a major impact on risk for heart disease. It is

recommended that you maintain a low LDL-C (bad cholesterol) and a high HDLC (good cholesterol)(Al-Ajlan,2011).

Elevated levels of triglyceride, cholesterol and LDL-C are documented as risk factors for atherogenesis.LDL-C in its oxidized or acetylated form has been identified as amajor atherogenic particle;as it not only load macrophages with cholesterol for the formation of foam cells but also because it ischem otactic for circulating monocytes,is cytotoxic and can adversely alter coagulation pathways.The blood level of HDL-C in contrast bears an inverse relationship of the risk of atherosclerosis and coronary heart disease that is higher the level (Abubakar *et al*.,2009).

Body mass index (BMI) is a commonly used indicator of obesity and has been associated with an unfavourable lipid profile consisting of elevated triglycerides, total cholesterol, and low density lipoprotein (LDL) cholesterol and low high density lipoprotein (HDL) cholesterol in men and women. BMI is often used in clinical settings to estimate body fat and to assess risk among adults . Waist circumference(WC), a simple measure of abdominal fat, has been observed to be a stronger predictor of obesity-related risk factors than BMI in older adults (Brenner *et al*.,2010).There may be gender differences associated with central body size (men may tend to have greater central body size than women)(Cugnetto *et al*.,2008).

The present study yielded to study the effects of TSH,T3 and T4 on serum lipids and also evaluated the relationship between TSH,T3 and T4 and the lipid status. Investigation of the relationship among BMI with thyroid hormone and lipid profile in healthy people.

Subjects and methods

Subjects

The study was carried out in the province of Babylon,by taking 56 healthy people(28 male:28 female),aged 30-60 years. Exclusion criteria were participants having a personal history of thyroid disease and have been taking thyroxine or antithyroid drugs for treatment; taking medication affecting thyroid function such as glucocorticoid, antiepileptic and contraceptive drugs:lipid lowering agent;pregnant women; participants with overt hypothyroidism or overt hyperthyroidism.

Sample collection and storage:

Blood samples were collected with a record of age and sex, from all of the subjects who came for the determination of hormones and the lipid profile. About 5 ml of fasting blood(8-12 h.) was collected from each individual. After the centrifugation of the collected blood, the serum samples were collected in plain tubes and stored at -20º C. For each sample, the TSH, T3,T4, total cholesterol (TC), TG, HDL,LDL and VLDL levels were measured. To determine the serum TSH, T3 and T4 the quantitative sandwich enzyme immunoassay technique were used.Total serum cholesterol and HDL cholesterol were measured by an enzymatic colorimetric cholesterol esterase method, and HDL cholesterol was measured after precipitation with phosphotungstate and magnesium ions. Triglycerides were also measured with an enzymatic colorimetric (GPO-POD) method. LDL cholesterol was calculated using the Friedewald formula: LDL cholesterol=total serum cholesterol – HDL cholesterol – one-fifth of the triglyceride concentration.

Anthropometrical measurements

Body mass index (BMI) was calculated using the formula BMI= weight (kg)/ height^2 (m)^2 and classifying under weight (BMI<18), normal (BMI 18 - 24.9), overweight (BMI 25 - 29.9) , obesity (BMI 30-39.9) and morbid obesity (BMI > 40) (WHO,2004). The waist circumference was measured while the subject standing up , at the narrowest point of the torso width-wise, usually just above the belly button ,which is ≤ 102 cm in male and ≤ 88 cm in female (WHO,2004).

Statistical Analysis:

Analysis were performed using the Statistical Package for Social Sciences (SPSS version 18.0). Quantitative data was presented as Mean ± SD. For comparing means of different variables between males and females, independent sample t-test was used. Bivaraite correlations were performed using the Pearson correlation coefficient .P value ($P<0.05$) was considered statistically significant .

Results

The results of the present study showed that there were significant differences in triglyceride level (TG) and triiodothyronine(T3) between male and female subjects.Levels of TG was significantly higher($p<0.05$) in males than females while the levels of T3 was significantly higher ($p<0.05$) in females than males of healthy people.

Table(1):comparision of various parameters according to gender

Index	Male(N=28) Mean± SD	Female(N=28) Mean± SD	P value
BMI(kg/m^2)	28.28 ± 5.19	30.95 ± 6.49	0.18
WC(cm)	101.11 ± 9.82	96.22 ± 12. 7	0.20
Cholesterol(mmol/l)	4.26 ± 1.06	4. 28 ± 0.85	0.94
TG(mmol/l)	1.82 ± 0.93	1.29 ± 0.37	0.03 *
HDL(mmol/l)	1.08 ± 0.26	1.16 ± 0.16	0.24
LDL(mmol/l)	2.55 ± 1.02	2.59 ± 0.81	0.90
VLDL(mmol/l)	0.57 ± 0.40	0.45 ± 0.20	0.31
TSH(mIU /ml)	2.79 ± 1.72	2.85 ± 1.90	0.92
T3(ng/ml)	0.50 ± 0.07	0.59 ± 0.08	0.002 *
T4(mg/dl)	5.14 ± 0.34	5.51 ± 1.49	0.31

BMI: Body Mass Index, WC: Waist Circumference, TG: Triglyceride, HDL: High Density Lipoprotein, LDL-C: Low Density Lipoprotein,VLDL: Very Low Density Lipoprotein ,TSH: Thyroid-Stimulating Hormone,T3: Triiodothyronine,T4: Thyroxine,p<0.05*.

Correlation analysis in healthy males showed positive corellation among BMI with WC, cholesterol and LDL (r=0.81,p=0.001),(r=0.60,p=0.008) and (r=0.66,p=0.003) respectively, also in healthy females the results indicated positive correlation between BMI and WC (r=0.59 ,p=0.01) as shown in table(2).

Table(2): Correlation of BMI with WC and lipid profile of both gender

Groups \ Index		WC (cm)	Cholesterol (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL (mmol/l)	VLDL (mmol/l)
BMI(kg/m^2) Male(N=28)	r	0.81	0.60	0.11	-0.15	0.66	0.06
	p	0.001*	0.008*	0.65	0.55	0.003*	0.79
BMI(kg/m^2) Female(N=28)	r	0.59	0.37	0.21	-0.03	0.32	0.43
	p	0.01*	0.12	0.39	0.90	0.19	0.07

Correlation coefficient (r) , * Correlation is significant ≤ 0.05 level (2-tailed)

The study revealed that in healthy males there was positive correlation among WC with cholesterol and LDL (r=0.46,p=0.05) and (r=0.48,p=0.04) respectively, and there was also positive correlation between WC and VLDL in healthy females (r=0.45,p=0.05).

Table(3): Correlation of WC and lipid profile of both gender

Groups \ Index		Cholesterol (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL (mmol/l)	VLDL (mmol/l)
WC(cm) Male(N=28)	r	0.46	0.01	-0.08	0.48	0.09
	p	0.05*	0.95	0.72	0.04*	0.71
WC(cm) Female(N=28)	r	0.32	0.43	-0.21	0.28	0.45
	p	0.19	0.07	0.40	0.25	0.05*

Correlation coefficient (r) , * Correlation is significant ≤ 0.05 level (2-tailed)

There was an inverse correlation between TSH with T4 in healthy males (r= -0.54 ,p=0.01), also there was an inverse correlation between TSH and VLDL in healthy females(r= - 0.60,p=0.008).

Table(4): Correlation of thyriod stimulating hormone with thyriod metabolic hormone ,BMI ,WCand lipid profile of both gender

Groups \ Index		T3 (ng/ml)	T4 (mg/dl)	BMI (kg/m^2)	WC (cm)	Cholesterol (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL (mmol/l)	VLDL (mmol/l)
TSH(mIU/ml) Male(N=28)	r	-0.28	-0.54	0.02	0.16	-0.17	-0.04	0.46	-0.22	-0.27
	p	0.25	0.01*	0.93	0.52	0.49	0.86	0.055	0.37	0.27
TSH(mIU/ml) Female(N=28)	r	-0.29	-0.33	-0.02	-0.14	-0.22	-0.10	0.36	-0.19	-0.60
	p	0.23	0.17	0.92	0.58	0.36	0.68	0.14	0.43	0.008*

Correlation coefficient (r) , * Correlation is significant ≤ 0.05 level (2-tailed)

In healthy males,there was an inverse correlation between T3 and HDL (r= -0.51,p=0.03), while there was positive correlation among T3 with BMI and WC in healthy females (r= 0.48, p=0.04) and (r= 0.49,p=0.03) respectively.

Table(5): Correlation of T3 with BMI ,WC and lipid profile of both gender

Groups \ Index		BMI (kg/m^2)	WC (cm)	Cholesterol (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL (mmol/l)	VLDL (mmol/l)
T3(ng/ml) Male(N=28)	r	-0.12	-0.35	-0.10	0.05	-0.51	-0.01	0.15
	p	0.62	0.15	0.66	0.83	0.03*	0.94	0.54
T3(ng/ml) Female(N=28)	r	0.48	0.49	0.31	0.04	-0.001	0.35	0.09
	p	0.04*	0.03*	0.20	0.86	0.99	0.14	0.69

Correlation coefficient (r) ,* Correlation is significant ≤ 0.05 level (2-tailed)

The results showed there was positive correlation between T4 and LDL in healthy females(r= 0.48, p=0.04).

Table(6):Correlation of T4 with BMI ,WC and lipid profile of both gender

Groups \ Index		BMI (kg/m^2)	WC (cm)	Cholesterol (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL (mmol/l)	VLDL (mmol/l)
T4(mg/dl) Male(N=28)	r	0.26	0.17	0.24	-0.21	0.22	0.33	-0.31
	p	0.28	0.48	0.33	0.38	0.38	0.17	0.21
T4(mg/dl) Female(N=28)	r	0.02	-0.12	0.42	-0.06	-0.19	0.48	0.13
	p	0.93	0.62	0.07	0.80	0.43	0.04*	0.59

Correlation coefficient (r) * Correlation is significant ≤ 0.05 level (2-tailed)

Discussion

Levels of TG was significantly higher($p<0.05$) in males than females while the levels of T3 was significantly higher ($p<0.05$) in females than males of healthy people(Table 1) . Triiodothyronine (T3) upregulates LDL receptors by controlling the LDL receptor gene activation. This T3 mediated gene activation is done by the direct binding of T3 to specific thyroid hormone responsive elements (TREs). Furthermore, T3 controls the sterol regulatory element-binding protein-2 (SREBP-2), which in turn regulates LDL receptor's gene expression . T3 has also been associated with protecting LDL from oxidation. Another effect of T3 is the up-regulation of apolipoprotein AV (ApoAV), which plays a major role in TG regulation . Indeed, increased levels of ApoAV have been associated with decreased levels of TGs . Proposed mechanisms for this effect include the decrease of hepatic VLDL-TG production and the increase of plasma LPL levels and activity, resulting in increase of lipoprotein remnant generation due to enhanced LPL-mediated lipolysis of VLDL-TG . Moreover, a greater clearance of lipoprotein core remnants, caused by increased hepatic up-take due to an enhanced affinity for the LDL receptor, has also been ascribed to ApoAV (Pearce *et al*.,2008 ;Rizos *et al*.,2011). The elevation of TGs in hypothyroidism is caused by a reduced removal rate of TG from plasma due to a decrease in the activity of hepatic TG lipase(Park *et al*.,2011).

As the thyroid hormones especially T3 regulate both the resting metabolic rate and thermogenesis and lead to lipolysis,changes in thyroid hormones could also point to an adaptation process in obesity(Reinehr and Andler,2002).

Thyroid hormone also influences the transport of TG-rich lipoprotein through its effects on the lipoprotein lipase enzyme system.Hepatic lipase has also been shown to decrease in severe thyroid deficiency and to increase after L-thyroxine administration (Turhan *et al*., 2008).

Correlation analysis in healthy males showed positive corellation among BMI with WC, cholesterol and LDL, also in healthy females the results indicated positive correlation between BMI and WC as shown in table(2).The study also revealed that in healthy males there was positive correlation among WC with cholesterol and LDL, and there was also positive correlation between WC and VLDL in healthy females as shown in table(3).

The correlations observed in the current study between waist circumference and BMI were high and statistically significant in both sexes ,which agreement with study of Dagan *et al*., (2013).

In study of Faheem *et al*.,(2010) showed apositive correlation between cholesterol and BMI .BMI is often directly associated with total and LDL-cholesterol plasma concentrations, whereas an inverse relationship has been reported between HDL-cholesterol and BMI. In the present study, the association of total and LDL-cholesterol with BMI was found to be lower in women than in men which agreement with studies of Schroder *et al*.,(2003) and Weinbrenner *et al*.,(2006).Other study showed that obesity was associated with lower HDL-C levels in men (Al-Ajlan,2011).

Also serum lipid concentrations showed stronger correlations in males than in females not only with BMI but also with total body fat (TBF)(Choi *et al*.,2002). Yildiran *et al*.,(2011) shown that low HDL-C, high LDL-C and high TG levels are positively associated with an increase in BMI .

BMI and WC were positively correlated. WC was observed to be the more useful measure in predicting blood lipids for both sexes, especially among men. The differences in the strength of the association observed in men versus women could be due to higher muscle mass in men(Brenner *et al*.,2010).

There was an inverse correlation between TSH with T4 in healthy males, also there was an inverse correlation between TSH and VLDL in healthy females as shown in (Table 4).

Thyroid stimulating hormone (TSH) which is secreted by the pituitary gland, has an inverse logarithmic relationship with free thyroxine (fT4).TSH can be influenced by several factors. TSH shows diurnal variation, being high at night and low in the morning . Fasting status and estrogen also affect TSH levels. Estrogens cause increased secretion of thyroid binding globulin (TBG).On the other hand, TBG levels are depressed by androgens (Kaur *et al*.,2007).However, conflicting factors such as fasting status or diurnal change were excluded in our study because all blood samples were taken at 8-10 AM, after fasting. Thyroid dysfunction in the elderly is more prevalent than in the young . Moreover, the prevalence of thyroglobulin antibodies and thyroid peroxidase antibodies increases with age (Lee *et al*.,2011).

Other study demonstrates that average serum TSH concentrations and the prevalence of antithyroid antibodies are greater in women(Hollowell *et al*.,2002).The inverse loglinear relationship between TSH and free thyroid hormone due to the negative feedback of these hormones on the pituitary is well described (van Deventer *et al*.,2011).

TSH within the reference range may be positively associated with total serum cholesterol and LDL cholesterol, and negatively associated with high-density lipoprotein (HDL) cholesterol. It has also been shown that total serum cholesterol and LDL cholesterol were reduced after thyroxine treatment in individuals with TSH in the upper part of the reference range (Asvold *et al*.,2007).

Other study showed adipocytes and preadipocytes expressed TSH receptors, TSH binded with TSH receptors and induced preadipocytes to produce and release adipokines, some of them such as leptin played a very important role in the onset of metabolic syndrome and cardiovascular diseases(Lai *et al*.,2011).Positive associations between the TSH level and waist circumference, body mass index and blood pressure have been described (Wanjia *et al*.,2012).

TSH was associated positively with serum TG and negatively with serum HDL-C in women. In the whole population, TSH remained significantly and positively associated with TG after adjustment for age, sex, and BMI. TSH was positively associated with TC in the healthy population and positively associated with TC and LDL-C in healthy women .The combination of serum TSH, sex, and BMI has important effects on serum lipid parameters (Lu *et al*.,2011).

In healthy males,there was an inverse correlation between T3 and HDL, while there was positive correlation among T3 with BMI and WC in healthy females as shown in (Table 5).

Leptin affects thyroid deiodinase activities with activation of T4 to T3 conversion. and a positive association has been reported between the FT3 to FT4 ratio and both waist circumference and BMI in obese patients . This finding suggests a high conversion of T4 to T3 in patients with central fat obesity due to increased deiodinase activity as a compensatory mechanism for fat accumulation to improve energy expenditure(Biondi,2010).

The results showed there was positive correlation between T4 and LDL in healthy females as shown in (Table 6).

Study of Xu *et al*.,(2012) have confirmed the presence of an inverse relationship between serum thyroxin and cholesterol levels. Interestingly, *in vivo* and *in vitro* research on the function of TSH has shown that TSH, independent of thyroid hormones, can upregulate the expression of hepatic 3-hydroxy-3-methyl-glutaryl coenzyme A reductase (HMGCR), which is the rate-limiting enzyme in cholesterol synthesis, and increase the cholesterol content in the liver . Therefore, we hypothesized that TSH, independent of thyroid hormones ,would be positively associated with the serum cholesterol level .

Reverse cholesterol transport can also involve the transfer of cholesterol esters from HDLs to VLDLs and LDLs. This transfer requires the activity of the plasma glycoprotein cholesterol ester transfer protein (CETP). The transfer of cholesteryl esters from HDLs to VLDLs via CETP activity also involves an exchange of triglycerides from the VLDLs to the HDLs. VLDLs are eventually converted to LDLs and the cholesterol acquired from HDLs can be returned to the liver via the interaction of LDL with the hepatic LDL receptor. HDL cholesterol levels also tend to decrease because levothyroxine stimulates the cholesterol ester transfer protein (CETP)(Shekhar *et al*.,2011).

Conclusions: Thyroid metabolic hormone especially T3 regulate the metabolic rate and the mild thyroid dysfunction was linked to significantly changes in body weight ,lipid profile and likely represents risk factor for healthy and obesity.

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