THYROID FUNCTION IN OBESE CHILDREN AND ADOLESCENCE

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ABSTRACT

***Background**: Childhood obesity is one of the most serious public health challenges of the 21st century. The problem is global and is steadily affecting many low- and middle-income countries, particularly in urban settings. The prevalence has increased at an alarming rate. Globally, in 2010 the number of overweight children under the age of five is estimated to be over 42 million. Close to 35 million of these are living in developing countries.*

***Objectives:** The aim of this prospective study to find out if there is any abnormalities in thyroid function in simple obese children and adolescence or not.*

***Patient and method:** This prospective randomized case- control study is carried out on a 60 children with simple obesity selected from Al-Hussen University Hospital including f38 obese children (55.6%) with a BMI of more than 95th percentile for age and sex, and 22 children (44.4%) with a BMI between (15^{th} percentile-85^{th} percentile) for age and sex serving as controls. group of the sixthly children, 27 were males (45.6%) and 33 were females (54.4%). The mean age of control and obese children, was 8.40 ± 1.32, and 8.28 ± 1.47 years respectively.*

***Conclusion**: Obesity is associated with increased TSH levels breastfeeding is very important as children who did not receive breast milk are at great risk of obesity, TV watching may be a contributing factor in the development of obesity, unhealthy eating habits are one of the risk factors of obesity, elevated blood pressure is not uncommon in obese children even in young age.*

INTRODUCTION

Childhood obesity in general means an excess of body fat. However, Centers for Disease Control and Prevention (CDC) defines childhood obesity as at or above the 95^{th} percentile of BMI for age and sex and overweight as between 85^{th} to 95^{th} percentile of BMI for age and sex ***(Barlow et al., 2007, and Qazi, 2011)***. BMI is calculated using a child's weight and height. BMI does not measure body fat directly, but it is a reasonable indicator of body fatness for most children and teens

(Centers of Disease Control and Prevention, 2012).

Estimates indicated that approximately 17 percent of children and adolescents in the United States are obese with a BMI at or above the 95^{h} percentile, and over 33 percent are either obese or overweight with a BMI at or above the 85th percentile ***(Ogden et al., 2006)***. In developing countries, the transition from rural agrarian to urban economies has accelerated the appearance of obesity, which is accompanied by a shift in overall health burden from infectious diseases and undernutrition to Western chronic diseases such as cardiovascular disease, cancer and diabetes mellitus (DM) ***(Oken and Gillman, 2008)***.

Information from both genetic and molecular epidemiology suggests that genetic factors are involved in determining the susceptibility to gaining or losing fat in response to diet and physical activity treatment. The same applies to the risk of developing some of the co-morbidities of obesity. **(Li et al., 2013).**

Complecations associated with obesity include increased risk of DM (type 2), hypertension, dyslipidemia, sleep apnea, osteoarthritis, colon cancer in men who were obese during adolescence, Low self-esteem has been found in obese children as young as 5 years of age, eating disorders (including binge eating and bulimia nervosa), poor performance in school, fatty liver disease, gallstones, gastro-esophageal reflux and a higher prevalence of asthma in overweight and obese children.

Long before the definition of the metabolic syndrome, alternation in thyroid function were reported in obese patients. Body composition and thyroid hormones appeare to be closely related since the later is known to be involved in the regulation of the basal metabolism and thermogenesis, playing an important role in lipids and glucose metabolism, food intake and fat oxidation **(Cali and Caproio 2008).**

Leptin is a cytokine secreted by adipocytes in proportion to body's fat content. It binds to receptors in two different specific populations of neurons of the arcuate nucleus of the hypothalamus. Pathogenic mutations in both leptin (LEP) and its receptor (LEPR) in extreme forms of early-onset obesity were identified **(Clement et al., 2013).**

Subjects with congenital leptin deficiency exhibit normal weight at birth but gain weight rapidly in the early postnatal period. Leptin deficiency is associated with

marked hyperphagia, impaired satiety, and excessive fat deposition in the trunk and limbs ***(Montague et al., 2015)***.

Although thyroid function is usually normal in obese subjects, it is known that TSH and BMI are positively correlated. Many studies in children, adolescents, and adults have demonstrated that TSH levels are slightly increased in obese subjects as compared to normal weight humans. Furthermore, not only a positive correlation between BMI and serum TSH has been demonstrated, but also a positive correlation between weight gain during 5 years and a progressive increase of serum TSH ***(Knudsen et al., 2005, and Nyrnes et al., 2006).***

The status of slightly increased TSH levels associated with normal peripheral thyroid hormone levels has been entitled subclinical hypothyroidism. Most obese children and adults with increased TSH level have no thyroid disease. It seems that increased TSH levels are rather a consequence than a cause of obesity ***(Reinehr, 2010)***.

AIM OF THE WORK

The aim of this prospective study to find out if there is any abnormalities in thyroid function in obese children and adolescence or not.

PATIENTS AND METHODS

Out of this prospective randomized case- control study will be conducted on a 60 children, 38% were obese and 22% were control.

Inclusion criteria:

All apparently healthy overweight and obese children are be included in this study.

Exclusion criteria:

1. Children with manifestations of hypothyroidism.
2. Children with other endocrinal diseases.
3. Children with Down Syndrome.

All enrolled children will be subjected to the following:

- Full clinical history.
- Clinical examination including:

1. General examination.
2. Length.
3. Weight.
4. Head circumference.
5. BMI.

- Laboratory investigations including:

1. Free T4.
2. T3
3. TSH
4. TGs
5. Thyroid antibodies.

RESULTS

Obesity in childhood is increasing worldwide. In recent years, there has been increasing focus on the relationship between thyroid function and weight status. Whereas it is well known that hyperthyroidism leads to weight loss and hypothyroidism is associated with weight gain, changes in thyroid homeostasis that occur in obesity are controversial In our study, it was noted that the mean TSH concentration was significantly higher in obese group 6.58 ± 4.74 µIU/mL compared with control group 1.95 ± 1.86 µIU/mL.

Table (1): Comparison between age and sex in patients and control groups.

		Control group No. = 40	Patients group No. = 60	Test value	P-value	Sig.
Age (years)	Mean ± SD	8.4 ± 1.32	8.3 ± 1.46	-0.348•	0.729	NS
	Range	6 – 10	6 – 10			
Sex	Males	18 (45.0%)	33 (55.0%)	0.960*	0.327	NS
	Females	22 (55.0%)	27 (45.0%)			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant; NA: Not applicable
*: Chi-square test; •: Independent t-test

This table show a comparison between age and sex in patients and control groups no statistically significant differences regarding items shown in (**Table 1).**

Table (2): Comparison between children characteristics of control and obese groups.

		Control group No. = 40	Patients group No. = 60	Test value	P-value	Sig.
Breast feeding	No Yes	6 (15.0%) 34 (85.0%)	33 (55.0%) 27 (45.0%)	16.141*	0.000	HS
Weaning age	Mean ± SD Range	4.63 ± 1.25 2 – 6	4.33 ± 1.07 2 – 6	-1.247•	0.215	NS
Meals/day	Mean ± SD Range	3.63 ± 0.49 3 – 4	3.77 ± 0.43 3 – 4	1.532•	0.129	NS
Fruit in gm /day	Mean ± SD Range	1.73 ± 1.34 0 – 5	2.12 ± 1.5 0 – 5	1.336•	0.185	NS
Vegetable in gm / day	Mean ± SD Range	1.28 ± 0.55 0 – 2	1.28 ± 0.61 0 – 2	0.069•	0.945	NS
Red meat gm/wk	Mean ± SD Range	0.75 ± 0.63 0 – 2	0.8 ± 0.63 0 – 2	0.388•	0.699	NS
Chicken gm/wk	Mean ± SD Range	1.58 ± 0.81 0 – 3	1.48 ± 0.87 0 – 4	-0.528•	0.598	NS
Soda bottle/wk	Mean ± SD Range	1.45 ± 1.08 0 – 5	3.43 ± 1.71 0 – 7	6.505•	0.000	HS

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant; NA: Not applicable
*: Chi-square test; •: Independent t-test

This table show a comparison between children characteristics of control and obese groups revealed that the mean of breast feeding (*p=0.000*), soda bottles/week (*p=0.000*), were significantly higher in obese group than control group **(Table 3).**

Table (3): Comparison between family history of control and obese groups.

Family history	Control group		Patients group		Test value*	P-value	Sig.
	No.	%	No.	%			
No	34	85.0%	28	46.7%	17.315	0.008	HS
Diabetes mellitus	3	7.5%	8	13.3%			
Hypertension	0	0.0%	2	3.3%			
Obesity	3	7.5%	10	16.7%			
Diabetes mellitus+ hypertension+obesity	0	0.0%	5	8.3%			
Diabetes mellitus+obesity	0	0.0%	2	3.3%			
Hypertension+obesity	0	0.0%	5	8.3%			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant; NA: Not applicable
*: Chi-square test

This table show a comparison between family history of control and obese groups which reveals a statistically significant difference.

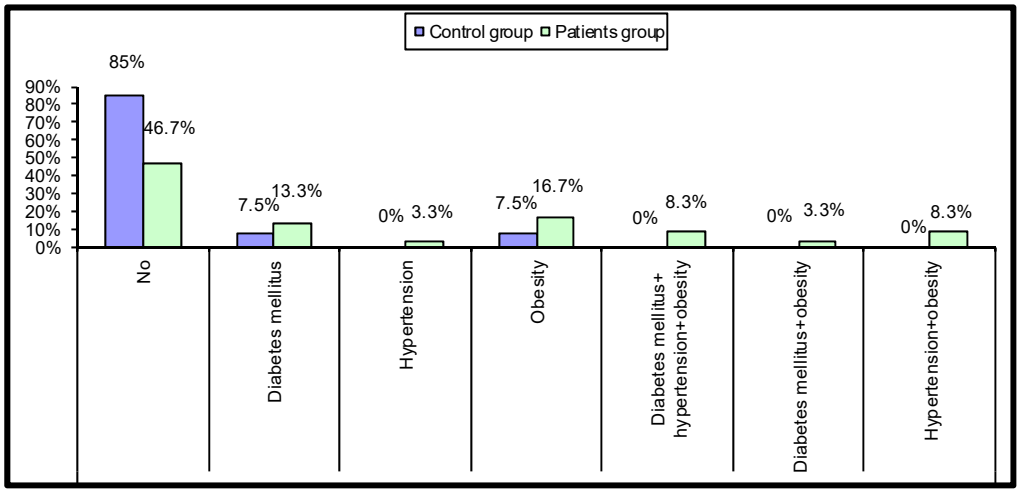

Figure (1): Comparison between family history of control and obese groups.

Table (4): Comparison between clinical data of patients and control groups.

		Control group	Patients group	Test value•	P-value	Sig.
		No. = 40	No. = 60			
Weight (kg)	Mean ± SD	27.75 ± 4.94	48.1 ± 10.49	11.443	0.000	HS
	Range	20 – 42	30 – 69			
Height (cm)	Mean ± SD	125.48 ± 7.4	131.8 ± 9.63	3.517	0.001	HS
	Range	110 – 141	110 – 149			
BMI (kg/m²)	Mean ± SD	17.5 ± 1.67	27.28 ± 2.71	20.404	0.000	HS
	Range	14.58 – 21.13	23.19 – 33.97			
Pulse (bpm)	Mean ± SD	81.75 ± 3.85	82.42 ± 4.27	0.795	0.428	NS
	Range	75 – 90	75 – 90			
Systolic BP (mmHg)	Mean ± SD	98 ± 5.16	100.33 ± 4.86	2.294	0.024	S
	Range	90 – 110	90 – 110			
Diastolic BP (mmHg)	Mean ± SD	63.25 ± 4.74	65.17 ± 5.04	1.907	0.059	NS
	Range	60 – 70	60 – 70			
Temp (°c)	Mean ± SD	37 ± 0	37 ± 0	NA	NA	NA
	Range	37 – 37	37 – 37			
Respiratory rate (cpm)	Mean ± SD	20.2 ± 1.02	20.63 ± 1.41	1.670	0.098	NS
	Range	18 – 22	18 – 24			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant; NA: Not applicable
•: Independent t-test

This table show statistically significant difference in weight p-value (0.000), height p-value (0.001), BMI p-value (0.000) and systolic BP p-value (0.024) between patients and control group.

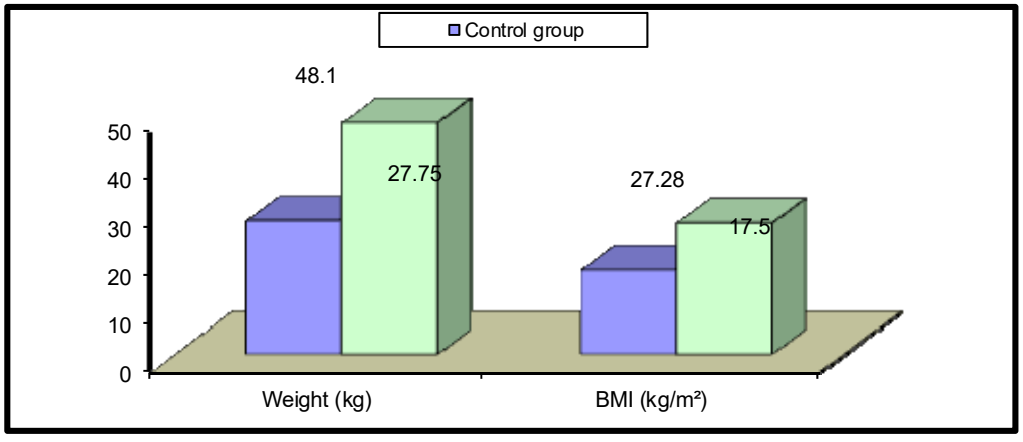

Figure 2: Comparison between weight and BMI of patients and control groups.

Table (5): Comparison between biochemical characteristics (thyroid functions) of control and obese groups.

		Control group	Patients group	Test value	P-value	Sig.
		No. = 40	No. = 60			
TSH	Mean ± SD	1.95 ± 1.86	7.24 ± 5.03	6.352•	0.000	HS
	Range	0.1 – 9	0.5 – 22			
	Normal	35 (87.5%)	27 (45.0%)	28.635*	0.000	HS
	Elevated	2 (5.0%)	33 (55.0%)			
	Low	3 (7.5%)	0 (0.0%)			
T4	Mean ± SD	8.52 ± 3.72	4.13 ± 2.98	-6.516•	0.000	HS
	Range	0.6 – 16.5	0.2 – 12.4			
	Normal	30 (75.0%)	26 (43.3%)	26.860*	0.000	HS
	High	7 (17.5%)	1 (1.7%)			
	Low	3 (7.5%)	33 (55.0%)			
T3	Mean ± SD	1.43 ± 0.49	0.81 ± 0.55	-5.731•	0.000	HS
	Range	0.4 – 2.1	0.1 – 2			
	Normal	36 (90.0%)	39 (65.0%)	14.391*	0.001	HS
	High	2 (5.0%)	0 (0.0%)			
	Low	2 (5.0%)	21 (35.0%)			
TPO	Mean ± SD	76.2 ± 20.33	72.81 ± 14.45	-0.976•	0.332	NS
	Range	0.12 – 91.42	0.86 – 91.19			
	Normal	38 (95.0%)	57 (95.0%)	0.000*	1.000	NS
	High	2 (5.0%)	3 (5.0%)			
	Low	0 (0.0%)	0 (0.0%)			
TG	Mean ± SD	24.14 ± 35.85	47.93 ± 28.07	3.713•	0.000	HS
	Range	1.8 – 227	15.2 – 163.1			
	Normal	38 (95.0%)	55 (91.7%)	0.410*	0.522	NS
	High	2 (5.0%)	5 (8.3%)			
	Low	0 (0.0%)	0 (0.0%)			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant; NA: Not applicable
*: Chi-square test; •: Independent t-test

Comparison between biochemical characteristics of control and obese groups revealed significant difference regarding the mean of TSH *(p=0.000)*, T_4 *(p=0.000)* and T_3 *(p=0.000)*.

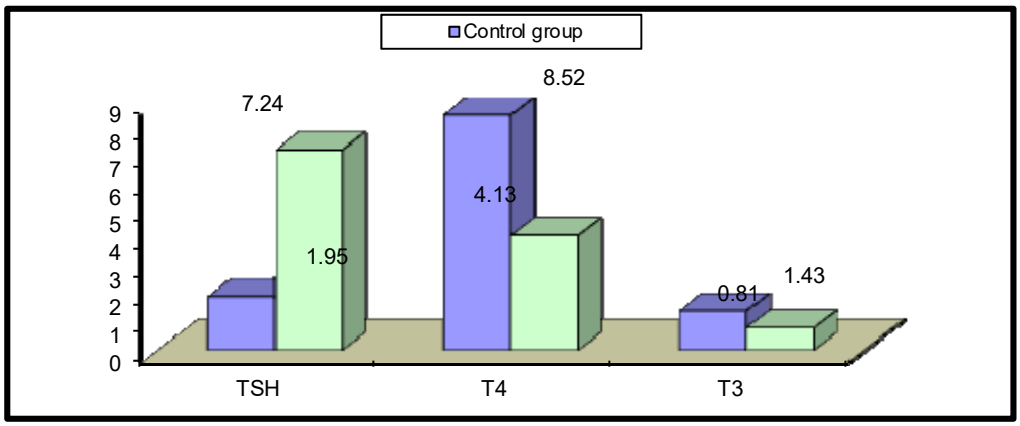

Figure (3): Comparison between biochemical characteristics of control and obese groups.

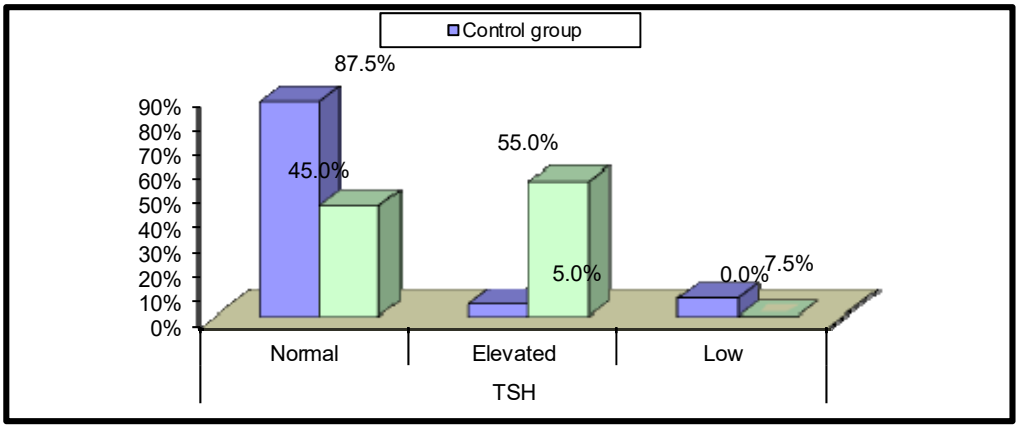

Figure (4): Comparison between biochemical characteristics of control and obese groups.

Table (5): Comparison between exercise or sports and TV Hrs/day of control and obese groups

		Normal TSH	Elevated TSH	Test value	P-value	Sig.
		No. = 27	No. = 33			
Exercise or sports	No	25 (92.6%)	31 (93.9%)	0.043*	0.835	NS
	Yes	2 (7.4%)	2 (6.1%)			
T.V hrs/day	Mean ± SD	4.19 ± 1.18	4.42 ± 1.12	0.804•	0.425	NS
	Range	2 – 6	2 – 6			

P-value > 0.05: Non significant; P-value < 0.05: Significant; P-value < 0.01: Highly significant; NA: Not applicable
*: Chi-square test; •: Independent t-test

DISCUSSION

In our study, it was noted that the mean TSH concentration was significantly higher in obese group 6.58 ± 4.74 μIU/mL compared with control group 1.95 ± 1.86 μIU/mL which comes in agreement with the study done by **Bhowmick et al.,** (2007) who studied 308 obese children and 286 non obese children. Elevated TSH levels were noted in 36 patients within the obese group (11.7%) but only two in the control group (<0.7%). **Reinehr and Andler, (2002); Reinehr et al., (2006); Reinehr et al., (2008); Kumar et al., (2009); Grandone et al.**

It was noted that the mean T4 concentration was significantly higher in control group 8.52 ± 3.72 μg/dL compared with obese group 4.38 ± 3.10 μg/dL. Also the mean T3 concentration was significantly higher in control group 1.43 ± 0.49 ng/mL compared with obese group 0.81 ± 0.54 ng/mL.

In our study, it was noted that the mean T.V hours/day was significantly higher in obese group 4.32 ± 1.15 hour compared with control group 2.73 ± 1.01 hour which comes in agreement with the study done by **Suresh et al., (2011)** who found that TV viewing in adolescence and early adulthood was found on change in BMI over time.

It was noted that the mean SBP was significantly higher in obese group 100.60 ± 4.70 mmhg compared with control group 98.00 ±5.16 mmhg, while no significant difference as regards DBP which comes in agreement with the study done by **Tu et al., (2011)** who studied 1111 children in India and the SBP and DBP in obese children with normal TSH gives no significant difference with SBP and DBP in obese children with elevated TSH.

Also it was noted that obese subjects with elevated TSH values had a mean body mass index similar to that for obese subjects with normal TSH levels (28.02 ± 2.80 and 26.83 ± 2.57 kg/m2, respectively).

Our study comes in agreement with the study done by ***Dekelbab et al., (2010)*** obese subjects with increased TSH values had a mean body mass index similar to that for obese subjects with normal TSH levels (34.98 ± 6.12 and 34.29 ± 7.84 kg/m^2, respectively).

Our study comes in agreement with the study done by ***Ford et al., (2010)*** who studied 106 newly referred obese children, the study was conducted at a children's hospital in England. They found

that providing real time feedback to participants during meals to slow down speed of eating and reduce total intake by a computerized device had significantly lower mean BMI after 12 months.

In obesity, it is well recognized that adipose tissue secretes inflammatory cytokines and that some of these, for example tumor necrosis factor (TNF)-a, inter-leukin (IL)-1, and IL-6, are secreted into the general circula-tion and may be associated with systemic symptoms. These cytokines inhibit sodium iodide transporter mRNA expression and iodide uptake activity in human and rat thyroid cells, perhaps playing a role in the compensatory rise in TSH levels observed in obese patients ***(Kershaw and Flier, 2006).***

CONCLUSIONS

- Obesity is associated with increased TSH levels.
- Breastfeeding is very important as children who did not receive breast milk are at great risk of obesity.
- TV watching may be a contributing factor in the development of obesity.
- Unhealthy eating habits are one of the risk factors of obesity.
- Elevated blood pressure is not uncommon in obese children even in young age.

REFERENCES

1. **Athanasopoulos DT, Garopoulou AI, Dragoumanos VP**. Childhood obesity and associated factors in a rural Greek island. Rural Remote Health 2011; 11(4):1641-1949.
2. **Beyerlein A, Toschke AM, Kries R.** Breastfeeding and Childhood Obesity: Shift of the Entire BMI Distribution or Only the Upper Parts? Obes 2008; 16**:** 2730–2733.
3. **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(7): 773-776
4. **Biondi B, Cooper DS.** The clinical significance of subclinical thyroid dysfunction. Endocrinol Rev 2008; 29: 76-131.
5. **Centers of Disease Control and Prevention**. Overweight and obesity. Available at: http://www.cdc.gov/ obesity/childhood/basics.html. Accessed on August, 20, 2012.*
6. **Dekelbab BH, bou Ouf HA, Jain I**. Prevalence of elevated thyroid-stimulating hormone levels in obese children and adolescents. Endocr Pract 2010; 16:187-190.
7. **Farooqi IS, Bullmore E, Keogh J, Gillard J, O'Rahilly S, Fletcher PC.** Leptin regulates striatal regions and human eating behavior. Sci 2007; 317(5843):1355-1361.

8. **Ford AL, Bergh C, Sodersten P, Matthew A, Sabin MA, Hollinghurst S, Hunt LP, Shield JP.** Treatment of childhood obesity by retraining eating behaviour: randomised controlled trial. BMJ 2010; 340:5388-5394.

9. **Goyal RK, Shah VN, Saboo BD, Phatak SR, Shah NN, Gohel MC, Raval PB, Patel SS**. Prevalence of Overweight and Obesity in Indian Adolescent School Going Children: Its Relationship with Socioeconomic Status and Associated Lifestyle Factors. JAPI 2010; 58:151-158.

10. **Grandone A, Santoro N, Coppola F, Calabro P, Perrone L, Giudice EM.** Thyroid function derangement and childhood obesity: an Italian experience. BMC Endocr Disord 2010; 10:8-14.

11. **Han JC, Lawlor DA, Kimm SY.** Childhood obesity. Lancet 2010; 375(9727):1737-1748.

12. **Hay WW, Levin MJ, Sondheimer JM, Deterding RR.** Endocrine disorders. Current Diagnosis and Treatment: Pediatrics. 19th edition. McGraw Hill; 2009.

13. **Huh SY, Rifas-Shiman SL, Taveras EM, Oken E, Gillman MW.** Timing of Solid Food Introduction and Risk of Obesity in Preschool-Aged Children. Pediatr 2011; 127 (3): 544-551.*

14. **Jelalian E, Steele RG.** Health consequences of obesity in children and adolescents. Handbook of Childhood and. Adolescent Obesity. Springer; 2008.

15. **Klijn PH, Baan-Slootweg OH, Stel HF.** Aerobic exercise in adolescents with obesity: preliminary evaluation of a modular training program and the modified shuttle test. BMC Pediatr 2007; 7:19-29.

16. **Kumar KH, Verma A, Muthukrishnan J, Modi KD.** Obesity and Thyrotropinemia. Indian J Pediatr 2009; 76:933-935.

17. **Licinio J, Ribeiro L, Busnello JV, Delibasi T, Thakur S, Elashoff RM, Sharma A, Jardack PM, Depaoli AM, Wong ML.** Effects of leptin replacement on macro- and micronutrient preferences. Int J Obes 2007; 31(12):1859-1863**.**

18. **Marras V, Casini MR, Pilia S, Carta D, Civolani P, Porcu M, Uccheddu AP, Loche S.** Thyroid function in obese children and adolescents. Horm Res Pediatr 2010; 73:193-197.

19. **Melmed S, Polonsky KS, Larsen PR, Kronenberg HM.** Adrenal cortex and endocrine hypertension. Williams textbook of endocrinology. 12th edition. Saunders Elsevier; 2011.

20. **Mushtaq MU, Gull S, Abdullah HM, Shahid U, Shad MA, Akram J.** Waist circumference**,** waist-hip ratio and waist-height ratio percentiles and central obesity among Pakistani children aged five to twelve years. BMC Pediatr 2011; 11:105-120.

21. **Nathan BM, Moran A.** Metabolic complications of obesity in childhood and adolescence: more than just diabetes. Curr Opin Endocrinol Diabetes Obes 2008; 15: 21-29.

22. **Qazi HA.** Childhood obesity and parks and playgrounds: A review of issues of equality, gender and social

support. J Res Med Sci. 2011; 16(4): 553-558. *

23. **Radetti G, Kleon W, Buzi F, Crivellaro C, Pappalardo L, Iorgi N, Maghnie M.** Thyroid function and structure are affected in childhood obesity. J Clin Endocrinol Metab 2008; 93:4749-4754.

24. **Reinehr T, Isa A, de Sousa G, Dieffenbach R Andler W.** Thyroid hormones and their relation to weight status. Horm Res 2008; 70:51-57.*

25. **Reinehr T.** Obesity and thyroid function Molecular and Cellular. Endocrinol 2010; 316: 165–171.

26. **Reinehr T.** Thyroid function in the nutritionally obese child and adolescent. Curr Opin Pediatr 2011; 23(4):415-420.

27. **Rivkees SA, Dinauer C.** An optimal treatment for pediatric Graves' disease is radioiodine. J Clin Endocrinol Metab 2007; 92:797-800.

28. **Suresh V, Rupnath K, Ramesh V, Rojarani M, Ramadevi T, K R S Sambasivarao.** Television watching and sleep promotes obesity in urban and semi-urban children in India. J Toxicol Environ Health Sci 2011; 3(1):1-7.

29. **Tu W, Eckert GJ, DiMeglio LA, Yu Z, Jung J, Pratt JH.** Intensified Effect of Adiposity on Blood Pressure in Overweight and Obese Children. Hypertension 2011; 58(5):818-824.

30. **World Health Organization. Childhood overweight and obesity.** Available at: http://www.who.int/dietphysicalactivity/childhood/en/. Accessed on August 22, 2012.

وظيفة الغدة الدرقية في الأطفال والمراهقين البدناء

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كلية طب الأزهر

تعرف السمنة بأنها زيادة معامل الكتلة الجسمية على ٩٥ بالنسبة للعمر والجنس حسب منحنيات النمو، وهي مشكلة صحية منتشرة في العالم كله.

إن الأطفال الذين يعانون من السمنة معرضون بشدة لخطر الإصابة بمرض ارتفاع ضغط الدم، ومرض السكر، وكذلك اختلال نسبة الدهون بالدم' مما يؤدي إلى زيادة خطر الإصابة بأمراض الأيض والقلب والأوعية الدموية.

لقد تم إجراء هذا البحث على 60 طفل من الجنسين ممن يزيد معامل الكتلة الجسمية لديهم عن ٩٧ بالنسبة للعمر والجنس حسب منحنيات النمو، و٤٠ طفل من الجنسين ممن يتراوح معامل الكتلة الجسمية لديهم ما بين (١٥-٨٥) بالنسبة للعمر والجنس حسب منحنيات النمو.

وقد أجري هذا البحث بقسم الأطفال بمستشفي الحسين الجامعي, جامعه الازهر. وقد خضع جميع الأطفال لتحاليل تراي ايدوثيرونين, والثيروكسين، والهرمون المحفز للغدة الدرقية، والأجسام المضادة للغدة الدرقية. وقد شمل البحث الأطفال ما بين (٦-١٠) سنوات من الجنسين مع أخذ تاريخ مرضي و فحص جسماني كامل لهم.

وقد بين البحث أن متوسط الهرمون المحفز للغدة الدرقية أعلى في الأطفال المصابين بالسمنة مقارنة بالأطفال ذوي الأوزان الطبيعية.

أما متوسط تراي ايدوثيرونين فقد كان أعلى في الأطفال ذوي الأوزان الطبيعية مقارنة بالأطفال المصابين بالسمنة.

وكذلك كان متوسط الثيروكسين أعلى في الأطفال ذوي الأوزان الطبيعية مقارنة بالأطفال المصابين بالسمنة.

وبخصوص متوسط ساعات مشاهدة التلفزيون في اليوم فقد كانت أعلى في الأطفال المصابين بالسمنة مقارنة بالأطفال ذوي الأوزان الطبيعية.

وقد تلقى الرضاعة الطبيعية ٣٤ (٨٥٪) من الأطفال ذوي الأوزان الطبيعية, و٢٤ (٤٨٪) من الأطفال المصابين بالسمنة.

أما بالنسبة للتاريخ المرضي فقد كان ٦ (١٥٪) من الأطفال ذوي الأوزان الطبيعية لديهم تاريخ مرضي من أمراض السمنة، أو السكر، أو ارتفاع ضغط الدم, بينما ٢٧ (٥٤٪) من الأطفال المصابين بالسمنة لديهم تاريخ مرضي.

لم يكن هناك فارق يذكر بالنسبة لعدد الوجبات في اليوم, و لكن بالنسبة لعدد علب الصودا في الأسبوع مثلا, فقد كان أعلى في الأطفال المصابين بالسمنة مقارنة بالأطفال ذوي الأوزان الطبيعية.

وفي الختام, نجد أن السمنة تقترن بنقص نشاط الغدة الدرقية، وبالرضاعة الغير طبيعية, وبمشاهدة التلفزيون, والعادات الغذائية الغير صحية, ولذلك نوصي بأن يتم دراسة تأثير إنقاص الوزن على وظائف الغدة الدرقية في الأطفال.