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Study of Growth Hormone and Cortisol in Nutritionally Stunted and Obese School Age Children

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Abstract: The prevalence of obesity is increasing worldwide even in developing countries that have rationally experienced high rates of malnutrition. Obesity is prevalent across all economic levels and age groups due to reduced physical activity and consumption of high fat diets. Childhood obesity can lead to life threatening condition including diabetes, high blood pressure, heart disease, sleep problems concern psychological disorders. This study aimed to investigated the relationship between stunted growth and overweight status for children between 9-11 years. The relation between chronic malnutrition (in the form of stunted growth) and obesity and level of growth hormone and cortisol).**Subject and methods:** The target population was school age children both sexes, 9-11 years in Giza Governorate. Children were screened for weight, height and BMI was calculated, dietary history was taken in addition to biochemical investigation including level of growth hormone, plasma cortisol, lipid profile and then data were analyzed using SPSS/PC. **Results:** Obesity was associated with an observe lipid profile there were significantly higher cholesterol, triglyceride, growth hormona, cortisol levels among obese than non obese children. On the contrary HDL-C was statistically significantly lower among obese children than non-obese one growth hormone was also significantly higher among obese than non-obese children. Cortisol level was higher among obese children than non-obese ones. Family back grounds has an impact on incidence of obesity. Obese children was breakfast skippers, had consume more fast foods and ate less fresh foods. **Conclusion:** Results of this study indicate negative effect of obesity on lipid profile and cortisol, association of obesity and chronic malnutrition will super add to cardiovascular risk factor among obese children results of this study may help policy markers in designing accurate intervention programs.

Key words: Obesity- School children- Short stature- Cortisol- Grwoth hormone – Lipid profile.

INTRODUCTION

The prevalence of obesity is increasing worldwide; even in developing countries that have traditionally experienced high rates of malnutrition. Obesity is prevalent across all economic levels and age groups. Traditional explanations for these observations include reduced physical activity and consumption of high-fat diets (Popkin; 2006). childhood obesity can lead to life-threatening conditions including diabetes, high blood pressure, heart disease, sleep problems, cancer, and psychological disorders (Kimm & Obarzanek, 2002).

Research has suggested that malnutrition in early life may play a role in promoting adult obesity. In particular, studies on 3 continents showed that nutritional stunting, which is usually caused by chronic malnutrition; is positively associated with adult fatness (Popkin *et al*, 1996). The mechanisms by which early nutrition exerts long-term programming effects in humans are not known, but proposed mechanisms include functional alterations in growth processes, gene expression, cell number, and clonal selection within organs and tissues (Branca & Ferrari ;2002).

In addition, previous study observed an association between excess weight gain and dietary fat content in stunted Brazilian children but not in nonstunted control children (Hoffman *et al*, 2000). This is suggestive of an increase in the efficiency of dietary fat utilization that could lead to increased body fat content over time.

Many of the detrimental health effects of obesity can be traced to a low-level, chronic inflammatory state, the result of adipocyte cytokine production in the body's fat stores. Many of these adipose-derived mediators

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have been related to alterations in catecholamines, glucocorticoids, insulin, and growth hormone (GH) (Stratakis CA, 2006). Consequently, an increased understanding of the mechanisms that control body composition will be essential to optimally "re-balance" energy intake and expenditure in today's children and adolescents. Of particular importance are the ways in which obesity alters the hormonal response to physical activity, a major factor in the distribution of fat and lean tissue in adults and children. GH and other elements of the insulin like growth factor-I (IGF-I) axis, key regulators of fat and lean tissue, are remarkably sensitive to brief bouts of physical activity and to fitness in general. Previous studies suggested that the GH response to brief exercise may be attenuated by obesity (Hershberger *et al*; 2004).

Low height-for-age is a characteristic finding among children in underdeveloped countries and among some children from underprivileged families in developed countries. Short stature is presumed to represent the effects of chronic malnutrition combined with the impact of infectious diseases. However, in some populations low height-for-age is paradoxically accompanied by increased weight-for-height. This pattern has been demonstrated in surveys in Egypt, in Latin American populations children, and among Hispanic and Native American children in the US (Brink *et al*, 1978;De Assis M *et al*, 2007).

Co-existence between malnutrition and obesity are associated with important metabolic changes. Results from stunted children showed higher susceptibility to the effects of higher fats diets, lower fat oxidation, and higher central fat and higher body fat gain. A model to explain how early malnutrition alters energy balance in adults is outlined. In the presence of a relative food intake insufficiency, a higher cortisol/insulin ratio, associated with lower levels of IGF-1 will lead to lower muscle gain and linear growth, impaired lypolysis and fat oxidation (Fanjiang G. *et al*., 2007). When these hormonal changes are combined with a higher fat/carbohydrate and/or marked decreased in physical activity, obesity with short stature will occur (Reinehr *et al*; 2007).

Obesity is a major related cardiovascular disease risk factor, and excess body weight is closely linked to low serum HDL-cholesterol concentrations. Large-scale studies now indicate that <5—10% of the population variation in serum HDL cholesterol can be explained by weight adjusted for height expressed as body mass index (BMI; in kg/m2) (Nancy F *et al*., 2007; Popkin *et al*,1996).

MATERIALS AND METHODS

This study Was analytical case- control study

Subjects:

Target population is school-age children, both sexes. Age range: 9-11years. The study Was done in 2 governmental primary schools at. Dokki district Giza governorate (ABou Baker El sedek Primary School & Alormon Primary School).

Children Was screened for Weight, Height. Height/age, Weight/age, and Body Mass Index/age (BMI) Was calculated according to WHO Standerds.Children Was divided into two groups by using body mass index (BMI), defined as the weight in kilograms divided by the square of the height in meters (kg/m2). A BMI > 95^{th} percentiles of The World Health Organization (WHO) Standards is defined as obese. And a BMI ≥ 10^{th} ≤ 85^{th} percentile of WHO Standards is defined as normal weight. Each group will include normal height/age children (>-2 SD of the WHO references) and low height/age children (≥ -2 SD of the WHO references); matching in age and sex.

Inclusion Criteria:

1. Children between 9 to 11 years .i.e. at 4^{th} primary to six primary grades
2. Both sexes are included.
3. Pre pubescent children (tanner stage 1)

Exclusion Criteria:

1. Children below 9 or over 11years.
2. Endocrinal and genetic obesity.
3. Children with chronic debilitating diseases: e.g. (diabetes mellitus, rheumatic and congenital heart diseases, and chronic lung diseases, hypertension),and children on corticosteroid therapy.
4. Mentally affected children.

Ethical Criteria:

Written informed consent will be obtained from parents after explanation of the aim of the study.

Methods:

All children Was subjected to:

1-Thouroug history taking: that includes information on dietary pattern and physical activity.

2-Meticulous Medical Examination: that includes heart, chest, abdomen, blood pressure and resting heart rate measurements.

3-Anthropometric measurements, that include**:** weight, height, skin fold thickness (triceps, biceps) and circumferences (Head and mid upper arm, waist & hip). Weight for age & height for age Z scores and BMI will be calculated using the anthro 1.01 program of the computer.

4- Pubertal development Was assessed according to Tanner's score.

5-Biochemical investigations: 5 ml venous blood Was withdrawn from each child after an over night fast of 12 hours. Plasma Was separated and kept at -20^{o} c until analysis.

○***Growth Hormone(GH):***

Was determined by immuno-enzymatic assay using commercial kit purchased from monobind Inc . (ACCU Bind U.S.A Elisa microwells product code 1725-300)

○***Plasma Cortisol Level:***

Was determined by immuno-enzymatic assay using commercial kit purchased from monobind Inc . (ACCU Bind U.S.A Elisa microwells product code 3625-300)

Lipid Profile:

- Plasma total cholesterol: by quantitative enzymatic calorimetric techniques using commercial kit purchased from Biocon diagnostic (Germenany) (BD-GB-CHOL-02/01)
- Plasma triglyceride: by quantitative enzymatic calorimetric techniques after the method of Titeze using commercial kit purchased from Biocon diagnostic (Germenany) (BD-GB-CHOL-03/2)
- Plasma high density lipoprotein- Cholesterol (HDL):by phosphotungstate precipitation method of Lopez-Virella using commercial kit purchased from Biocon diagnostic (Germenany) (BD-GB-CHOL-03/02)
- Plasma low density lipoprotein – Cholesterol (LDL)
  Was calculated according to Friedwald *et al.*

Statistical Analysis:

Data Was analyzed using the statistical package for social science for personal computers (SPSS/PC);11.5: frequency distribution, percentage distribution, range, mean & standard deviation; student's t-test one way analysis of variance (ANOVA) and pearson¢s correlation coefficients (r) P Was used for analysis and presentation of data values less than 0.05 Was considered statistically significant.

RESULTS AND DISCUSSION

Table 1: Mean (S.D) of lipid profile growth hormone and cortisol among non- stunted and stunted groups of obese children

Obese	Non-stunted (N= 27)	Stunted (N= 13)	t	P
Cholesterol(mg/dl)	171.6 ±22.6	184.0 ±21.1	1.6	0.1
Triglyceride(mg/dl)	111.2 ±33.2	141.2 ±25.2	3.1	0.003
H-IDL (high-density)(mg/dl)	46.6± 9.1	51.3 ±9.2	1.5	0.13
LDL (low-density)(mg/dl)	87.6 ±9.8	88.7 ±8.1	0.32	0.74
Growth hormone(GH)(ng/dl)	4.9 ±1.9	3.3 ±0.9	2.9	0.006
Cortisol(µg/dl)	9.4 ±4.0	11.1 ±3.8	2.2	0.05

N.S= non significant.
T= Student T test
P = P £ 0.05 (Significance Level)

Table 2: Mean (S.D) of lipid profile, growth hormone and cortisol among non- stunted and stunted groups of non-obese children

Non Obese	Non stunted (N= 19)	Stunted (N= 21)	t	P
Cholesterol(mg/dl)	147.4 ± 8.6	154.9 ± 9.8	2.5	0.01
Triglyceride (TG)(mg/dl)	64.2 ±10.3	72.5 ± 15.6	1.9	0.06
HIDL (high-density)(mg/dl)	56.8 ± 6.5	48.9 ±7.9	3.4	0.002
LDL (low-density)(mg/dl)	82.4 ± 9.5	95.2 ±6.5	4.9	0.000
GI I (growth hormone)(ng/dl)	4.2 ±6.59	3.3 ±0.6	2.7	0.01
Cortisol(µg/dl)	7.8 ± 2.9	9.6 ±3.2	1.98	0.05

N.S= non significant.
T = Student T test
P = P £ 0.05 (Significance Level)

Table 3: Comparison between stunted and non stunted subgroups of obese and non-obese children as regard biochemical parameters

	Obese		Non obese		F	P
	Non-stunted (N=27) Mean + SD	Stunted (N=13) Mean ± SD	Non stunted (N= 19) Mean ± SD	Stunted(N=21) Mean ± SD		
Cholesterol(mg/dl)	171.6 ±22.6	184.0 ± 21.1	147.4 ± 8.6	154.9 ± 9.8	15.7	0.00
Triglyceride(mg/dl)	111.2+33.2	141.2 ± 25.2	46.2 ± 10.3	72.5±15.6	44.0	0.00
HDL -C high density(mg/dl)	46.6 ± 9.1	51.3 ± 9.2	56.8 ± 6.5	48.9 ± 7.9	5.9	0.00
LDL-C low- density(mg/dl)	87.6 ± 9.8	88.7 ± 8.1	82.4 ± 9.5	95.2 ± 6.5	7.2	0.00
GH growthhormone(ng/dl)	4.9 ± 1.9	3.3 ± 0.9	4.2 ± 6.59	3.3 ± 0.6	10.5	0.00
Cortisal(µg/dl)	9.4 ± 4.0	11.1 ± 3.8	7.8 ± 2.9	9.6 ± 3.2	12.2	0.00

NS = non Significant
Significance level p £ 0.05
F = one way analysis of variants (ANOVA)

Table 4: Comparison between stunted and non stunted subgroups of obese and non obese children as regardanthropometric parameters

	Obese		Non obese			
	Non-stunted (N=27) Mean ± SD	Stunted (N=13) Mean ± SD	Non Stunted(N= 19) Mean ± SI)	Stunted (N=21) Mean ± SD		FP
Age(year)	10.1 ± 0.7	10.3 ± 0.6	10.0 ± 0.4	10.0 ± 0.5	0.8	NS
Weight (Kg)	59.6 ± 11.6	55.7 ± 5.5	38.1± 3.9	25.6 ± 3.1	104.9	0.00
Height (cm)	122.5 ± 11.0	129.0 ± 4.2	140.1 ± 3.3	121.6 ± 3.3	40.7	0.00
BM I (wt/Ht)^2	9.5 ± 3.8	33.5 ± 3.4	19.4 ± 1.4	17.4 ± 1.6	140.1	0.00
MAC Circumference (cm)	29.6 ± 3.7	28.2 ± 1.9	20.4 ± 1.8	17.4 ± 1.4	117.6	0.00
WaistCircumference(cm)	9.2 ± 8.7	77.8 ± 6.7	58.9 ± 1.5	62.6 ± 3.0	129.6	0.00
Hip Circumference (cm)	96.8± 9.3	89.5 ± 6.5	73.6 ± 2.4	69.8 ± 1.6	96.1	0.00
Waist / Height (cm)	0.9 ± 0.05	0.9 ± 0.03	0.8 ± 0.03	0.8 ± 0.02	20.7	0.00
Biceps/ skinfold (mm)	13.9 ± 4.2	13.4 ± 2.2	6.8 ± 1.2	5.2 ± 1.3	53.1	0.00
Triceps /skin fold (mm)	1.9 ± 5.6	21.5 ± 3.4	12.1± 3.5	8.5 ± 2.1	55.9	0.00

NS = non Significant Significance level p £ 0.05
F = one way analysis of variants (ANOVA)

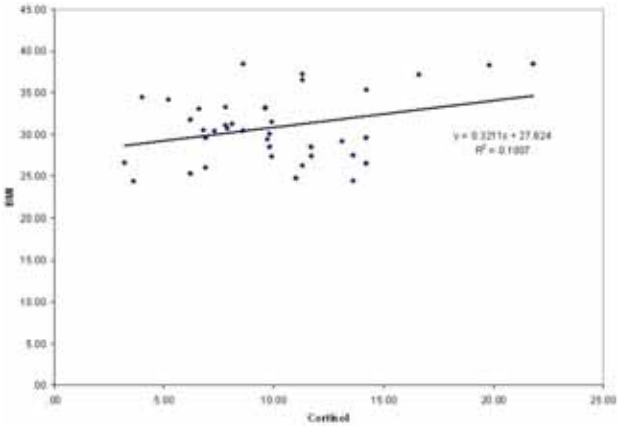

Fig. 1: Simple Correlation between BMI and Cortisol in Obese Group

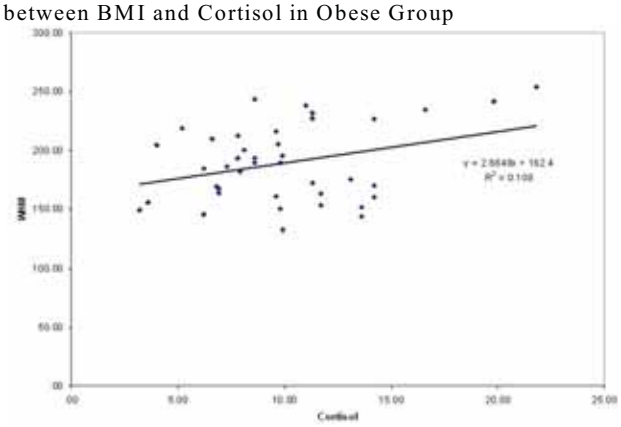

Fig. 2: Simple Correlation between BMI and Cortisol in Obese Group.

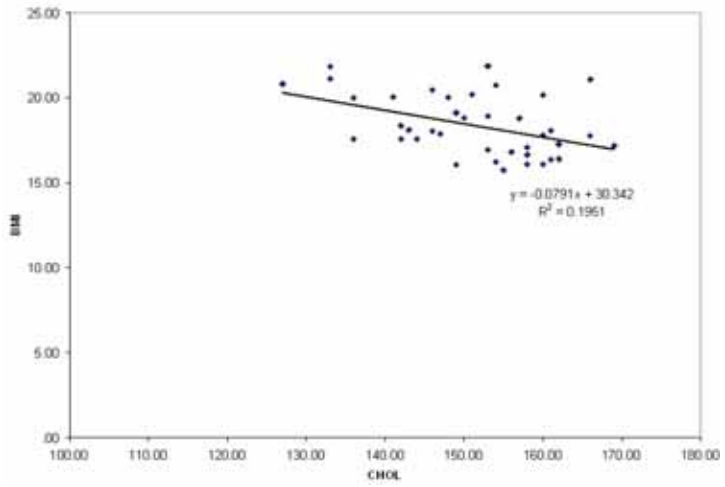

Fig. 3: Simple Correlation between BMI and CHOL in Control Group.

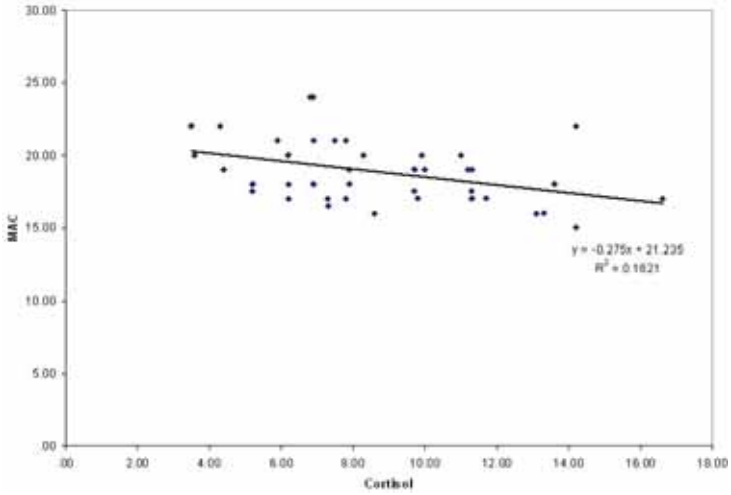

Fig. 4: Simple Correlation between MAC and Cortisol in Control Group.

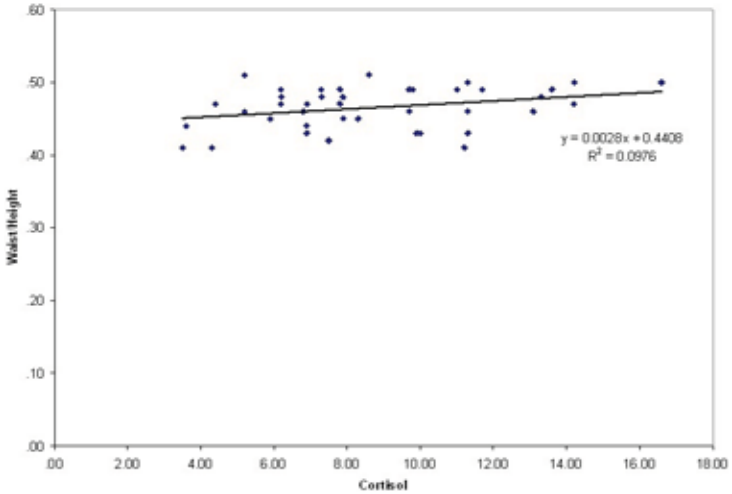

Fig. 5: Simple Correlation between Waist/Height and Cortisoi in Control Group.

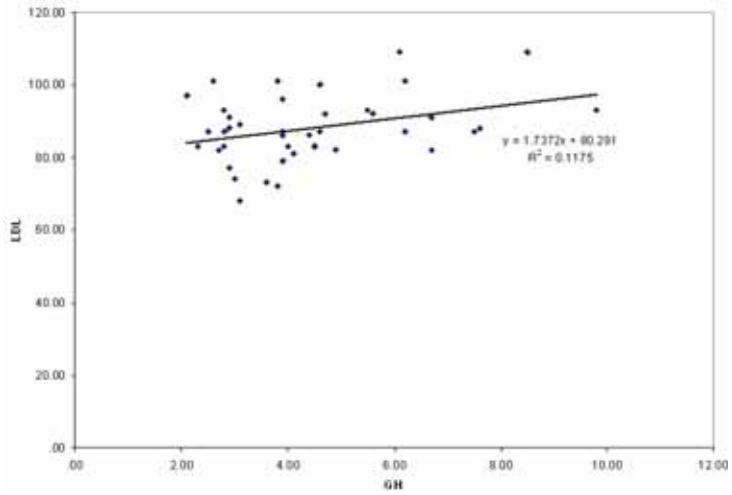

Fig. 6: Simple Correlation between LDL and GH in Obese Group.

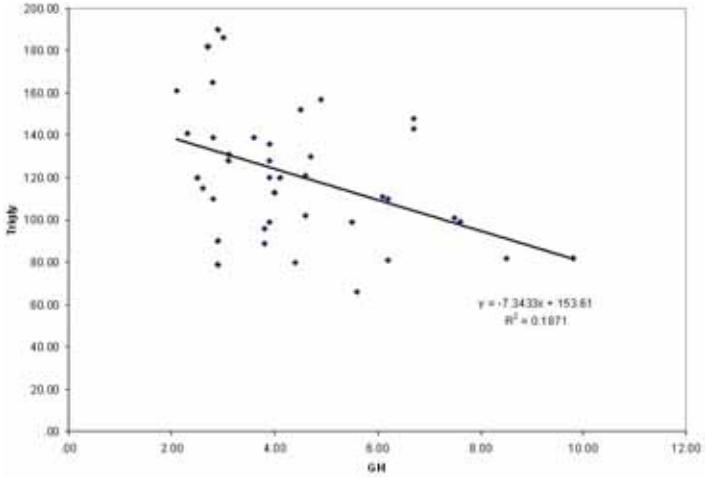

Fig. 7: Simple Correlation between Trigly and GH in Obese Group.

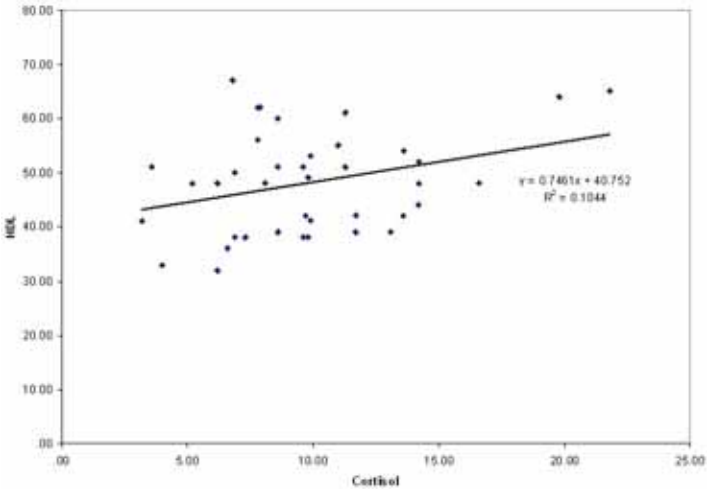

Fig. 8: Simple Correlation between HDL andCortisol in Obese Group.

Dyslipidemia is a condition in which there is an abnormal lipid or lipoprotein in the blood. It is well established that it is determined by both genetic and environmental factors (Everaldo and Joao Guiherme., 2006).

In this study obesity was associated with an adverse lipid profile. We found significantly higher triglyceride and cholesterol levels and lower HDL-C in obese children than non obese children. This unfavorable lipid profile parallels previous observations in other cross- sectional studies in other populations (Garces *et al.,* 2005; De franca and Alves, 2006; Friedland *et al*., 2002). We did not find any significant association between obesity and low-density lipoprotein-cholesterol (LDL -C) levels. Although this associations has been reported in some studies in older children (Friedland *et al*., 2002; Chu *et al*., 1998). Other studies did not observe relations in children at this age (Weiss *et al*., 2004; Graces *et al*., 2005). The changes in the plasma lipid level is recognized as risk marker for coronary atherosclerosis (National cholesterol Education program" NCEP", 2001). Atherosclerotic cardiovascular disease is one of the most serious problems in public health in many countries, because many subjects with lipid disorders remain unidentified, and therefore, continue to have an unfavorable blood lipid profile, increasing the risk for coronary disease (Gotto, 2003). This is a dangerous situation because the atherosclerotic process and obesity related to lipid levels formerly observed only in adults now begin early in childhood (Berenson, 2002; Severing *et al*., 2004; Horri & Vakili, 2006). However , it is interesting to mention that in some longitudinal studies performed in a group of four to ten years old children revealed that the lipid abnormality preceded the development of increased body fat and doubts have raised about which comes first (Tershakovec, *et al*., 2002; Tershakovec *et al*., 2003).

Cholesterol screening during childhood has received increased attention in recent years. The detection of high blood cholesterol during childhood is of potential value in identifying those children who are at increased risk for developing CVD as adults and who might benefit more intensive dietary interventions (Everaldo and Joao Guiherme, 2006).

In the present study cholesterol levels was higher among obese children than non-obese children other studies reported the same results (De franca & Alvez, 2006).

Visceral fat increase is considered one of the most relevant risk factor for CVD, Diabetes and other metabolic conditions. Different methods used to study visceral fat are currently in use, one of the simplest and easy to use and to reproduce is waist circumference (Goran *et al*., 2003).

The present study reported significant correlation between waist circumference and BMI as well as weight for height. There was a significant correlation between waist circumference and cholesterol, Triglyceride, as well as HDL-c levels.

In a study carried out with severely obese children mean age 10.15±2.01 years-abdominal fat index (sum of skinfolds as related to limbs) proved to be positively correlated to total cholesterol (TC) triglycerides (TG), LDL-c, and negatively to HDL-C. Marelli *et al*., 1993 have found significant correlation between waist-hip ratio and TG level. Those results confirm that even in childhood severe obesity is related to lipid changes, and that abdominal adiposity is associated to profile worsening (Roseli *et al*., 2006).

Results from the *Bogalusa Heart study* have also emphasized the relevance of body fat distribution, especially waist circumference. According to the authors that measure may help identify lipid and serum insulin changes.

In this study family characteristics have significant in pact on obesity among children, both father's education and occupation had significant association with obesity , meaning that the highly educated fathers have higher income, provide their family with all needs including snack foods (fat saturated) in combination with more sedentary life. While the opposite was found with the highly educated mothers, with more health backgrounds, the obesity rate increased in association with illiterate mothers (68.5% of children, $p < 0.01$). Maternal occupation also showed relevant effect on obesity, where mothers with professional fops i.e. highly educated had obese children less than mothers with lower jobs this may indicate that higher education among mothers increase their knowledge about obesity hazards, they also seek medical advise for their children more that mothers with lower jobs the same association was reported by others (Popkin BM, 2006; Weker, 2006; Mansour *et al*., 2004). In contract to Ismail (1996) and Baughcum *et al*., (1998), who mentioned that working mothers always overfed their children at have to make it up for then after working hours.

In this study obese children consumed fast food (in the form of high saturated fat, high calories) more than there non-obese peers, and most of them skipped breakfast (67.7%). Those who skip breakfast may eat more later on in the day, also already over weight and obese children may tend to skip breakfast in attempt to control weight. These results agree with Keiss *et al*. (2001) and Mansour *et al*. (2004).

The results of this study showed that obese children tend to be less active than their non-obese peers 64.3% of obese children did not practice sports lessons at school 63.8% did not have any physical activity outside schools, they also tend to go to school via transportations, the same results were reported by Deforceh *et al*. (2003).

CVD is uncommon in young adults, and it has different characteristics from that in older patients. However, children and adolescents with sedentary lifestyles and a high-fat and high-sugar diet are at higher risk for coronary disease (Everaldo and Joao Guiherme, 2006).

Many of the detrimental health effects *of* obesity can be traced to a low-level chronic inflammatory state, the result of adipocyte cytokine production in the body's fat stores.

(Dandona *et al*., 2004) many of these adipose-derived radiators have been related to alterations in catechalamiens, glucocorticoids, insulin and growth hormone. Consequently, understanding of the mechanisms that control body composition is essential to optimally "re-balance" energy intake and expenditure (McMurray & Hackney, 2005). Growth hormone: Insulin like growth factor-I axis, which are key regulators of fat and lean tissue, are remarkably sensitive to brief bouts of physical activity in the form of reduced GH - IG f-I axis. This may explain the need for physical activity and exercise, as part of the treatment regimen for obese children (Eliakim *et al*., 2006). This may explain the reduced physical activity pattern and the significant increase in growth hormone level (P< p.001) among obese children of this study more than non-obese children.

Several investigators (e.g., Kanaley and coworkers (19) speculated that the beneficial effects of exercise in obese subjects might be limited due to the suppressed GH response to exercise. However, it is now well established that many of the health effects of exercise training are mediated by IGF-1 and are GH independent (Eliakim *et al*., 2006).

Cortisol is the most important glucocorticoid involved in obesity, it is also a key hormone involved in the body's response to stress, both physical and emotional. However cortisol has a twofold effect on fat metabolism. When human is facing stress, cortisol increases lypolysis to supply the body with rapid source of energy, on the other side corticotropine releasing hormone (CRH) will be secreted via hypothalamus which will lead to suppression of appetite and decreased digestive system function. CRH will also triggers the release of cortisol to help mobilize carbohydrate and fat for quick energy. Cortisol level will increase and will help to bring the body back to homeostasis through increasing appetite, and increased tendency to consumption of high carbohydrate and fat which may lead to weight gain (Salehi *et al*., 2005; Jessop *et al*., 2001).

This finding is consistent with the present study; cortisol level was significantly higher among obese children than non-obese children ($p< 0.02$).

Our study also showed that cortisol level is significantly correlated with Body Mass index, weight to height ratio, waist to height ratio, waist circumference as waist to height ratio. Body mass index is a common indicator of obesity which is used in epidemiological studies, but it does not give an idea about fat distribution (Hoffman *et al*., 2008).

Abdominal obesity is a well know risks factor cardiovascular Disease risk and Diabetes mellitus it is due to the development of visceral adipose tissue, which leads to metabolic disorders (Barat *et al*., 2008). Waist to height ratio as well as waist circumference are recommended as a mean of identifying people at risk of morbidity associated with central adiposity. Waist to height ratio > 0.5 is considered at risk CVP risk it does not require sex-and age specific percentiles (Ganett *et al*., 2008).

In our study waist /height ratio among obese children is 0.6 ± 0.04 versus 0.04 ± 0.02 among non-obese children.

Our results showed that cortisol level is positively correlated with waist /height ratio among obese children ($r = 0.39$, $p < 0.01$) this finding is consistent with Barat *et al*. (2007) who suggested that hypothalamic-pituitary adrenal axis could be involved early in life.

Cortisol directly effects fat storage and weight gain in stressed individuals. Tissue cortisol concentrations are controlled by a specific enzyme 11 – hydroxyl steroid dehydrogenase that converts inactive cortisone to active cortisol. This particular enzyme is located in adipose tissues. Studies with human visceral and subcutaneous fat tissue have demonstrated that the gene for this enzyme is expressed more by obese conditions. It has also been demonstrated in research that human visceral fat cells have more of these enzymes compared to subcutaneous fat cells. Thus, higher levels of these enzymes in these deep fat cells surrounding the abdomen may lead to obesity due to greater amounts of cortisol being produced at the tissue level. As well, deep abdominal fat has greater blood flow and four times more cortsiol receptors compared to subcutaneous fat. This may also increase cortisol's fat accumulating and fat cell size enlarging effect. (Morris and MB Zemel, 2005).

In this study obese-stunted children as well as non-obese stunted children had higher cortisol level than did non stunted children whether obese or not obese ($F= 12.2$, $p <0.001$ table 16). Similar finding were previously shown by Fernald & Granthan Mc. Gregor (1998) while studying non-obese school age children. Sawaya *et al*., 1998 also found the same results stunted obese Brazilian children. Hoffman *et al*. (2000) found association between excess weight gain and dietary fat content in stunted children, but not in non stunted control children. This is suggestive of au increase in the efficiency of dietary fat utilization that could lead to increased body fat content over time.

Low height for age or stunting is an characteristic finding among children in underdeveloped countries and among some children from underprivileged countries short stature may follow chronic caloric deficits which may, in turn, reflect repeated illness, household food shortages, and parental misperceptions of children's dietary needs, short stature or stunting is paradoxically accompanied by increased weight for height, this pattern had been demonstrated in Egypt, Latin American, and Hispanic American children (De Assis, 2007).

It is more common among low income children.

Co-existence between malnutrition and obesity are associated with important metabolic changes in the form to higher fat diets, lower fat oxidation, and higher body fat gain (Fanjiang & Kleinman, 2007).

In the present study stunted obese children had higher central fat in the form of higher waist/hip ratio than in obese non-stunted children, the same was applied for stunted non obese versus non-stunted non-obese children ($F= 12.9$, $p< 0.000$, table: 17).

Elevated levels of the low density lipoprotein cholesterol (LDL-C) and decreased amount of the high density lipoprotein cholesterol (IIDL-C) have been long recognized as important risk factors for CVD, many studies demonstrated a relation between the rate of prenatal and postnatal growth and obesity on chronic disease in later life (Barker *et al*., 2005, Power *et al*., 2006).

Our study demonstrated significant altered lipid profile among stunted groups of children whether obese or non-obese in the form of (↑LDL-C) and ↓HDL-C than did the non-stunted groups (both obese & non-obese) which may be due to altered hypothalamic pituitary adrenal (HPA) axis early in life (Phillips *et al*., 2000; Power *et al*., 2006).

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