Biochemical Profile of Some Nutritionally Stunted Egyptian Children

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ABSTRACT

Childhood nutritional stunting, an indicator of chronic malnutrition, has been suggested as one factor that can contribute to high incidences of obesity, and impaired lipid and glucose metabolism in developing countries. This study aimed to determine health risks of developing hyperlipidemia, obesity, and diabetes as non-communicable diseases (NCDs) among nutritionally stunted Egyptian children. A total of 90 children were enrolled; 60 nutritional stunted children and 30 non- stunted of matched age and sex. Clinical, demographic characteristics were determined for the studied children. Lipid profile levels, fasting plasma levels of glucose, and insulin were measured in these subjects. Pancreatic beta cells' function (HOMA-β) and insulin resistance (HOMA-IR) were calculated by homeostasis model assessment (HOMA). Stunted children exhibited significantly lower values for weight, weight-for-age Z score (WAZ), height, and height-for-age Z score (HAZ) as compared to normal ones. Significant elevation in total cholesterol (TC), and low density lipoprotein cholesterol (LDL-c), significant decrease in high density lipoprotein cholesterol (HDL-c) and HDL/LDL ratio were detected compared to that of the healthy control subjects. There were no significant differences in the levels of fasting glucose, fasting insulin, homeostasis model assessment of beta-cell function (HOMA-β), and homeostasis model assessment of insulin resistance (HOMA-IR) between the two compared groups. ***Conclusion:*** *Alterations in lipid pattern among nutritionally stunted Egyptian children may predict the future of NCDs later in life.*

Key words: *Children; Stunting; lipid profile; Insulin resistance; Nutrition.*

INTRODUCTION

Stunting is an important public health problem in developing countries. It is the primary chronic malnutrition that often occurs in utero and/or during the first two years of life **(Hoffman. 2014).** Nutritional deprivation during critical periods of development could cause long-lasting changes that lead to obesity and associated co-morbidities in adulthood **(Rodriguez *et al.*, 2015).** In 2010, it is estimated that 167 million children in developing countries were stunted **(De-Onis *et al.*, 2012).** World Health Assembly targets aim to reduce stunting by 40% between 2010 and 2025 (Prendergast and Humphrey. 2014).

Several epidemiological studies explain the association between growth retardation and alternation in lipid metabolism; they reported that adults who experience intrauterine growth retardation are more likely to suffer from atherogenic lipid profiles and cardiovascular diseases than those who developed normally **(Tanner *et al.*, 2014; Alves *et al.*, 2014).** Moreover, **Hoffman *et al* (2012)** reported that 3 to 4 year-old stunted children from a cohort study of maternal nutrition education had significantly higher TC concentrations compared to children who were not stunted.

Alongside, the metabolism of glucose and insulin also changes in those who suffered nutritional deprivation in early life. Where, physiological and metabolic mechanisms are not fully matured at birth. If the malnutrition also continues during this period, it can induce alterations in glucose metabolism generating a predisposition for diabetes **(Flanagan *et al.*, 2000).** In this context, **Gonz´alez-Barranco *et al* (2003)** submitted a group of young adults, each of whom had a history of malnutrition in the first year of life, to the oral glucose tolerance test and detected elevated levels of glucose and insulin in comparison with the control group, independent of birth weight, body mass index (BMI), or age. **Martins and Sawaya (2006)** reported that the function

of pancreatic beta cells was diminished while insulin sensitivity was increased among nutritionally stunted children.

Moreover, stunted individuals exhibited a greater susceptibility to decreased total energy expenditure that considered to be a further factor in the predisposition to weight gain, accumulate abdominal fat, elevated respiratory quotients (indicating high carbohydrate oxidation), and reduced lipid oxidation rates **(Hoffman *et al.*, 2000). Florêncio *et a*l (2007)** reported that short stature represented a risk factor for alterations in lipid profile, insulin resistance, and abdominal obesity in adult women from slums in Maceió, north-eastern Brazil. All of these studies reinforce the hypothesis that stunting alters the regulation of physiological mechanisms that are responsible for energy conservation and fat accumulation, resulting in obesity in adult life with high risk of NCDs.

In view of that, the present study aimed to shed more light on the lipid pattern, the level of glucose and insulin as well as pancreatic beta cells' function (HOMA-β) and insulin resistance (HOMA-IR) status in some nutritionally stunted Egyptian children in order to determine their cooperative role in the development of NCDs in the adulthood.

SUBJECTS & METHODS

Subjects

A total of 90 children 60 stunted (32 girls, 28boys) and 30 non-stunted (15 girls, 15 boys), age ranged from 5 to 10 years, were enrolled in this study. Stunted children were visitors of the outpatient clinic of National Nutrition Institute (NNI), Cairo, Egypt. Children with chronic diseases, endocrine or metabolic disorders, those taking any medications that alter growth or hormones, or having parents' height lower than 150 cm were excluded. Written informed consent was obtained from the parents of children and the study was approved by the Ethics Committee of the National Organization for Teaching Hospitals and Institutes, Cairo, Egypt.

All children were subjected to full history including age, sex, parental heights and family history. Clinical examination, routine investigation including complete blood picture, weight and height were measured, followed by the calculation of body mass index (BMI) using the formula BMI= weight (Kg) / height^2 (meter). Height-for-age, weight-for-age and BMI for age z-scores were calculated using Anthro plus program depending on WHO cutoffs (2007). According to the WHO reference for children 5-19 years, those having Height-for-age more than 2 standard deviation score (SDS) below the median of the WHO child growth standards were considered stunted.

Preparation of samples and biochemical analyses

Blood samples were drawn by venipuncture in the morning after an overnight fast. Serum was separated by centrifugation. Serum total cholesterol (TC) and high density lipoprotein cholesterol (HDL-c) levels were determined calorimetrically according to the method of **Allain *et al* (1974) and Grove (1979)** respectively, using kits supplied by Bio-Systems (Spain). Serum triglycerides (TG) levels were estimated by enzymatic colorimetric method according **to Fossati and Prencipe (1982)** using Chemelex kit (Spain). Low density lipoprotein cholesterol (LDL-c) concentration was calculated using formula by **Friedewald *et al* (1972)**: LDL-c= TC – (HDL- c + 1/5×TG). Very low density lipoprotein cholesterol (VLDL-C) was calculated from the following formula: TG/5 =VLDL-C according to **Warnick *et al.* (1990).** Parameters of lipid profile were expressed as mg/dl. Serum fasting blood glucose was determined using Stanbio Laboratory kit (New York, USA), and was expressed as mmol/L. Insulin was determined by the ELISA kit provided by DRG International Inc (USA).

The function of pancreatic beta-cells (HOMA-B) and insulin resistance (HOMA-IR) were evaluated by the homeostasis model assessment

(HOMA) according to equation by **Matthews et al (1985):**

HOMA-IR = fasting insulin (µIU/mL) × fasting glucose (mmol/L)/22.5

HOMA-B= [20 × fasting insulin (µIU/mL)]/ [fasting glucose (mmol/L) − 3.5]

Statistical analysis

The data were analyzed using version 16.0 of the computer based statistical package of Statistical Product and Service Solutions (SPSS) 2007. All the data are expressed as Mean± standard error of mean (SDE) and the range is stated between parentheses. To evaluate the differences between stunted and control children, independent samples t test and Mann–Whitney test were performed, respectively, in continuous variables with normal distribution and without normal distribution. Correlations were carried out by the bivariate correlation using the spearman correlation coefficient.

RESULTS

Data in table 1 showed that stunted children exhibited significantly lower values for weight, weight-for-age Z score (WAZ), height, and height-for-age Z score (HAZ) as compared to normal ones. Significant elevations in serum TC and LDL-c levels along with significant reduction in HDL-c levels were also detected in stunted children as compared to control values. These changes resulted in significant decrease in the HDL/LDL ratio of stunted children relative to normal values. Also, Stunted children have a tendency to have a higher TG (p= 0.179) and VLDL-C (p= 0.197) levels however, not significantly changed Table (2). Table (3) showed that there were no significant differences in the levels of fasting glucose, Insulin, HOMA-β, and HOMA-IR between the two compared groups. Furthermore, Table (4) showed that there were significant positive correlations between HDL-C levels and weight (r=0.231, p=0.030), height (r=0.290, p=0.006), HAZ

(r=0.233, p=0.029), among stunted children.

DISCUSSION

The intrauterine or early life malnutrition occurred in fetus can lead to dyslipidemia and metabolic disorders in childhood; these changes considered a major factor for the occurrence of NCDs in the long term **(Alves *et al.*, 2014).** The present study aimed to investigate the lipid pattern as well as glucose and insulin levels to calculate HOMA-β and HOMA-IR in some nutritionally stunted Egyptian children in order to determine their cooperative role in the development of NCDs in the adulthood.

The present study showed marked decrease in weight, weight-for-age Z score (WAZ), height, and height-for-age Z score (HAZ) of stunted children versus healthy one. Similar findings have been previously reported by **Martins and Sawaya (2006).** This may be the result of parasitic infections, deficiencies in macronutrients intake (Protein, carbohydrates and fats) and micronutrients (Vitamins and minerals) **(Katona and Katona-Apte. 2008**). On other hand, the difference in the mean of BMI between the two studied groups was not statistically significant. **Walker *et al* (2007)** suggested that there has been some catch-up in adiposity among the stunted subject in adulthood, in spite of the thinner in early childhood and this may support the current results.

Intrauterine malnutrition can change the lipidemic profile to become more atherogenic **(Godfrey and Barker. 2000).** In the present study stunting was found to cause dyslipidemia as evidenced by the significant elevations in TC and LDL-c along with the significant decrease in HDL-c levels detected in studied stunted children. The possible explanation for these findings, it is presumed that malnourished children tend to have high levels of plasma growth hormone (GH) and low levels of insulin-like

growth factor-1 (IGF-1) and that may be the major cause of reduced growth. Thus, in order to maintain the functioning of vital organs, metabolic adaptations occur, such as increased serum levels of cortisol, which promotes protein catabolism and increased respiratory quotient. These factors lead to a lower lipid oxidation, and consequent accumulation of fat in the liver **(Martins and Sawaya 2006).** Growth retardation occurred in the last of pregnancy can lead to permanent changes in liver structure and lipid metabolism, either by overproduction of VLDL-C and LDL-C and/or defects in the expression of the enzyme lipoprotein lipase (LPL**) (Yajnik 2000).**

The present study agree with **Lumey *et al* (2009) and Hoffman *et al* (2012)** who found that stunting appears to have permanent effects on cholesterol metabolism, resulting in decrease in the number and activity of active hepatic receptors, which regulate cholesterol synthesis. These changes leading to elevated the level of TC, LDL-c. Also, **Cong *et al* (2012)** found that malnutrition during the gestational period cause's changes in liver structure and active transcription genes for 3-hydroxy-3-methylglutaryl-CoA reductase (HMGCR) that resulting in an over enhanced basal cholesterol synthesis. Moreover, **Martins *et al* (2011)** assumed that during growth, the malnourished children had decrease in the circulating levels of free tri-iodothyronine (T3) due to the decrease in carrier proteins (albumin and prealbumin), in addition to the decrease in peripheral activity of the enzyme that converts thyroxine (T4) into T3 (5-deiodinase), these situation favors gluconeogenesis and release of fatty acids from adipose tissue and inhibits the actions of GH dependent on somatomedin-C (IGF-1). This condition may contribute to elevated TC and LDL-C levels observed in malnourished children.

HDL plays an important role in mechanism against atherosclerosis. In addition to being a component of the reverse cholesterol transport system, HDL serves both as an antioxidant, reducing oxidized LDL and decreases the expression of adhesion molecules by vascular endothelial cells induced by cytokines **(Cao *et al.*, 2015). Esteve *et al* (2005)** suggested that the changes in lipid pattern occurring due to inflammation weren't only associated with the diminution HDL-C levels, but also alerted in HDL composition and metabolism to become proinflamatory particle. The altered HDLs enhance LDL oxidation and attracting monocytes to engulf the oxidized LDLs **(Bindu *et al.*, 2010).** All these changes may lead to hypercatabolism that characterized by loss of muscles (with or without loss of fat mass) and decrease in lean body mass in malnourished children **(Mehta *et al.*, 2013).**

In the current study, dyslipidaemia was characterized by the significant low HDL-C levels together with non significant hypertriglyceridemia. In this context two main hypotheses have been put forward, firstly: (i) the reduced activity of lipoprotein lipase may hamper the maturation of HDL particles and lower clearance of circulating TG. Secondly (ii) the increased activity of the protein that promotes the transfer of cholesteryl esters from HDL to triglyceride-rich lipoproteins may reduce the levels of HDL particles **(Xiao *et al.*, 2008).** The present study agree with **Hoffman *et al* (2012) and Alves *et al* (2014)** who found that stunted children had dyslipidemia characterized by lower HDL cholesterol; they explain that the lower HDL was due to the rapid catabolism of HDL-C, but not to the decreased production of its particles.

The poor maternal nutrition or poor delivery of nutrients to the fetus due to other causes (eg, placental dysfunction) contributing to beta cell abnormalities and diabetes later in life **(Reusens *et al.*, 2011).** The present study showed

that there were no significant differences in the levels of fasting glucose, fasting insulin, HOMA-β, and HOMA-IR between the two compared groups. The first explanation to the observed results may be attributed to the fact that calcium is necessary for the release of insulin from the pancreas and for the uptake of glucose by the muscles; at the same time extending the length of a bone requires an enormous amount of calcium. Thus, by keeping the bones short, the calcium that would have gone into bone growth can be diverted to assure an adequate supply of insulin and an efficient glucose uptake mechanism in the muscle and fat cells **(Soriaac *et al.,* 2010)**. The other explanation for the observed non significant changes in the fasting glucose, insulin, HOMA-β, and HOMA-IR between the two compared groups may be related to the body composition that altered with age in stunted children as suggested by **Trowbridge *et al* (1987). Clemente et al (2009)** reported that stunted children did not show any tendency towards overweight at age 11–12 years, and the obesity becomes more common among female than males at age older than 12 years. In (2003) Steinberger and Daniels concluded that insulin resistance varied directly with the degree of adiposity where it was significantly related to an abnormal lipid profile in heavy children but not in thin children. **Florencio *et al* (2007)** reported that short stature in childhood represented a risk factor for insulin resistance, and abdominal obesity in adolescents. Moreover, **Clemente *et al* (2014)** showed the significant higher levels of, glucose, insulin, diminished function of beta cells, and increased insulin resistance among overweight adolescents with mild stunting and attributed these results to a higher number of insulin peripheral receptors, especially in the adipose and muscle tissues.

Fat distribution in thinners stunted children increased risk of obesity, metabolic and endocrine disorders, hypertension or cardiovascular diseases in

adolescence **(Benefice *et al.*, 2001).** The association between stunting and later overweight thus appears to be inconsistent and may depend on environmental factors such as the change in conditions sufficient to produce a shift from dietary deficit to excess **(Martorell *et al.*, 2001).** Furthermore, in the present study there were significant positive correlations between HDL-C levels and anthropometric parameter indices including weight, height, HAZ among stunted subjects. Similar findings were previously reported by **Veiga *et al* (2010) and Alves *et al* (2014)** since they demonstrated that the levels of HDL-c were inversely related to malnutrition severity.

CONCLUSION

Nutritionally stunted children exhibited alterations in lipid pattern that may predict the future of NCDs later in life.

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Table 1): The demographic and anthropometric characteristics for healthy control and stunted children

Parameters	Control (N=30)	Stunted children (N=60)
	Mean ± SE	Mean ± SE
Age (years)	7±0.3	**6.9±0.2**
Sex (M/F)	15/15	**28/32**
Weight (kg)	21.4±0.9	**17.4±0.4 ***
Weight-for-age Z score (WAZ)	-0.6±0.1	**-1±0.1 ***
Height (cm)	116.5±1.9	**104.5±1.2 ***
Height-for-age Z score(HAZ)	-0.9±0.2	**-3±0.1 ***
Body mass index (BMI)(kg/m^2)	15.5±0.2	**15.5±0.2**
BMI-for-age Z score(BMIAZ)	**-0.1±0.1**	**-0.1±0.1**

Data are expressed as mean ±SEM.
N: the number of subjects in each group
**: Significant difference from control at P < 0.05.*

Table 2): Lipid profile in healthy control and stunted children

Parameters	**Normal value ****	**Control (N=30)**	**Stunted children (N=60)**
		Mean ± SEM	**Mean ± SEM**
Total cholesterol (TC) (mg/dl)	<170 mg/dl	144.5±3.8	**160.6 ±3.6 ***
Triglycerides (TG) (mg/dl)	<150 mg/dl.	66.4±2.6	**73.1 ±3.1**
High density lipoprotein cholesterol (HDL-c) (mg/dl)	≥35 mg/dl	47.4±1.5	**42.4±1 ***
Low density lipoprotein cholesterol (LDL-c) (mg/dl)	<110 mg/dl	83.8±3.2	**103.6±3.5 ***
Very low density lipoprotein cholesterol (VLDL-c) (mg/dl)	2-30 mg/ dl	13.3±0.5	**15±1**
HDL-C/LDL-C ratio		**0.6±0.3**	**0.4±0.2 ***

Data are expressed as mean ±SEM.
**: Significant difference from control at P < 0.05.*
***: NCEP, (1992).*

Table 3): Fasting glucose, insulin, homeostasis model assessment of beta-cell function (HOMA-β), and homeostasis model assessment of insulin resistance (HOMA-IR) in healthy control and stunted children

Parameters	**Normal value**	**Control (N=30)**	**Stunted children (N=60)**
		Mean ± SEM	**Mean ± SEM**
Fasting glucose (mmol/L)	3.9-5.5 mmol/l	5 ±0.1	**5 ±0.1**
Fasting insulin (µIU/ml)	1-8 µIU/mL	6.5 ±0.4	**6.4 ±0.4**
HOMA-β		96 ±9.3	**100 ±7**
HOMA-IR		**1.4±0.1**	**1.4 ±0.1**

Data are expressed as mean ±SEM.

Table 4): Correlation between high density lipoprotein cholesterol (HDL-c) and anthropometric parameters in stunted children

parameters	*Correlation coefficient (r=)*	*P-value*	*Significance*
Age (years)	0.172	0.109	***Non significance***
Weight (kg)	0.231	0.030	***Significance***
Weight-for-age Z score (WAZ)	0.162	0.133	***Non significance***
Height (cm)	0.290	0.006	***Significance***
Height-for-age Z score(HAZ)	0.233	0.029	***Significance***
Body mass index (BMI)(kg/m^2)	0.021	0.847	***Non significance***
BMI-for-age Z score(BMIAZ)	**0.028**	**0.794**	***Non significance***

الصوره البيوكيميائيه لدي الأطفال المصريين المصابين بالتقزم الناتج عن سوء التغذيه

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٢- جامعه حلوان- كليه العلوم- قسم الكيمياء- القاهره- مصر.

الملخص العربي

يعد قصر القامة لدى الاطفال من المشاكل الصحية الهامة في البلدان النامية . حيث يعتبر من الامراض الناتجة عن سوء التغذية. كما يعتبر من احد العوامل التي يمكن ان تسهم في اضطراب ايض الدهون والسكر مما قد يؤدي الى حدوث السمنة . تهدف هذه الدراسة الى تقييم المخاطر الصحية والامراض غير المعدية كا ارتفاع مستوى الدهون والسمنة ومرض السكري بين بعض الاطفال المصريين المصابون بقصر القامة الناتج من سوء التغذية . شارك بهذه الدراسة ٩٠ طفلا منهم ٦٠ طفل يعانون من قصر القامة الناتج عن سوء التغذية و ٣٠ طفل طبيعيين ذات اطوال مناسبة " المجموعة الضابطة" من نفس الفئة العمرية ، من الزوار المنتظمين بعيادة الخاصة للمعهد القومي للتغذية بالقاهرة –مصر . تم تحديد المقايس الجسمية والخصائص اليموغرافية لجميع الاطفال المشاركين بالبحث . كما تم اجراء التحاليل المعملية في عينات مصل الدم بعد صيام (١٠-١٢ ساعة) لتحديد مستوى الدهون والجلوكوز والانسولين بالدم وحساب وظيفة خلايا بيتا بالبنكرياس ومقاومة الانسولين. اوضحت النتائج عن انخفاض معنوي في الوزن و الوزن بالنسبة للعمر (WAZ) وكذلك الطول ودرجة الطول بالنسبة للعمر (HAZ) لدى الاطفال الذين يعانون من قصر القامة بالمقارنة مع ذويهم الطبيعيين . اما تاثير قصر القامة على مستوي الدهون اوضحت النتائج ارتفاع مستوى كل من (الكوليسترول الكلي – والكوليسترول منخفض الكثافة) مع انخفاض مستوى الكوليسترول عالى الكثافة لدى الاطفال المصابيين بقصر القامة مقارنة بالاطفال الاصحاء. كما اظهرت الدراسة عدم وجود اختلاف معنوي في مستوى السكر والانسولين و حساب وظائف خلايا البنكرياس بيتا ومقاومة الانسولين بين اطفال المجموعتين. من هذة النتائج يستنتج ان اي تغيير يطرأ على مستوى دهون الدم لدى الاطفال المصابون بقصر القامة الناتج عن سوء التغذية قد يكون مؤشر بينيء بالاصابة بالامراض الغير معدية بالمستقبل.

الكلمات المفتاحية: *التحاليل البيوكيميائية – قصر القامة الناتج عن سوء التغذية*