# Glucose Intolerance in Obese Egyptian Adolescents

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**Abstract:** Impaired glucose tolerance (IGT) and Type2 diabetes mellitus (T2DM) in children and adolescents is an important Public Health problem which runs in parallel with childhood obesity. The rates of (IGT) and (T2DM) in youth are increasing recently. The aim of the present study is to assess glucose tolerance and metabolic syndrome in obese adolescent and determine the factors associated with it. The study included (88) adolescents divided into two groups: A. 60 Obese adolescents and B. 28 non-obese adolescents**.** All adolescents are subjected to the following: Clinical assessment for: congenital or acquired illness, anthropometric measures: (height, weight, BMI, W/C and waist/hip ratio), blood pressure in the studied subjects, pubertal state by tanner classification**,** family history of diabetes and/ or hypertension, FBS, PPBS, $HbA_{1C}$, lipid profile, fasting serum insulin levels to assess insulin resistance by HOMA equation. The main findings of the present study are that (IGT) and other components of metabolic syndrome are present in obese group and not in normal BMI group. Overweight and obesity in childhood and adolescence tend to persist into young adulthood with their long-term effects on mortality and morbidity. Insulin resistance is the earliest component of metabolic syndrome occurring as a consequence for obesity in adolescence predisposing to other components of metabolic syndrome and ending lastly into IGT and T2DM.
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**Keywords:** glucose intolerance, type 2 diabetes, obese adolescents

## 1. Introduction

The prevalence of obesity in adolescence has more than doubled in the last 15 years in many regions of the world **(Monzavi *et al.,* 2008)**. This phenomenon is associated with rapidly increasing cases of type 2 diabetes in childhood. obesity alone increases the risk of hypertension, cholecystits, and psychological symptoms in obese children **(Torok *et al.,* 2001).**Obesity has been associated with increased plasma levels of insulin, this denotes insulin resistance that resulting in diminished ability of insulin to stimulate glucose uptake by the skeletal muscles and adipose tissue, in addition to reducing insulin's ability to suppress hepatic glucose Production and output **(Weiss *et al.,* 2004)**

The most appropriate definition for metabolic syndrome is the one proposed by the international diabetic federation (IDF). It divided children into age groups. There was not a well defined proposal for children under 6 years of age, due to the lack of data. In this definition, for a matter of convenience, the cut-offs were fixed for pressure, lipids, glycemia, and abdominal circumference points were assessed by percentile. In children aged 6-10, the cut-offs of metabolic and blood pressure variables were not well defined, assessing simply adiposity (considering abdominal circumference over the $90^{th}$ percentile). The same criteria would be used for children aged 10-16; regarding glycemic metabolism, fasting glycemia ≥100 mg/dL, triglycerides ≥150 mg/dL, HDL cholesterol below 40 mg/dL or using a lipid lowering drugs, and blood pressure limits ≥130 systolic or ≥85 mmHg diastolic or using a antihypertensive drugs. If the patient had altered abdominal circumference and two more factors, the metabolic syndrome diagnosis would be established. The difference is that, for adolescents over 16 years of age, there is a differentiation between HDL ≤40 for men and ≤50 for women **(Zimmet *et al.,* 2007)**.

IDF international definition, with minor modification mainly related to the WC cut points was used to classify Met. S for adolescents aged 10–16, central obesity (WC≥$90^{th}$ percentile) by age and gender according to the Chinese reference for children and adolescents, and the presence of any two of the following four factors, ie; elevated BP (systolic ≥130/diastolic ≥85 mmHg), low HDL-C (<40 mg/dl), elevated TG (≥150 mg/dl ), IFG (fasting plasma glucose ≥100 mg/dl); ii) for those aged 16 years or older: the IDF criteria for adults were used, namely central obesity ( WC≥90 cm for Chinese men and ≥80 cm for Chinese women) plus any two of the following conditions: elevated BP (systolic ≥130/diastolic ≥85 mmHg), low HDL-C (<40 mg/dl in males and <50 mg/dl in females), elevated TG

(≥150 mg/dl), and IFG (fasting plasma glucose≥100 mg/dl).**( Ma *et al.*, 2010).**

**Aim of the work:** The aim of the present study is to assess glucose tolerance and metabolic syndrome in obese adolescent and determines the factors associated with it.

**2. Patients& Methods**

This study was carried out on 88 Egyptian adolescents (age ranged from 16-18 years) 60 of them are obese (BMI >25), 32 females, 28 males and 28 non-obese (BMI <25), 16 females and 12 males. All of them are collected from El- Hussein Hospital, Al-Azhar University between December 2012 and June 2013. Patients with secondary obesity and the use of drugs that alters blood pressure, glucose or lipid metabolism were excluded from the study.

Obese group was divided into: (group 2); 36 obese adolescents with one or two (less than three) = Obese adolescents without metabolic syndrome (20 males, 16 females), (group 3); 15 obese adolescents with metabolic syndrome (three or more criteria) without glucose intolerance (4 males, 11 females) and (group 4); 9 obese adolescents with metabolic syndrome and impaired glucose tolerance (4 males, 5 females).

**All subjects were subjected to: Full history:** including personal history (name, age, sex, residence.....etc)**,** family history (gestational diabetes, diabetes, hypertension, obesity.....etc). Assessment of pubertal state, measurements of blood pressure and all Anthropometric measures were performed twice:

**Waist circumference** was measured at the narrowest area above the umbilicus or mid way between the coastal margin and the iliac crest, in a horizontal plane at the end of normal expiration, with the tape measure snugly fitted. WC of (94+) cm in males, or (80+) cm in females used as cut-off values to identify adolescents with abdominal obesity if above age of 16 according to IDF definition of metabolic syndrome. **Hip circumference** was measured at the maximal gluteal protrusion or at the most prominent area of the buttocks at the level of symphysis pubis in a horizontal plane. The tape measure was held snugly against the body but without compression. **Waist hip ratio (WHR)** was calculated by dividing waist by hip circumference, and abdominal obesity was diagnosed when the WHR was >0.80 in girls and 0.95 in boys. **(Zimmet *et al.*, 2007)**

**Height** was measured without shoes to the nearest 0.1 cm using a tape meter. **Weight** was measured in light clothing to the nearest 0.1 kg using a digital scale. **BMI** was calculated as the ratio of weight (kg) to height (m) squared (kg/$m^2$).

**Laboratory investigations:** Venous blood was sampled for the measurement of fasting plasma concentrations of **glucose**,**2 hrs post prandial blood glucose** and serum concentrations of total cholesterol, **HDL**-cholesterol, **triglycerides** were measured by colorimetric method, and HbA1c was measured for each case before centrifugation of sample by immunoassays. **Fasting serum insulin** was measured by microparticle enzyme immunoassay kit to assess insulin resistance by HOMA IR**. HOMA IR**: is an equation for measurement of insulin resistance: Fasting Glucose (mg/dl) x fasting Insulin (µU/mL) / 405.Lower HOMA index values (<4) indicated higher insulin sensitivity, whereas higher values (>4) indicated lower insulin sensitivity **(55%)**

**Metabolic Syndrome** was defined by these criteria: Central **obesity** (defined as waist circumference ≥ 94cm for men and ≥ 80cm for women), Plus any two of the following four factors:

- **Raised triglycerides** :( ≥150 mg/dl)
- **Reduced HDL-cholesterol**: (<40 mg/dL) in males and (<50 mg/dL) in females, or specific treatment for these lipid abnormalities
- **Raised blood pressure**: systolic BP ≥130 or diastolic BP ≥85mm Hg, or treatment of previously diagnosed hypertension
- **Impaired fasting glycemia** (IFG): fasting plasma glucose (FPG) (≥100 mg/dL), or previously diagnosed type 2 diabetes, 2hours Post prandial glycemia: 2hr PPG (≥140mg/dL), or previously diagnosed type 2 diabetes (**Zimmet *et al.*, 2007).**

**Statistical analysis** Statistical analyses of the result were performed using an $x^2$test (associated to Yates's correction when necessary); data are expressed as M ± SD, and Student's t test for unpaired data. *P*-value ≤ 0.05 and r-value ≥ 0.50 were considered significant**.** Logistic regression analyses were performed to evaluate the association between metabolic syndrome and other parameters.

**3. Results**

In the present study the following parameters (weight, BMI, W/C, H/C, H/C ratio, SPB, DPB, FBS, PPBS, HbA1c, TG, fasting insulin and insulin resistance) were significantly higher, while serum levels of HDL were significantly lower in obese versus non-obese subjects, but no significant changes between them as regards age, sex and height (Table 1).

In our study the constituent factors for Metabolic Syndrome were as follows, one factor was present in (19) adolescents with a prevalence of 31.7% (group I), two factors in (17) adolescents with a prevalence of 28.3%, while (24) adolescents 40% of the sample of the study were having three or more criteria of metabolic syndrome and all adolescence with normal pubertal state. Of the single components of the metabolic syndrome, **dyslipidemia** was the most frequent, decreased serum HDL was reported in 31 of 60 obese adolescents (51.6%), also increased serum TG was reported in 31 of 60 obese adolescents (51.6%). The prevalence of **hypertension** was 15 of 60 obese adolescents (25%). Total **impaired glucose tolerance** prevalence rate was 9 of 60 obese adolescents (15%). No cases of type 2 diabetes were seen and total prevalence of **insulin resistance** was (75%). N.B. There were no obese adolescents with impaired glucose tolerance without metabolic syndrome in this study.

All studied parameters were significantly impaired in group I than non-obese, in group II than group I, in group III than group II and in group VI than group III (*p*<0.011, for all, (Table 2).

Logistic regression analysis proved that the most independent factor for prediction of metabolic syndrome was W/H ration (OR 3.85, CI1.46-5.84, *p*<0.001), followed by W/H ratio (OR 3.71, CI 1.73-4.95, *p*<0.001), BMI (OR 3.24, CI 1.62-4.73, *p*<0.001), FBS (OR 3.24, 1.57-4.56, p<0.001), HbA1c (OR2.95, CI 2.1-4.28, *p*<0.01), HDL (OR 1.95, CI 2.8-3.8, *p*<0.01), triglycerides (OR 1.8, CI 2.1-3.4, *p*<0.01) and family history of diabetes and hypertension (OR 1.5, CI 1.9-2.8, *p*<0.01), (Table 3).

Fasting blood glucose correlated with all studied parameters specially BMI and HOMA-IR (r=0.97 and 0.99), table 4. Also numbers of component of metabolic syndrome correlated with all studied parameters specially BMI and triglycerides (r=0.92 and 0.94), table 5.

**Table (1): Studied of all parameters in obese and non-obese adolescence**

| Parameters | Obese adolescence (N=60) | Non-obese adolescence (N=28) | T-Test | *P*-value |
|---|---|---|---|---|
| Age (years) | 17±0.77 | 17±0.80 | 0.89 | 0.26 (NS) |
| Sex (M/F) | 28/32 | 12/16 | ----- | ----- |
| Weight (Kg) | 82±7.75 | 54±5.9 | 9.4 | <0.001 (VHS) |
| Height (cent) | 158±6.8 | 156±66 | 0.88 | 0.15 (NS) |
| BMI | 31.7±1.9 | 22±0.72 | 1.6 | <0.05(S) |
| W/C (centimeter) | 94±7.3 | 66±5.1 | 1.3 | <0.05 (S) |
| H/C (centimeter) | 96±3.1 | 82±1.9 | 3.2 | <0.01 (HS) |
| W/H ratio | 0.97±0.05 | 0.81±0.05 | 3.8 | <0.01 (HS) |
| SBP (mmHg) | 125±9 | 105±4.9 | 6.48 | <0.001 (VHS) |
| DBP (mmHg) | 80±4.5 | 65±4.7 | 2.95 | <0.01 (HS) |
| FBS (mg/dl) | 92.5±6.7 | 73±3.10 | 7.93 | <0.001(VHS) |
| PPBS (mg/dl) | 113.5±1.7 | 89±4.2 | 1.52 | <0.05 (S) |
| HbA1c | 5.3±0.33 | 4.3±0.32 | 4.1 | <0.01 (HS) |
| HDL (mg/dl) | 35±5.6 | 52±4.3 | 3.45 | <0.01 (HS) |
| TG (mg/dl) | 150±9.83 | 76±6.8 | 3.35 | <0.01 (HS) |
| Fasting insulin | 20.3±3.1 | 5.4±0.92 | 4.96 | <0.01 (HS) |
| HOMA-IR | 4.4±1.1 | 0.96±0.18 | 6.1 | <0.001 (VHS) |

*P*<0.5 (significant), *P* <0.01 (highly significant), *P* <0.001 (very highly significant), NS=non-significant, S=significant, HS=highly significant, VHS=very highly significant

**Table (2): Study of all parameters among all studied groups**

| Parameters | Non-obese | Obese adolescence | | | ANOVA | |
|---|---|---|---|---|---|---|
| | | Group II | Group III | Group VI | F | *P* |
| BMI | 22.1±0.7 | 31.2±0.6 | 32.3±0.6 | 36±1.9 | 959.2 | <0.001** |
| W/C (cent.) | 73.6±5.1 | 97.5±3.6 | 98.5±3.2 | 109.2±6.7 | 231.2 | <0.001** |
| W/H ratio | 0.84±0.1 | 0.94±0.1 | 0.93±0.1 | 1.01±0.1 | 52.2 | <0.001** |
| SBP (mmHg) | 1.5±4.7 | 121±3.1 | 127±5.3 | 143±6.6 | 195.7 | <0.001** |
| DBP (mmHg) | 64.6±4.3 | 78.5±2.9 | 81±3.4 | 87±3.6 | 144.4 | <0.001** |
| FBS (mg/dl) | 72.9±3.1 | 90.1±2.8 | 94.2±2.1 | 106.5±6.3 | 311.7 | <0.001** |
| PPBS (mg/dl) | 88.6±4.2 | 111 ±3.5 | 116.4±4 | 137±17.1 | 159.5 | <0.001** |
| HbA1c | 4.4±0.2 | 5.2±0.2 | 5.4±1 | 6±0.2 | 236.2 | <0.001** |
| HDL (mg/dl) | 50.5±4.3 | 46.2±4.5 | 43.6±3.4 | 36.5±5 | 25.5 | <0.001** |
| TG (mg/dl) | 77.2±6.8 | 144.7±6.3 | 165.4±9.4 | 172±12 | 808.5 | <0.001** |
| Fasting insulin | 5.2±0.9 | 18.5±1.6 | 20.9±1.7 | 25.7±3.1 | 580.2 | <0.001** |
| HOMA-IR | 0.9±0.2 | 4.1±0.5 | 4.9±0.5 | 6.8±1.2 | 370.5 | <0.001** |

r<0.5 (significant) *, r<0.7 (highly significant) ** and r>0.90 (very highly significant) ***

**Table (3): Logistic regression analysis for predicted metabolic syndrome in subjects with one, two or more clinical feature considered**

| Parameters | OR | 95% CI | p-value |
|---|---|---|---|
| W/H ratio | 3.85 | 1.64-5.84 | <0.001 (VHS) |
| HOMA-IR | 3.71 | 1.73-4.95 | <0.001 (VHS) |
| BMI | 3.24 | 1.62-4.73 | <0.001 (VHS) |
| FBS | 3.24 | 1.57-4.56 | <0.001 (VHS |
| HbA1c | 2.95 | 2.1-4.28 | <0.01 (HS) |
| HDL | 1.95 | 2.8-3.84 | <0.01 (HS) |
| Triglycerides | 1.84 | 2.10-3.4 | <0.5 (S) |
| Family history of DM+H | 1.54 | 1.9-2.8 | <0.5 (S) |
| Family history of either DM OR H. alone | 1.24 | 1.5-2.2 | 0.784 (NS) |
| SBP | 1.42 | 1.4-2.12 | 0.854 (NS) |
| DBP | 1.57 | 0.57-1.54 | 0.957 (NS) |

r<0.5 (significant) *, r<0.7 (highly significant) ** and r>0.90 (very highly significant) ***

**Table (4): Correlations between FBS and other parameters of the study**

| | Fasting blood sugar | |
|---|---|---|
| | r-value | *p*-value |
| **Age** | -0.05 | 0.633 (NS) |
| **Weight** | 0.85 | 0.001(HS) |
| **Height** | -0.04 | 0.735(NS) |
| **BMI** | 0.97 | 0.001(HS) |
| **W/C** | 0.93 | 0.001(HS) |
| **H/C** | 0.92 | 0.001(HS) |
| **W/H ratio** | 0.8 | 0.001(HS) |
| **SBP** | 0.94 | 0.001(HS) |
| **PPBG** | 0.97 | 0.001(HS) |
| **HBA1c** | 0.98 | 0.001(HS) |
| **HDL** | -0.68 | 0.001(HS) |
| **TG** | 0.92 | 0.001(HS) |
| **F Insulin** | 0.97 | 0.001(HS) |
| **HOMA IR** | 0.99 | 0.001(HS) |
| **DBP** | 0.91 | 0.001(HS) |

r<0.5 (significant) *, r<0.7 (highly significant) ** and r>0.90 (very highly significant) ***

**Table (5): Correlation between numbers of component of metabolic syndrome and other parameters**

| | Numbers of components of metabolic syndrome | |
|---|---|---|
| | r-value | p-value |
| **Age** | -0.01 | 0.895 |
| **Weight** | 0.73 | <0.001* |
| **Height** | -0.12 | 0.253 |
| **BMI** | 0.92 | <0.001* |
| **W/C** | 0.78 | <0.001* |
| **H/C** | 0.85 | <0.001* |
| **W/H ratio** | 0.68 | <0.001* |
| **SBP** | 0.89 | <0.001* |
| **FBS** | 0.92 | <0.001* |
| **PPBG** | 0.89 | <0.001* |
| **HBA1c** | 0.91 | <0.001* |
| **HDL** | -0.61 | <0.001* |
| **TG** | 0.94 | <0.001* |
| **F Insulin** | 0.9 | <0.001* |
| **HOMA IR** | 0.91 | <0.001* |
| **DBP** | 0.88 | <0.001* |

r<0.5 (significant) *, r<0.7 (highly significant) ** and r>0.90 (very highly significant) ***

**4. Discussion**

Obesity, together with environmental and genetic factors, leads to progression of insulin resistance phase to type2 DM and failure of pancreatic β-cells **(Reaven, 1995)**. Obese children and adolescents with impaired glucose tolerance are predisposed to being high risk for type2 DM in the future after undergoing a mediating period **(Weiss *et al.,* 2005)**.

The prevalence of Metabolic Syndrome in our study according to the IDF definition of metabolic syndrome in adolescents more than the age of 16 was (40%) of the studied obese adolescents (16-18 years) Our findings can be compared with that of **Eapen *et al.* (2010**) in United Arab Emirates who found that (44%) of 260 obese adolescents (12-17 years) were having Metabolic Syndrome.

**Nicola *et al.* (2013)** in Italia, have reported that prevalence of metabolic syndrome was (29.2%) among obese children and adolescents (8-16 years). Also in El-Kuwait, **El-Bayoumy and Shalaby, (2012)** have reported that the overall prevalence of metabolic syndrome was (28.4%) of 352 adolescents (11-17 years). While In Brazil, **Leticia *et al.* (2013)** have reported that (27.6%) of 65 obese adolescents (10-18 years) were having metabolic syndrome according to IDF definition of metabolic syndrome.

Other researchers in Mexico have found that (62%) of 110 obese children and adolescents (8-16 years) has metabolic syndrome **(1Maria *et al.,* 2013)**.

In our study the constituent factors for Metabolic Syndrome were as follows, one factor was present in (19) adolescents with a prevalence of 31.7%, two factors in (17) adolescents with a prevalence of 28.3%,while (24) adolescents 40% of the sample of the study were having three or more criteria of metabolic syndrome.

Our findings can be compared with that of **El-Bayoumy and Shalaby, (2012)** who found the constituent factors for Metabolic Syndrome were as follows, one factor is 26.3% of obese adolescents, two factors in 35.6%, while 98 adolescents (27.8%) of the sample of the study i.e 352 obese adolescents were having three or more criteria of Metabolic Syndrome.

In another study The presence of one, two, or three or more components associated with the metabolic syndrome was 22%, 38% and 30%, respectively **(Cruz *et al.,* 2014).** The increase in prevalence of metabolic syndrome in our study may be due to our adolescents were older (16-19 years) while in **Cruz *et al.,* 2004** (8-13 years) and in **El-Bayoumy and Shalaby (2012)** (11-17 years).

Also our sample was more obese as the mean BMI of sample of **Cruz *et al.* (2004)** was 28.1 and in **El-Bayoumy and Shalaby, (2012)** was 27.3, while in our study the mean BMI was 32.1.

The mechanisms underlying the development of the metabolic derangements that occur in Metabolic Syndrome are not fully understood. The most widely accepted hypothesis involves a complex interaction between insulin resistance and obesity that is modified by social, environmental, and genetic factors **(Pereira *et al.,* 2002).**

Pediatric researchers have found that these criteria persist from childhood to adulthood, leading to the suspect mat the metabolic syndrome continues into adulthood **(Liese *et al.,* 1997)**. Also obese adolescents have a lower exercise capacity than normal weight adolescents **(Vanhala *et al.,* 1999)**. Also obesity alone increases the risk of hypertension, cholecystits, and psychological symptoms in obese children **(Torok *et al.,* 2001).**

Obesity has been associated with increased plasma levels of insulin, this denotes insulin resistance that resulting in diminished ability of insulin to stimulate glucose uptake by the skeletal muscles and adipose tissue, in addition to reducing insulin's ability to suppress hepatic glucose Production and output **(Weiss *et al.,* 2004)** this comes in agreement with our study as the prevalence of insulin resistance in our obese sample was 75%.

Hypertension is recognized as an important component of metabolic syndrome where 25% of obese adolescents in our study have had high systolic blood pressure and 18.3%have had high diastolic blood pressure with a total of 25% of obese adolescents are hypertensive according to the IDF definition of metabolic syndrome, this comes in agreement with **Anapaula *et al.* (2013) in Brazil** who have found that (21%) 321 obese adolescents have high Pressure ≥130/85 mm/Hg, and also with that of **Nicola *et al.* (2013) in Italia** who have found that prevalence of hypertension was (24.1%)of 1080 obese Italian adolescents, and can be compared with that of **Maria *et al.*(2013) in Mexico** who have found that (35%) of 110 obese adolescents are hypertensive.

Besides acting as the central regulator of glucose, insulin also plays an important role on lipid homeostasis. It has long been known that there is a highly significant relation among insulin resistance, compensatory hyperinsulinemia, and hypertriglyceridemia **(Reaven, 2006).**

Insulin has three main influences on lipids: it enhances triglyceride synthesis in liver and adipose tissues, it increases the breakdown of circulating lipoproteins by stimulating lipase activity in adipose tissue, and it suppresses lipolysis in adipose tissue and muscles **(Keskin *et al.,* 2005).**The presence of obesity, visceral body fat, and insulin resistance, and the consequent finding of an impaired lipid profile is well known **(Cook and Kavey, 2011).**

**Weiss *et al.* (2004) and El-Bayoumy and Shalaby, (2012)** suggesting that glucose intolerance may develop later on than other metabolic syndrome abnormalities, **El-Bayoumy and Shalaby, (2012)** had found (5.7%) of their adolescents have impaired glucose tolerance with no cases of tye2 DM.

All of the above researchers do agree with our research as impaired glucose tolerance or high fasting blood glucose was reported only among (15%) of the studied obese adolescents as it is present in (15.6%) of obese females and in (14.3%) of obese males and no cases of type2 DM, although insulin resistance was observed in (75%) of them, this comes also in agreement with **Anapaula *et al.* (2013) in Brazil who** have found that (2%) only of 321 obese adolescents have impaired glucose tolerance Fasting blood glucose ≥100 mg/dl, without any case of type2 DM, although insulin resistance was observed in ( 65%) of the same sample of adolescents.

Several longitudinal studies of adults in USA, demonstrated that hyperinsulinemia can precede the development of type 2 DM by more than 10 years **(Cruz *et al.,* 2004). Beck and Groop, (1994)** have proposed three stages model for development of type 2 diabetes mellitus.

**Stage 1** includes fasting hyperinsulinemia with normal or slightly increased blood glucose. **Stage 2** is characterized by prediabetic glucose intolerance with insulin resistance. **Stage 3** is the development of type 2 diabetes mellitus.

Many of macro vascular changes associated with diabetes mellitus and cardiovascular complications begin in stage 1 and 2 before diagnosis of diabetes.

In our study we found that decrease HDL < 40 mg/dl in males and <50 mg/dl in females according to IDF definition of metabolic syndrome in obese adolescents above age of 16 years was (51.6%) of 60 obese adolescents and hypertriglyceridemia was present also in (51.6 %) that can be compared with that of **Nicola *et al.* (2013)** in Italia who found that (43.6%) of 1080 Italian obese adolescents have decrease HDL and increase triglycerides (TG).

Also in El-Kuwait, **El-Bayoumy and Shalaby, (2012)** have reported that (65.3%) of 352 obese adolescents have decrease HDL and (33.5%) have hypertriglyceridemia. In Mexico, **Maria *et al.* (2013)** have reported that HDL was decreased in (60%) of 110 obese adolescents and (85%) have had increase TG.

## 5. Conclusion

**In Conclusion** overweight and obesity in childhood and adolescence tend to persist into young adulthood with their long-term effects on mortality and morbidity. Insulin resistance is the earliest component of metabolic syndrome occurring as a consequence for obesity in adolescence predisposing to other components of metabolic syndrome and ending lastly into IGT and T2DM.

## References


1. Anapaula CB Rizzo, Tamara BL Goldberg, Carla C Silva, Cilmery S Kurokawa, Helio RC Nunes and José E. (2013): Metabolic syndrome risk factors in overweight, obese, and extremely obese Brazilian adolescents. *Nutrition Journal*, 12:19: 2891-12-19.
2. Beck N and Groop LC. (1994): Metabolic and genetic characterization of states: sequence of events leading to non -insulin-dependent diabetes mellitus.
3. Brown NJ, Agirbasli M and Vaughan DE. (1999): Comparative effect of angiotensin-converting enzyme inhibition and angiotensin II type 1 receptor antagonism on plasma fibrinolytic balance in humans. Hypertension; 34: 285-290.
4. Cook S and Kavey REW. (2011): Dyslipidemia and pediatric obesity. Pediatric Clinical North. Am.; 58:1363-1373.
5. Cruz ML, Weigensberg MJ, Huang TTK, Ball G, Shaibi GQ and Goran MI. (2004): The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. *Journal of Clinical Endocrinology and Metabolism.* 89(1):108–113.
6. Eapen V, Mabrouk A. and Yousef S. (2010): Metabolic Syndrome among the young obese in United Arab Emirates. JTMP 56:325-328.
7. El-Bayoumy and Shalaby (2012): Metabolic Syndrome among Obese Kuwaiti Adolescents (11-17 Years) J Obesity Weight loss Therapy, 2012, 2:1http://dx.doi.org/10.4172/2165-7904.1000110.
8. Keskin M, Kurtoglu S, Kendirci M, Atabek ME and Yazici C. (2005): Homeostasis model assessment is more reliable than the fasting glucose/insulin ratio and quantitative insulin sensitivity check index for assessing insulin resistance among obese children and adolescents. Pediatrics. 115:500-3.
9. Liese AD, Mayer-Davis EJ, Tyroler HA, Davis CE and Keil U. (1997): Development of the multiple metabolic syndrome in the ARIC cohort: Joint contribution of insulin, BMI, and WHR: Atherosclerosis Risk in communities. Ann Epidemiol 7: 407-416.
10. *Ma GS, Ji CY, Ma J, Mi J and Yt Sung R. (2010): Waist circumference reference values*

*for screening cardiovascular risk factors in Chinese children and adolescents. Biomed Environ Sci 23 ((1)): 21–31.*

11. Maria Lola Evia-Viscarra, 1Edel Rafael Rodea-Montero, 1EveliaApolinar and 1Silvia Quintana-Vargas. (2013): Metabolic syndrome and its components among obese (BMI ≥ 95th) Mexican adolescents Published online before print 2013, doi: 10.1530/EC-13-0057EndocrConnectEC-13-0057
12. Monzavi R, Dremaine D and Geffner ME. (2008): Improvement in risk factors for metabolic syndrome and insulin resistance in overweight youth who are treated with lifestyle intervention. Pediatrics. 117:e1111-e1118.
13. Nicola S, Alessandra A, Anna G, Carmine B, Piera S, NT, Pierluigi M, Laura P and Emanuele MG. (2013): Predicting Metabolic Syndrome in Obese Children and Adolescents: Look, Measure and Ask, Obesity Facts, The Europian journal of obesity 2013;6:48–56
14. Pereira MA, Jacobs DR Jr, Van Horn L and Slattery ML. (2002): Dairy consumption, obesity and the insulin resistance syndrome in young adults: The CARDIA study. JAMA 287: 2081-2089
15. Reaven GM. (1995): Pathophysiology of insulin resistance in human disease. Physiol Rev. 75(3):473–486.
16. Reaven GM. (2006): The metabolic syndrome: is this diagnosis necessary? Am J Clin Nutr; 83:1237-47.
17. RobabehG and 55Ali T. (2010): Prevalence of impaired glucose tolerance and insulin resistance among obese children and adolescents There Clin Risk Manag. ; 6: 345–349. Published online 2010 July 21.
18. Torok K, Szelenyi Z, Porszasz J and Molnar D. (2001): Low physical performance in obese adolescent boys with metabolic syndrome, Int J Obes Relat Metab Disord. 25: 966-970
19. Vanhala MJ, Vanhala PT, Keinanen-Kiukaanniemi SM, Kumpusalo EA and Takala JK. (1999): Relative weight gain and obesity as a child predict metabolic syndrome as an adult. Obes Relat Metab Disord 23: 656-659.
20. Weiss R, Dziura J, Burgert TS, Tamborlane WV and Taksali SE. (2004): Obesity and the metabolic syndrome in children and adolescents. N Engl JMed350:2362-23742:951–957.
21. Weiss R, Taksali SE, Tamborlane WV, Burgert TS, Savoye M, Caprio S. (2005): Predictors of changes in glucose tolerance status in obese youth. Diabetes Care; 28:902–909.
22. Zimmet P, Alberti G, Kaufman F, Tajima N, Arslanian S, Wong G, Bennett P, Shaw J and Caprio S. (2007): International Diabetes Federation Task Force on Epidemiology and Prevention of Diabetes: The metabolic syndrome in children and adolescents. Lancet, 369:2059-2061

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