

## Research Article

# Lifestyle and Environmental Factors Associated With Insulin Resistance in Children

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**Abstract:** Sedentary life style, environmental changes and dietary habit like sitting against TV and internet, junk food, no exercise or physical activity causes Obesity. This study was carried out on 100 overweight children, age between 5-16 years of either gender. Patients were selected according to their life style and history of diabetes. Their Body Mass Index (BMI), and waist hip ratio (W/H) was measured by following the criteria of the national heart lung and blood institute. Normal ranges of BMI and waist hip ratios of children are same as adults but calculation is different. BMI and waist hip ratio in children is calculated by Z score. Fasting blood sugar (FBS), insulin and insulin resistance were analyzed in both genders separately. The statistical analysis of anthropometrics study was noted in both the genders of children. It was observed that body mass index was slightly increased in boys  $28.05 \pm 0.33$  than girls  $27.94 \pm 0.34$  with non-significant P value ( $P > 0.05$ ), whereas W/H was significantly increases in girls  $1.399 \pm 0.08$  when compared with boys  $1.89 \pm 0.074$ , but with non-significant P value ( $P > 0.05$ ). FBS was increased in girls  $160.04 \pm 2.55$  when compared with boys  $151.96 \pm 2.32$  with highly significant P value ( $P < 0.001$ ), whereas same level of insulin and insulin resistant (IR) was observed in both cases of children i.e. insulin in boys  $5.82 \pm 0.24$  and girls  $5.77 \pm 0.25$  and insulin resistance in boys  $1.88 \pm 0.061$  and girls  $1.82 \pm 0.054$  with highly significant P value ( $P < 0.001$ ).

**Keywords:** FBS, Insulin resistance

## INTRODUCTION

We hypothesized that lifestyle and environmental factors can defect and alter genes Activity. Adiponectin is a gene which is involved in the body's conversion of food to energy, with the help of oxidative expression phosphorylation [1]. The *Adiponectin* gene is located on chromosome 3q27, which has been reported to be coupled to Type Two Diabetes (T2 DM) and the metabolic syndrome [2]. Reduced plasma adiponectins are also commonly observed in a variety of states frequently associated with insulin resistance, such as cardiovascular disease [3] and hypertension [3]. Adiponectin expressed in adipose tissue directly sensitizes the body insulin by the process of gene Transcription [4]. Transcription is a multi-step process in which information in the genes is used to make proteins. Proteins, in turn, direct cell activity [5]. The *Adiponectin* gene encodes a secreted protein expressed exclusively in both WAT and brown adipocytes [6]. Adipocytes produce non-esterified fatty acids, which inhibit carbohydrate metabolism via substrate competition and impair intracellular insulin signaling. In children, as in adults, several 'adipocytokines' have been related to adiposity indexes as well as to insulin resistance [7]. Adiponectin is one of

the most common cytokines produced by adipose tissue, with an important insulin-sensitizing effect which is associated with anti-atherogenic properties [8].

Insulin is a hormone that influences many processes in the body. Its main role is to manage circulating concentrations of nutrients primarily glucose and fatty acids, the body's two main fuels [9]. It does this by encouraging the transport of nutrients into cells from the circulation, and discouraging the export of nutrients out of storage sites, in response to an increase in circulating nutrients (glucose or fatty acids) [10]. Insulin resistance is characterized by a decrease in the ability of insulin to stimulate the use of glucose by muscles and adipose tissue and to suppress hepatic glucose production and output. Furthermore, it accounts a resistance to insulin action on protein and lipid metabolism and on vascular endothelial function and genes expression [11]. Insulin resistance affects a number of steps in lipid and lipoprotein metabolism in pathway, including hepatic synthesis and secretion of apolipoprotein B, and the enzyme activity of hepatic lipase [12]. Insulin resistance at level of the visceral adipose tissue increases lipolysis and increase synthesis of triglycerides, increase TG blood level, in turn, leads

to lower level of HDL level and increases LDL particle [13].

The occurrence of Type-2 diabetes reported in children has increased frighteningly. Resistance of the body to the actions of insulin results in increased production of this hormone by the pancreas and consequent hyperinsulinemia[14].

Individual lifestyle patterns should be analyzed to identify situations in which altering personal habits can counteract obesity, prevent type 2 diabetes, and promote overall good health [15].

**MATERIALS AND METHODS**

This study was carried out by arranging free camp for obesity in government higher secondary school in north Karachi, after obtaining written Consent from the parents of the subjects and approval from Baqai university ethical committee. For assessment overweight children were involved with a history of sedentary life style and dietary habits and Diabetes mellitus.

The measurement of BMI, and waist hip ratio and some biochemical parameters of FBS, insulin and insulin resistance were included in this study. FBS was measured by enzymatic method by Randox, insulin hormones is estimated by ELISA method (immunochematographic) method and insulin resistance was calculated by homa calculator. The BMI, and waist hip ratio was measured by following the criteria of the national heart lung and blood institute. Cut off body mass index was taken as normal subject < 23 kg/ m<sup>2</sup> and obese subject > 25 kg/m<sup>2</sup>.

Height and weight were recorded with help of height and weight scale. Standing height and weight measured with subject in length clothing and without shoes. Height recorded to the nearest cm and weight to the nearest 0.1 kg. Body mass index (BMI) was calculated by using formula given below;

$$BMI = \text{weight in kg} / \text{height in m}^2$$

Cut off body mass index was taken as normal subject < 23 kg/ m<sup>2</sup> and obese subject > 25 kg/m<sup>2</sup>. Centrally obesity was defined on the basis of waist circumference in cms.

Waist circumference measured at minimum circumference between the lower border of the ribs and iliac crest on mid axillaries' line. Cut off value for waist circumference was taken > 90 cm in men and > 80 cm in women.

**Note:** The formula of children obesity (BMI, Waist hip ratio) is same as Adults but statically analysis was done by Z score.

**Statistical Analysis**

All values expressed as mean ± SEM of that mean and all of the parameters were statistically analyzed by SPSS version 10. To evaluate the significance of the difference between the compared means, two – tailed paired student test was done. (P < 0.001) was considered significant.

**RESULTS**

In Table1 the statistical analysis of anthropometrics study was compared in both genders of children. It was observed that body mass index was slightly increases in boys 28.05± 0.33 than girls 27.94± 0.34. Whereas waist hip ratio was significantly increased in girls 1.399± 0.08 when compared with boys 1.89± 0.074,(Normal range of BMI and waist hip ratio of children are same as adults but calculation is different BMI and waist hip ratio in children is calculated by Z score). The statically analysis was also applied on FBS, it was noted that FBS was increases in girls 160.04± 2.55 when compares with boys 151.96± 2.32 ,whereas same level of insulin and insulin resistant was observed in both cases of children i.e. insulin ( boys 5.82± 0.24 and girls 5.77± 0.25) and insulin resistance in boys\*1.88± 0.061and girls 1.82± 0.054(Table2, Fig. 1).

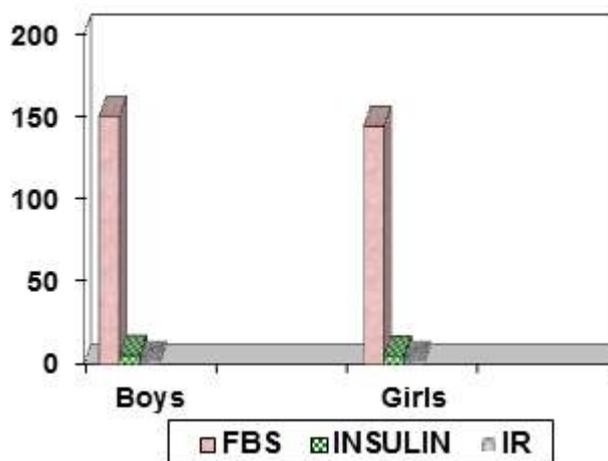
**Table 1: Comparative study of Body mass index, Waist hip ratio in children**

Parameter	Boys(5-10yrs) Mean± SD (20)	Girls(5-10yrs) Mean± SD (18)	P value
BMI	28.05± 0.33	27.94± 0.34	Δ P >0.05
W-H	1.399± 0.08	*1.89± 0.074	Δ P >0.05

\* P <0.05=S (Significant), \*\* P <0.001=H.S (Highly significant), ΔP >0.05=N.S. (Non-significant)

**Table2: Comparative study of Fasting blood sugar, insulin, and Insulin resistance level in obese children**

Parameter	Boys(5-10yrs) Mean± SD (20)	Girls (5-10yrs) Mean± SD (18)	P value
FBS mg/dl	*151.96± 2.32	*160.04± 2.55	P<0.001
Insulin ulu/ml	*5.82± 0.24	*5.77± 0.25	P<0.001
IR	*1.88± 0.061	*1.82± 0.054	P<0.001



**Fig. 1: Comparison of Fasting blood sugar, insulin and insulin resistance in obese children**  
(Each bar Shows Clinical Parameter of fasting blood sugar, insulin, insulin resistance in Obese children)

## DISCUSSION

Sedentary life style, environmental changes and dietary habit like junk food and sitting against TV and internet, no exercise, no physical activity causes Obesity. It plays a central role in the insulin resistance syndrome, which includes hyperinsulinemia, hypertension, hyperlipidemia, type- 2 diabetes mellitus, and an increased risk of atherosclerotic cardiovascular disease [16]. Obesity is generally coupled with an increased release of metabolites by adipose tissue where as adiponectin are inversely related to adiposity. [17]. Therefore, reduced levels of this adipocytokine has been implicated in the pathogenesis of insulin resistance and metabolic syndrome. Decreased levels of adiponectin are associated with insulin resistance in children and adolescents [18]. Adipose tissue also produces tumor necrosis factor- $\alpha$ , an inflammatory factors such as Interleukin-6 (IL-6), C-reactive protein, which can alter insulin action at different levels in the intracellular pathway. Hyperinsulinemia, can lead to increased storage of fat, leading to obesity hypertension (raised blood pressure), and atherosclerosis (hardening/clogging of the arteries). Furthermore, when combined with impaired beta-cell function (beta cells are pancreatic cells that produce insulin), insulin resistance typically leads to type- 2 diabetes [19]. Weight loss is associated with a decrease in insulin concentration and an increase in insulin sensitivity in adults and adolescents [21]. Insulin resistance progresses towards Diabetes Type -2 due to diet high in refined Carbs / low micronutrients. Sedentary lifestyle and Genetics changes causes increase in insulin level in blood along with increase in blood glucose. Pancreatic  $\beta$  cells gets exhausted therefore the insulin production from pancreas decreases leading to

Diabetes Mellitus type as illustrated in flow chart [22].

Diet which is high in refined Carbs / low micronutrients and the sedentary lifestyle along with Genetics will cause Insulin level to increases in the

blood stream. Cells become resistance to insulin, leading to high Blood glucose and insulin level. The Pancreas will decrease insulin Production and blood glucose level remains high causing Type2 diabetes.

## CONCLUSION

It was concluded that, sedentary life style, environmental changes and bad dietary habits alter Gene activity leading to obesity, high levels of Insulin, insulin resistance and Diabetes Mellitus, in children.

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