

# Interrelation between birth weight, body mass index, insulin resistance, C-Reactive Protein-hs, adiponectin and leptin in children (Age 10 to 20 years)

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## Abstract

**Background:** India is projected to have one of the largest populations of obese children over the next decade. Birth weight reflects maternal nutrition and fetal programming which may be a leading cause of obesity. Adipokines also play an important role in development of obesity, inflammation and insulin resistance. The aim of this study is to evaluate serum adiponectin, leptin in Indian school-going children in the age group of 10–20 years. This study also describes distribution of adipokines concentrations and examines the inter-relationship of adipokines with birth weight, body mass index (BMI), C - reactive protein high sensitive (CRP-hs), and insulin resistance (IR). **Methods:** A cross-sectional study involves a total of 200 school going children between the age group of 10-20 yrs. Fasting plasma glucose, insulin, adiponectin, leptin, CRP-hs, were measured for the school going children and birth weight was recorded. BMI was defined as weight (kg) divided by height (m) squared. Insulin resistance was assessed using the homeostasis model (HOMA-IR). **Results:** Children with higher BMI had higher birth weight and insulin. Obese children had lower adiponectin and higher leptin levels suggestive of an inverse association with adipokines. Children with higher BMI had lower glucose. **Conclusion:** Leptin indicator of fat mass induces insulin resistance and inflammation. The derangement observed in all related parameters including insulin resistance, leptin, adiponectin and CRP-hs were due to high fat mass. However, the relationship of birth weight and adipokines needs further evaluation.

**Keywords:** Birth weight, BMI, Insulin resistance, CRP-hs, Adiponectin and Leptin

## Introduction

A positive relationship is reported between birth weight and BMI in childhood or adulthood. Adults with low birth weight were found to have an increased risk of developing diabetes, hypertension, coronary heart disease and stroke in adulthood [1]. This focused attention on the importance of intrauterine life as a determinant of later health. The link between intrauterine exposure, obesity and type 2 diabetes has been explained by the concept of fetal programming [2,3]. In 1991 Hales & Barker reported that low birth weight and thinness at birth are risk factors for type 2 diabetes [4,5]. Obesity is the result of both environmental and

genetic factors. Obesity is associated with low insulin sensitivity and high plasma leptin concentrations [6]. Indians are also reported to have low body mass index but higher adiposity [7]. Rising prevalence of obesity in developing countries is attributed to rapid sedentary life style and reduced physical activity.

Genetically, childhood obesity in Asian Indians is upcoming cause of insulin resistance, diabetes and inflammation. Data analysis suggests birth weight and obesity in adolescent age are interrelated. Birth weight indicates the maternal nutrition and fetal programming. Obesity is one of the factors for insulin resistance, which is better understood by studying the supplementary factors influencing

Manuscript received: 17<sup>th</sup> November 2017  
Reviewed: 27<sup>th</sup> November 2017  
Author Corrected: 4<sup>th</sup> December 2017  
Accepted for Publication: 11<sup>th</sup> December 2017

insulin resistance and inflammation. Adipokines may contribute to the development of obesity and insulin resistance. Reduction in adiponectin plays a primary role in the development of insulin resistance. Adipokines such as leptin, resistin, TNF- $\alpha$ , adipon, visfatin, interleukines-6 and adiponectin are biological active molecules produced by adipose tissue. Limited data suggests that adiponectin is 33% lower in obese children with diabetes than those without diabetes [8].

Some pediatric studies [8–16] have shown a negative association between adiponectin and various measures of insulin resistance (IR), but the association was independent of adiposity in only five of these reports [8–12]. Puberty leads to changes in body fat, contributes to IR [12], and may modify the association between adiponectin and IR.

The present study is undertaken to evaluate serum adiponectin, leptin in Indian school children; to describe distribution of adiponectin concentrations in the age group of 10 – 20 years and to examine the interrelationship of adiponectin with birth weight, BMI, CRP-hs, leptin and insulin resistance.

## Methods

**Subjects:** This study involves a total of 200 school going children between the age group of 10-20 yrs. The children participating in this study mostly came from educated families who lived in an urban setting with relatively middle socio-economic background.

**Design-** A cross-sectional study and total students were categorized into two groups, based on their BMI: group I (120 subjects) as non-obese subjects with a BMI <25 kg/m<sup>2</sup>, group II (80 subjects) as obese subjects with BMI  $\geq$  25 kg/m<sup>2</sup> respectively. Obesity was defined as BMI  $\geq$  25 kg/m<sup>2</sup> [17, 18, and 19]. All the participants have given their informed consent and the study was approved by

## Results

Females had higher BMI ( $p < 0.05$ ), lower adiponectin and higher fat mass as compared to males. 50% of children had low birth-weight. There was significant gender difference in plasma leptin and adiponectin concentration. Obese children had higher leptin ( $p < 0.0001$ ) and lower adiponectin ( $p < 0.001$ ) concentration. 40% of the children were obese but none was diabetic. Low birth weight children remained thin at adolescence. There was significant inverse correlation between leptin and adiponectin. Glycemic status was directly related to adiponectin and inversely related to fat mass. However, insulin resistance was not associated with fat mass (Table 1).

the ethics committee of B. J. Medical College & Sassoon General Hospital, Pune (Ref letter no. BJMC/IEC/Pharmac/D1210156-58).

**Inclusion criteria-** All the willing school going subjects in the age group of 10-20 years children not suffering from any major illness.

**Exclusion criteria-** It consists of subjects not knowing the birth weight, those with diabetes mellitus or other chronic illnesses, and those on medications at the time of the study.

**Participants-** The participants were boys and girls who represent the middle socio-economic population of Pune School.

**Indexes-** The BMI was calculated as body weight (kg) / height (m)<sup>2</sup>. The homeostatic index of insulin resistance (HOMA-IR) was calculated as follows: HOMA-IR = fasting glucose (mg/dL) X fasting insulin (uU/mL) / 405 [20].

**Biochemical methods-** Blood samples of 10 ml were collected in the morning by venous puncture after overnight fasting (at least 08 – 12 hours of fasting) to measure the following biochemical parameters: serum insulin, adiponectin, leptin, CRP-hs concentration were measured by enzyme linked immunoassay respectively (ELISA; Calbiotech, Biovendor, and Diagnostics Biochem). Glucose was measured by GOD-POD method.

**Statistical Method-** Statistical Analysis was performed using Statistical Package of Social Sciences (SPSS, version 12). Means, Medians and Coefficient of Variation (CV) were calculated for all parameters. Independent Student's t test was used to perform gender comparisons as well as between obese and non-obese children. Pearson correlation coefficient ( $r$ ) was used for correlation analysis. A  $p$  value  $\leq$  0.05 was considered significant and ( $p < 0.001$ ) highly significant.

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Mean age of children studied was 13.6 years with 22.19 kg/m<sup>2</sup> BMI. Obese children were older than the non-obese children with higher birth weight. Obese children had lower glucose (p<0.05) and higher fat mass. Children belonged to medium Socio-economic status and physical activities were similar in all children. The significance remained similar after adjusting for gender (Table 1). Adiponectin was lower in obese groups as compared to the non-obese group. Birth weight was inversely associated with plasma adiponectin and glucose and directly with BMI at the time of study age. Inverse correlation has continued from birth to adolescent (Table 2).

**BMI** showed negative correlation with plasma glucose (0.01), Adiponectin (0.01) and positive with plasma insulin (0.05), leptin (0.01), birth weight and CRP-hs (0.01). **Adiponectin** showed negative co-relation with plasma insulin (0.05), HOMA-IR (0.05), CRP-hs (0.01), leptin (0.01) birth weight (0.01) and positively correlated with glucose (0.05). **Leptin** showed positive co-relation with CRP-hs (0.01) & negative correlation with plasma glucose (0.05). **Birth weight** had positive co-relation with BMI & negative co-relation with plasma glucose (0.05) & adiponectin (0.01). **HOMA-IR** showed positive co-relation to plasma glucose (0.01), insulin (0.01) (Table 3).

**Table-1: Clinical characteristics of participants, by gender.**

Variables	Male (94)	Female (106)	Significance (p) value
	Mean	Mean	
Age (years)	13.29 ± 2.97	13.93 ± 2.898	0.147
Weight (Kg)	49.83 ± 22.89	50.62 ± 17.99	0.004*
Height (cm)	147.65 ± 16.89	146.97 ± 12.9	0.005*
Birth Weight (Kg)	2.70 ± 0.712	2.63 ± 0.67	0.423
BMI (Kg/m <sup>2</sup> )	21.66 ± 6.172	22.67 ± 5.95	0.045*
Glucose (mg/dL)	90.97 ± 18.93	92.38 ± 22.69	0.652
Insulin (IU/mL)	12.79 ± 19.32	15.25 ± 19.77	0.404
HOMA-IR	2.99 ± 4.49	3.74 ± 6.28	0.361
CRP-hs (ng/mL)	1286.29 ± 1691.81	1564.14 ± 1994.48	0.145
Adiponectin (µg/mL)	48.44 ± 16.58	41.56 ± 17.10	0.007*
Leptin (ng/mL)	10.92 ± 13.04	16.67 ± 14.93	0.007*

Results are expressed as mean ± standard deviation. Normal weight was defined as a body mass index (BMI) < 25 kg/m<sup>2</sup>; Comparisons between genders was performed using Independent Samples T test.

\* Significant

**Table-2: Characteristics of obese and non-obese children (Kg/m<sup>2</sup>)**

Group (I)	Group (II)			Range	Significance
	Mean	Mean	Mean		
Age (Yrs)	12.32	15.4	13.6	10–20	0.026*
Birth weight (Kg)	2.58	2.80	2.67	1.0–4.6	0.607
Mean BMI (Kg/m <sup>2</sup> )	18.04	27.81	22.19	11.9-39.6	0.0001*
Glucose (mg/dL)	93.61	86.08	91.69	42 – 191	0.032*
Insulin (IU/mL)	13.18	15.15	14.02	0.20-148.1	0.975
HOMA-IR	3.341	3.42	3.36	0.0 – 51.9	0.577
CRP-hs (ng/mL)	868.86	2180.13	1425.6	0.0-11332.4	0.10
Adiponectin (µg/mL)	53.8	32.9	44.89	13.87- 85.46	0.003*
Leptin(ng/mL)	8.73	20.77	13.84	0.0 - 61.6	0.0001*

Group (I), BMI <25Kg/m<sup>2</sup>, N=120, Male=55, Female=65

Group (II), BMI >25Kg/m<sup>2</sup>, N=80, Male=39, Female=41

\*Significant

**Table-3: Correlations of different parameters of the study children.**

	Birth-Weight	BMI	Glucose	Insulin	Homa-IR	CRPhs	Adiponectin	Leptin
<b>BMI</b>								
(r)	0.253**	1.000	-.184**	0.163*	.100	.309**	-.726**	0.468**
(p)	.003	-----	0.010	.019	.102	.000	.000	.000
<b>Glucose</b>								
(r)	-.171*	-.184**	1.000	.195**	.288**	-.089	.161*	-.150*
(p)	.032	.010	-----	.005	.000	.119	.017	.024
<b>Insulin</b>								
(r)	-.087	.163*	.195**	1.0000	.970**	.105	-.157*	.092
(p)	.173	.019	.005	-----	.000	.083	.019	.113
<b>Homa-IR</b>								
(r)	-.128	.100	.288**	.970**	1.000	.104	-.132*	.046
(p)	.082	.102	.000	.000	-----	.085	.040	.273
<b>CRP-hs</b>								
(r)	.069	.309**	-.089	.105	.104	1.000	-.301**	0.313**
(p)	.229	.000	.119	.083	.085	-----	.000	.000
<b>Adiponectin</b>								
(r)	-.246**	-.726**	.161*	-.157*	-.132*	-.301**	1.000	-.392**
(p)	.004	.000	.017	.019	.040	.000	-----	.000
<b>Leptin</b>								
(r)	.102	.468**	-.150*	.092	.046	.313**	-.392**	1.000
(p)	.135	.000	.024	.113	.273	.000	.000	-----

\*\*Correlation is significant at the 0.001 level (1-tailed).

\*Correlation is significant at the 0.05 level (1-tailed).

## Discussion

The main purpose of the present study was to assess the interrelation between birth weight, obesity, glucose tolerance and adipokines of adolescent school children in Pune, India and to predict obesity in adulthood. The children participating in this study were from educated families who lived in an urban setting with relatively middle socio-economic background.

Children with increased birth weight were more obese in their adolescence. They had lower glucose, higher insulin resistance and fat mass. Obesity seems to be due to fat-mass, indicating birth weight are a reflection of obesity in childhood subsequently leading to obesity in adulthood. Girls were more insulin resistant than boys. Bavdekar. et.al, found that low birth weight children who became obese at the age of 8 years were at higher risk of type II diabetes than those who continued to remain thin [21] explained as catch-up growth. Yajnik et al, reported that low birth weight children had higher plasma glucose and insulin concentration at 30 min post glucose load independent of their current size

at 8 years of age with no significant difference in fasting glucose [22]. They also reported that mean glucose and insulin concentration were 8.1 mmol/L and 321 pmol/L in children whose birth weight had been 2.4 kg or less, compared with birth weight more than 3.0 kg with the glucose and insulin concentration 7.5 mmol/L and 289 pmol/L respectively. Whereas we observed mean glucose and insulin levels of non-obese were 90.97 mg/dL and 13.18 IU/mL respectively with mean birth weight 2.58 kg, compared with obese group whose mean glucose and insulin were 86.08 mg/dL and 15.15 IU/mL respectively, whose mean birth weight was 2.8 kgs.

The obesity found in 40% of the study group was due to increased fat mass and higher birth weight. Though there was no significant difference in plasma glucose and insulin between obese and non-obese group, the plasma glucose was lower in obese children with similar insulin levels than normal children indicating insulin insensitivity among obese children which has been derived from

the homeostatis model assessment [20]. Possible reasons for higher prevalence of obesity in our study are that firstly we studied the semi urban population in Pune town with better health conditions in comparison to rural population. Secondly, our study was limited to the children in schools who were usually from a middle socio-economic status with less physical activity.

In our study, in the whole group plasma CRP-hs was associated with BMI. However when we compared the CRP-hs concentrations of obese with non-obese, there was no significant difference. This indicates that adipocytes are not involved in production of low grade inflammation i.e. CRP-hs. It also implies that CRP-hs concentrations were not normally distributed among the children studied. Plasma CRP-hs showed strong positive association with leptin and negative association with adiponectin. Several studies are trying to evaluate whether agents traditionally used to improve glycemic control also significantly reduces CRP-hs [23].

Abdulla et al, found that 76% of obese children had very high CRP-hs levels indicating inflammatory process [24]. Although adiponectin levels did not correlate with CRP-hs levels, we observed dichotomous relationships of adiponectin and CRP-hs levels with generalized and abdominal obesity. We conclude that generalized obesity affects the adiponectin–insulin relationship in post pubertal Asian Indian males; however, the relationship of adiponectin with CRP-hs needs further evaluation [25].

Leptin is considered to be the missing link between obesity and diabetes, as it has been shown to regulate blood sugar via its control on appetite and fat storage. In our study, there was no significant correlation between birth weight and leptin but BMI shows a significant positive correlation with leptin. Leptin, showed a highly significant rise with increasing BMI ( $p < 0.01$ ); [24, 26].

Abdulla et al [24] and Park et al [26] reported a strong association of obesity with leptin and birth weight indicated the relation with current BMI and Naval et al and Marchini et al [25, 27] with maternal nutritional status during the conception of these children. We found obese children had higher plasma leptin concentrations indicating the

presence of higher fat mass. Jornayvaz et al have reported a correlation between leptin with body fat, body mass index and waist circumference [28]. Similarly, Masoud Y Al Maskari et al, found that a strong significant positive correlation between leptin levels and BMI [29]. Aaron R et al studied the association of leptin with BMI and found that adiposity determined leptin but leptin deficiency did not play an important role in obesity in the studied population [30].

Where as Jornayvaz et al found that adult leptin levels decreased consistently with increased birth weight. Overall, the decrease in leptin levels with birth weight suggest that the mechanisms by which low or high birth weight influence the development of obesity in later life might be different.

Interestingly, it has been suggested that children born with a low weight develop high leptin levels during catch-up growth [28]. Salbe et al reported childhood obesity to be the dominant predictor of obesity 5 yrs later in life. These results suggest that the strategies to prevent childhood obesity must be initiated at a very early age [32].

They have also found that fasting insulin concentration at 5yrs of age was a negative determinant of percentage of body fat and fasting leptin concentration was positive determinant of subsequent percentage of body fat. They found that children with higher fasting leptin concentration at base line were fatter and heavier at follow up. However, in our study leptin concentration was not associated with birth weight. Singhal et al found association of low birth weight with higher leptin concentration relative to fat mass suggesting that leptin physiology is programmable [33].

Obese children with reduced adiponectin levels were closely associated with insulin resistance. In this study we observed that obese children had low levels of adiponectin ( $p \leq 0.003$ ) which is in concordance with Arita Y et al. and M. K. Garg et al and Kuo et al who also found that adiponectin concentration was lower in obese subjects as compared to non-obese subjects [34-36]. On the contrary Hung et al reported an association of circulating adiponectin levels with inflammatory markers, insulin resistance and metabolic syndrome independent of obesity [37]. Lele R D et al, showed an inverse association of Adiponectin with obesity



and its indices (BMI and waist circumference) and strongly with insulin resistance. Indeed, intervention studies have shown increase in adiponectin values with weight loss [38]. The low adiponectin levels indicate the need and scope for therapeutic interventions aimed at raising the level.

Intake of diets rich in essential fatty acids protect against insulin resistance and obesity in rodents and increase insulin sensitivity in human [39]. In our study females had significantly higher BMI ( $p \leq 0.045$ ) than males, supported by significantly lower levels of adiponectin ( $p \leq 0.007$ ). On the contrary Antje Bottner et al. found that adiponectin levels progressively decline with physical and pubertal development, especially in boys.

One major finding of his study was that adiponectin levels decrease in parallel with the progression through puberty in boys, but this is not as pronounced in girls. The decline was significantly related to plasma androgen levels and subsequently leads to lower adiponectin levels in adolescent boys compared with girls after completion of puberty [40].

**Limitations:** We excluded adolescents who were not attending school. Children who attend school are likely to come from backgrounds that represent better education and, therefore, the study subjects may not be representative of the wider population.

Adiponectin circulates in different molecular weight complexes, and it appears that it is the high molecular weight (HMW) isoforms are more relevant in the prediction of insulin resistance [41,42].

Although a high correlation between HMW and total adiponectin has been demonstrated, future studies which can assess the HMW-to-total adiponectin ratio may be more informative.

## Conclusion

Better maternal nutrition during pregnancy has led to increased birth weight and offspring obesity and increased fat mass leading to obesity in these children when they were in-utero cannot be ruled out. Birth weight is responsible for obesity and insulin resistance. However, the relationship of birth weight and adipokines needs further evaluation.

**Funding:** Nil, **Conflict of interest:** None  
**Permission of IRB:** Yes

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**How to cite this article?**

Thakre R.R, Naik S.S, Abhang A.S. Interrelation between birth weight, body mass index, insulin resistance, C-Reactive Protein-hs, adiponectin and leptin in children (Age 10 to 20 years). *Int J Med Res Rev* 2017;5 (12):982-990.doi:10.17511/ijmrr. 2017.i12.03

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