Adiponectin, Insulin Resistance, and C-Reactive Protein in Postpubertal Asian Indian Adolescents

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High-sensitivity C-reactive protein (hs-CRP) levels are closely associated with adiposity and predict coronary heart disease and type 2 diabetes mellitus. However, relationships of CRP to adiponectin and other markers of insulin resistance have been inadequately researched in children. We measured fasting serum levels of adiponectin, insulin, hs-CRP, and lipoproteins, and recorded the anthropometric profile and percentage of body fat (%BF; bioimpedance method) in 62 (36 normal weight, 26 overweight) healthy, urban, postpubertal Asian Indian males (aged 14 to 18 years). Serum levels of adiponectin were lower (P = not significant [NS]), whereas those of fasting insulin (P = .01) and hs-CRP (P = .02) were higher in overweight subjects. Adiponectin levels inversely correlated with body mass index (BMI; $r = -0.26$, $P < .05$), %BF ($r = -0.24$, $P < .05$), fasting insulin ($r = -0.32$, $P < .05$) and insulin resistance measured by the homeostasis model of assessment (HOMA-IR; $r = -0.31$, $P < .05$), but not with hs-CRP levels. Fasting insulin and hs-CRP levels correlated significantly with BMI, %BF, waist circumference (WC), waist-to-hip circumference ratio (W–HR), and triceps and subscapular skinfold thickness. The correlation of adiponectin with insulin sensitivity was independent of abdominal obesity, but became nonsignificant after controlling for BMI and %BF. Further, BMI was an independent predictor of adiponectin levels and the ratio of adiponectin and %BF was an independent predictor of fasting insulin levels. Although adiponectin levels did not correlate with hs-CRP levels, we observed dichotomous relationships of adiponectin and hs-CRP levels with generalized and abdominal obesity, respectively. We conclude that generalized obesity affects the adiponectin–insulin relationship in postpubertal Asian Indian males; however, the relationship of adiponectin with hs-CRP needs further evaluation.

AN INCREASING trend of obesity in Asian Indian adolescents and young adults, associated with insulin resistance and dyslipidemia, has been noted recently.[1-3] These derangements may underlie the high predisposition of Asian Indians to develop excess adiposity, abdominal obesity, type 2 diabetes mellitus, and coronary heart disease (CHD) in adulthood.

Although an association between childhood obesity and insulin resistance has been shown,[4,5] the pathophysiological mechanism linking the 2 processes is poorly understood. Recently, a novel adipose tissue-derived cytokine, adiponectin, showed strong inverse correlations with insulin resistance, adiposity, and glucose intolerance in adults.[6-9] A similar relationship in children was demonstrated in a limited number of studies.[10-12] Only one study was done in South Asians, which showed lower adiponectin levels in 25 adult "Indo-Asians" as compared with Caucasians;[13] however, the investigators included diabetic subjects and did not provide the number of Asian Indians in this cohort consisting of many South Asian ethnicities.

C-reactive protein (CRP), synthesized in hepatocytes, is an acute-phase reactant that increases nonspecifically in infections, immunoinflammatory diseases, and malignancies. In the absence of infection, elevations of CRP level generally below 10 mg/L are associated with increased risk of development of atherosclerotic cardiovascular disease.[14] Adiposity is an important determinant of CRP in adults,[15] and in children of different ethnicities, including Asian Indians.[16] Correlation of CRP with insulin resistance, independent of body mass index (BMI), has also been reported.[17] In comparison to Caucasians, South Asians have higher CRP levels.[18] Recently, we reported a high prevalence of elevated CRP levels and its associations in Asian Indian adolescents and young adults residing in India.[16]

Adiponectin may reduce tumor necrosis factor-alpha (TNF-α) levels and cause amelioration of insulin resistance. An inverse correlation of adiponectin with TNF-α has been demonstrated in children of Hispanic and Asian-American origin.[11] Low-grade inflammation has been associated with decrease in plasma adiponectin levels, suggesting an inverse correlation in adults;[19,20] however, no data are available in children, specifically in Asian Indians. The relationships between adiponectin and subclinical inflammation might be mediated by TNF-α and interleukin-6 (IL-6). More importantly, close correlation of adiponectin, and other adipocytokines, with adiposity in childhood[11] may have important bearing on future development of CHD and type 2 diabetes. These issues have not been investigated in Asian Indians.

We hypothesized that serum adiponectin levels are low in overweight, postpubertal Asian Indian males, and are closely associated with insulin resistance and low-grade inflammation, as observed in adults. To test this hypothesis, we measured serum adiponectin levels in healthy postpubertal Asian Indian males selected randomly from a population-based study, and assessed anthropometry, percentage of body fat (%BF), lipoproteins, fasting insulin, insulin resistance, and high-sensitivity CRP (hs-CRP) levels.

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MATERIALS AND METHODS

Subjects

Subjects were selected from among participants of the large ongoing study, Epidemiological Study of Adolescents and Young Adults (ESAY), comprising postpubertal adolescents and young adults, 14 to 25 years of age, from schools and colleges located in southwest New Delhi. The details of the sampling methods used for the study were reported earlier.[16] Briefly, the methodology of multistage cluster sampling according to the modified World Health Organization Expanded Program of Immunization Sampling Plan was adapted.[21] A total of 40 "clusters," defined as a school or a college, were randomly selected based on the proportional allocation of socioeconomic strata. All students in a "section," considered as the primary sampling unit, were recruited. Written informed consent was obtained from the subjects ≥18 years of age and from the parents of those younger than 18 years of age. The study was initiated in August 2000 after approval by the Director of Education, Ministry of Education, Government of New Delhi and the institutional ethics committee.

From the target sample of 4,000 subjects for the ESAY study, 1,795 subjects were recruited until May 2003. For the present analysis, 62 postpubertal males (group 1: normal weight, n = 36; and group 2: overweight, n = 26) aged 14 to 18 years were included. The overweight subjects were older ($P = .02$) than the normal-weight subjects.

Anthropometry and Body Fat Profile

Anthropometric measurements were recorded by a single observer according to methods described earlier.[22] Briefly, we measured height (to the nearest 0.5 cm), weight (to the nearest 0.1 kg), waist and hip circumferences, and skinfold thickness at 4 sites (biceps, triceps, subscapular, and suprailiac). BMI, waist-to-hip circumference ratio (W–HR), sum of 4 skinfolds thickness (Σ4SF), and the product of %BF and waist circumference (WC), designated as BFWC, were calculated. The reproducibility of the skinfold thickness measurements was assessed for each skinfold and the coefficient of variation for the measurement error was less than 10%.

We used 4-point (leg-to-leg) bioelectrical impedance apparatus (Tanita TBF 300, TANITA Corp, Tokyo, Japan) to measure %BF. This method has been validated for Asian children and adolescents[23] and was previously used by us in adolescents and young adults.[16] Patients suffering from any acute illness, particularly those causing dehydration (vomiting, gastroenteritis, etc) were excluded. All subjects were evaluated after an overnight fast and were instructed to avoid drinking fluids and void urine 1 hour before, and just before the measurement was taken. The age, gender, and height of the subjects were manually recorded into the database of the apparatus. The subjects were asked to stand erect on the apparatus with both the bare feet in firm contact with the marked area on the surface of the apparatus, not touching any other object while the impedance was recorded.

Blood pressure was measured after the subject had rested for 5 minutes in the sitting position, using a standard mercury sphygmomanometer (Industrial Electronic and Allied Products, Pune, India) having an appropriate cuff size. If an abnormal recording was noted, another reading was taken after a 5-minute rest and the mean of the 2 values was calculated. The same physician measured the blood pressure using the same instrument, which was periodically validated against a Hawksley Random Zero Sphygmomanometer (Hawksley, Lancing, Sussex, UK).

Biochemical Analysis

After a 12-hour overnight fast, venous blood samples were drawn and transported immediately to the Metabolic Research Laboratory. Serum from blood samples was separated in a cold centrifuge (Plasto Crafts, Mumbai, India) at 2,000 rpm for 10 minutes and stored in a deep freezer at −20°C until assayed. Fasting blood glucose (FBG), total cholesterol (TC), serum triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) concentrations were estimated on the same day using commercially available reagent kits (Randox Laboratory, San Francisco, CA) on a semi-automated analyzer (das srl, Palombara, Sabina, Italy). The value of low-density lipoprotein cholesterol (LDL-C) was calculated according to Freidewald's equation if serum TG levels were less than 400 mg/dL.[24] Serum insulin levels were estimated by the radioimmunoassay method using a commercially available kit (Medicorp, Montreal, Canada). The intraassay coefficient of variation was 2.6%. Insulin resistance was estimated by the homeostasis model of assessment (HOMA) and was designated as HOMA-IR.[25] Measurement of hs-CRP levels was made using an enzyme-linked immunosorbent assay (ELISA) kit (Biocheck, Burlingame, CA). The normal hs-CRP concentration using this assay ranged from 0.068 mg/L to 8.2 mg/L, the lowest detectable limit being 0.005 mg/L according to the manufacturer. The intra-assay variation determined using duplicate samples ranged from 1.7% to 3.3%.

Adiponectin Assay

Adiponectin assay was performed using a radioimmunoassay-based kit (LINCO Research, St Charles, MO). ^{125}I-labeled murine adiponectin and multispecies adiponectin rabbit antiserum supplied along with the kit were used. Radioactivity was measured using a gamma counter (Stratec Biomedical Systems, Pfrozheim, Germany). The amounts of antigen present in unknown samples were deduced from the standard curve. All the samples were analyzed in a single assay and the intra-assay coefficient of variation was 3.5%.

Definitions

We used data from the total sample of the ESAY Study (n = 1,795) as reference to define the cut-off values for anthropometric and biochemical parameters. Values greater than 85th percentile of anthropometric parameters were used to define overweight (BMI ≥ 23.1 kg/m^2), high %BF (≥28.6%), high WC (≥79.1 cm), high W–HR (≥0.87), high triceps skinfold thickness (≥19.8 mm), high subscapular skinfold thickness (≥21.6 mm), and high Σ4SF (≥71.1 mm). Values greater than 95th percentile were used to define hypercholesterolemia (≥169 mg/dL), hypertriglyceridemia (≥119 mg/dL), and high levels of LDL-C (≥108 mg/dL).[16] Low level of HDL-C was defined as a value less than 5th percentile (<38 mg/dL) of the data from the reference population.[16] Hyperinsulinemia was defined as serum insulin values in the highest quartile (≥170 pmol/L). Elevated serum level of hs-CRP was defined as a value greater than 2.1 mg/L.[16]

Statistical Analysis

The data were managed on an Excel spreadsheet (Microsoft Corp, Redmond, WA). The distributions of anthropometric and biochemical parameters were confirmed for the approximate normality. Statistical comparison of normally distributed parameters between the groups was done using Student's *t* test. We used logarithmic transformation of several variables (adiponectin, HOMA-IR, the ratio of adiponectin to %BF, and the ratio of HOMA-IR to adiponectin) for correlation analysis, since their distribution was skewed. Square root transformation was the most appropriate transformation to convert fasting insulin values into normal distribution for correlation analysis since logarithmic transformed values showed significant departure from normal distribution. The correlations of hs-CRP with various parameters did not differ significantly even after various transformations; hence, nontransformed values were used in correlation analyses. Multivariate linear regression analysis was used to adjust for the effects of covari-

Table 1. Clinical, Anthropometric, and Biochemical Profiles

	Group 1 (n = 36)	Group 2 (n = 26)	Normal %BF (n = 20)	High %BF (n = 42)
Age (yr)	15.9 ± 1.2	16.6 ± 1.2*	15.7 ± 1.4	16.4 ± 1.3*
Systolic blood pressure (mm Hg)	117.4 ± 6.8	121.5 ± 9.7*	116.5 ± 6.6	120.1 ± 9.1
Diastolic blood pressure (mm Hg)	77.2 ± 5.8	78.5 ± 7.4	77.0 ± 4.7	78.4 ± 6.9
BMI (kg/m^2)	19.5 ± 2.9	26.4 ± 3.1‡	18.8 ± 3.7	24.1 ± 4.1‡
WC (cm)	70.7 ± 10.2	85.9 ± 9.8‡	66.7 ± 9.3	82.1 ± 11.2‡
Hip circumference (cm)	85.9 ± 8.2	96.8 ± 5.3‡	83.2 ± 7.9	94.0 ± 7.3‡
W–HR	0.81 ± 0.05	0.88 ± 0.06‡	0.79 ± 0.05	0.86 ± 0.06‡
Skinfolds thickness (mm)				
Biceps	7.2 ± 4.3	13.1 ± 5.3‡	5.9 ± 2.8	11.7 ± 5.7‡
Triceps	15.1 ± 6.9	23.7 ± 7.5‡	12.6 ± 5.8	21.7 ± 7.9‡
Subscapular	17.6 ± 9.7	30.6 ± 11.7‡	13.3 ± 6.3	28.0 ± 12.1‡
Suprailiac	16.8 ± 10.6	30.4 ± 9.9‡	11.6 ± 6.9	27.9 ± 11.2‡
Σ4SF	56.7 ± 30.2	97.9 ± 32.6‡	43.4 ± 20.6	89.3 ± 35.2‡
%BF	27.6 ± 8.9	35.5 ± 7.6‡	20.1 ± 4.9	36.0 ± 5.8‡
FBG (mg/dL)	90.9 ± 8.6	89.7 ± 10.4	91.0 ± 7.5	90.3 ± 10.4
TC (mg/dL)	141.1 ± 17.1	139.1 ± 25.1	146.3 ± 15.7	138.9 ± 22.6
Serum TG (mg/dL)	77.0 ± 25.6	98.2 ± 23.4†	75.7 ± 28.1	89.7 ± 25.3
LDL-C (mg/dL)	78.9 ± 20.4	76.6 ± 27.7	87.2 ± 20.7	76.6 ± 22.5
HDL-C (mg/dL)	44.7 ± 5.9	46.7 ± 6	42.6 ± 6.7	45.9 ± 4.7
Fasting insulin (pmol/L)	127.7 ± 46.0	156.6 ± 37.3†	103.9 ± 27.8	158.9 ± 40.7‡
HOMA-IR	4.01 ± 1.5	4.88 ± 1.5*	3.24 ± 0.89	4.99 ± 1.53‡
hs-CRP (mg/L)	2.5 ± 2.7	4.1 ± 2.4*	1.68 ± 1.89	3.94 ± 2.83†
Serum adiponectin (μg/mL)	12.0 ± 5.2	9.9 ± 3.8	12.5 ± 5.3	10.3 ± 4.1

NOTE. Data are expressed as mean ± SD. Group 1: normal-weight males; group 2: overweight males (BMI > 23 kg/m^2).

Abbreviations: BMI, body mass index; WC, waist circumference; W–HR, waist-to-hip circumference ratio; %BF, percentage of body fat (high %BF, >28.5%); FBG, fasting blood glucose; TC, total cholesterol; TG, serum triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; Σ4SF, sum of skinfold thickness at 4 sites (biceps, triceps, subscapular and suprailiac); HOMA-IR, insulin resistance measured by the homeostasis model of assessment; hs-CRP, high-sensitivity C-reactive protein.

*$P < .05$; †$P < .01$; ‡$P < .001$.

ates and to identify independent relationships. We used STATA 8.0, Intercooled version (STATA Corp, College Station, TX) for the statistical analysis. *P* values less than .05 were considered significant.

RESULTS

Clinical, Anthropometric, and Body Fat Profiles

Overweight (group 2) subjects had higher systolic blood pressure ($P < .05$) as compared to normal-weight (group 1) subjects. Excess %BF was recorded in 67.7% (n = 42) (58.3% in group 1 and 80.7% in group 2, P = NS), high WC in 38.7% (n = 24) (11.1% in group 1 and 76.9% in group 2, $P < .001$), and high W–HR in 29% (n = 18) (11.1% in group 1 and 53.8% in group 2, $P < .001$). The mean values of WC, W–HR, skinfold thickness at various sites (biceps, triceps, subscapular, and suprailiac), Σ4SF, and %BF were higher in group 2 subjects and in those with high %BF as compared with subjects in group 1 and in those with normal %BF, respectively ($P < .001$ for all variables, Table 1).

Biochemical Profile

Mean values of FBG and lipid parameters were comparable between the 2 groups, except serum TG that was higher in group 2 ($P = .001$, Table 1). The prevalence of dyslipidemia was not statistically different between the 2 groups.

Fasting Insulin, HOMA-IR, and hs-CRP

Mean values of fasting insulin, HOMA-IR, and hs-CRP were significantly higher in group 2 subjects than in group 1 subjects ($P < .05$) (Table 1). Similarly, subjects with high values of %BF, WC, and W–HR had higher levels of fasting insulin, HOMA-IR, and hs-CRP as compared to those with normal values of these parameters ($P < .05$ for all). Hyperinsulinemia was observed in 34.6% subjects in group 2 and 35% subjects with high %BF as compared to 16.6% subjects in group 1 ($P = .09$) and none of the subjects with normal %BF ($P = .003$). Elevated levels of hs-CRP were observed in 50% subjects, the prevalence being higher in subjects in group 2 (69.2%) and in subjects with high value of %BF (60%) as compared to subjects in group 1 (38.9%, $P = .01$) and those with normal value of %BF (26.3%, $P = .01$). Fasting insulin and hs-CRP levels correlated significantly with BMI, WC, W–HR, triceps and subscapular skinfold thickness, Σ4SF, %BF, and BFWC (Table 2). In addition, fasting insulin showed a positive correlation with systolic blood pressure (SBP) ($r = 0.25$, $P < .05$) but not with hs-CRP ($r = 0.25$, $P = .06$).

Serum Adiponectin

Mean serum adiponectin levels were lower in group 2 subjects as compared with those in group 1 ($P = .08$, Table 1), and in subjects with high %BF (10.6 ± 4.4 μg/mL) as compared

Table 2. Pearsons Correlations of Serum Adiponectin With Anthropometric and Metabolic Variables

Anthropometric and Metabolic Variables	Log (Adiponectin)	hs-CRP	Square Root (fasting insulin)	Log(Adiponectin/%BF)	Log(HOMA-IR/adiponectin)
Systolic blood pressure	−0.11	0.14	0.25*	−0.27*	0.26*
BMI	−0.26*	0.49‡	0.57‡	−0.50‡	0.47‡
WC	−0.17	0.52‡	0.57‡	−0.50‡	0.46‡
W–HR	−0.08	0.39†	0.46‡	−0.40†	0.35†
Triceps skinfold thickness	−0.17	0.44†	0.65‡	−0.50‡	0.51‡
Subscapular skinfold thickness	−0.19	0.44†	0.61‡	−0.52‡	0.51‡
Σ4SF	−0.22	0.47‡	0.66‡	−0.55‡	0.55‡
%BF	−0.24*	0.42†	0.61‡	−0.71‡	0.52‡
Log(adiponectin)	—	0.03	−0.32*	—	—
Square root (fasting insulin)	−0.32*	0.24	—	−0.59‡	0.76‡
Log(HOMA-IR)	−0.31*	0.19	0.96‡	−0.58‡	—
%BFWC	−0.19	0.49‡	0.61‡	−0.64‡	0.49‡
Serum TG	−0.11	0.17	0.17	−0.24*	0.14

*$P < .05$; †$P < .01$; ‡$P < .001$.

with those having normal %BF (12.5 ± 5.3 μg/mL, $P = .1$). Mean serum adiponectin levels were comparable among subjects with normal and high values of WC and W–HR, and in subjects with normal and high hs-CRP levels. Significant negative correlations of serum adiponectin levels with BMI, %BF, fasting insulin levels, and HOMA-IR, but not with hs-CRP levels, were observed (Table 2 and Fig 1). The correlation between serum adiponectin and fasting insulin levels remained significant after adjusting for WC ($r = -0.26$, $P = .04$) and W–HR ($r = -0.30$, $P = .02$). The correlation of adiponectin with HOMA-IR also remained significant after adjusting for W–HR ($r = -0.29$, $P = .02$). However, the correlation of adiponectin with fasting insulin level and HOMA-IR became nonsignificant after adjusting for BMI and %BF (Table 3).

The ratio of HOMA-IR to adiponectin was higher in group 2 subjects (0.54 ± 0.21, $P = .3$) and in subjects with high %BF (0.58 ± 0.33, $P < .01$) as compared to subjects in group 1 (0.45 ± 0.37) and those with normal %BF (0.32 ± 0.18). Similarly, the ratio of adiponectin to %BF was lower in group 2 subjects (0.28 ± 0.11) as compared to group 1 subjects (0.52 ± 0.36, $P < .01$). The ratio of HOMA-IR to adiponectin correlated positively and the adiponectin-to-%BF ratio correlated negatively with SBP, BMI, WC, W–HR, triceps and subscapular skinfold thicknesses, Σ4SF, %BF, BFWC, and fasting insulin (Table 2). In addition, the adiponectin-to-%BF ratio correlated negatively with serum TG levels (Table 2).

To determine the independent predictor(s) of serum adiponectin and hs-CRP levels, BMI, %BF, Σ4SF, WC, W–HR, TG, and HDL-C were included in the regression model. Among these, BMI independently predicted serum adiponectin levels ($t = -2.22$, $\rho = 0.03$; $R^2 = 0.08$), while WC was observed as an independent predictor of hs-CRP levels ($t = 2.18$, $\rho < .03$; $R^2 = 0.28$). The adiponectin-to-%BF ratio and Σ4SF were independent predictors of fasting insulin ($t = -2.52$, $\rho = 0.01$ and $t = 4.27$, $\rho < 0.001$, respectively; $R^2 = 0.54$) and HOMA-IR ($t = -2.21$, $\rho = 0.03$ and $t = 4.49$, $\rho < 0.001$, respectively; $R^2 = 0.53$) among those included in the regression model (BMI, WC, Σ4SF, %BF, serum adiponectin, and adiponectin-to-%BF ratio).

DISCUSSION

This is the first study to explore the relationships among serum adiponectin, insulin resistance, hs-CRP levels, and measures of adiposity in Asian Indian adolescents. An important observation of this study is the presence of a correlation between adiponectin levels with fasting insulin and BMI but not with hs-CRP levels.

Previously, 3 groups of investigators have studied adiponectin levels in children and adolescents.[10-12] While Stefan et al[12] studied 5- to 10-year-old Pima Indian children, Nemet et al[11] studied 12- to 14-year-old Hispanic and Asian children belonging to low socioeconomic stratum, and Weiss et al[10] studied adolescents. Similar to their data,[11,12] we noted a negative correlation of adiponectin levels with generalized obesity, but an absence of any correlation with abdominal obesity. While many other associations of adiponectin (eg, with physical fitness, levels of muscle TG, etc) have been investigated in children and adolescents, associations with hs-CRP have not been addressed previously. Furthermore, much lower mean adiponectin concentrations in adult Indo-Asians[13] were reported than those seen in the present study.

Similar to the data reported by Stefan et al,[12] we observed that the correlations of adiponectin levels with fasting insulin levels and HOMA-IR became nonsignificant after controlling for BMI and %BF. The ratio of adiponectin to %BF and Σ4SF were found to be the independent predictors of fasting insulin levels and HOMA-IR. Furthermore, the correlations of the ratio of HOMA-IR to adiponectin with BMI and %BF were stronger than those with WC and W–HR. These data suggest that, overall, generalized adiposity influences the adiponectin–insulin sensitivity association to a greater extent than abdominal obesity in postpubertal Asian Indian males. Conversely, the adiponectin–fasting insulin relationship appears to be independent of BMI and %BF in adults.[26] Moreover, the association of adiponectin with HDL-C and triglyceride levels independent of insulin sensitivity has been shown in adults.[13,26] Although we did not note any such association, an inverse correlation of TG

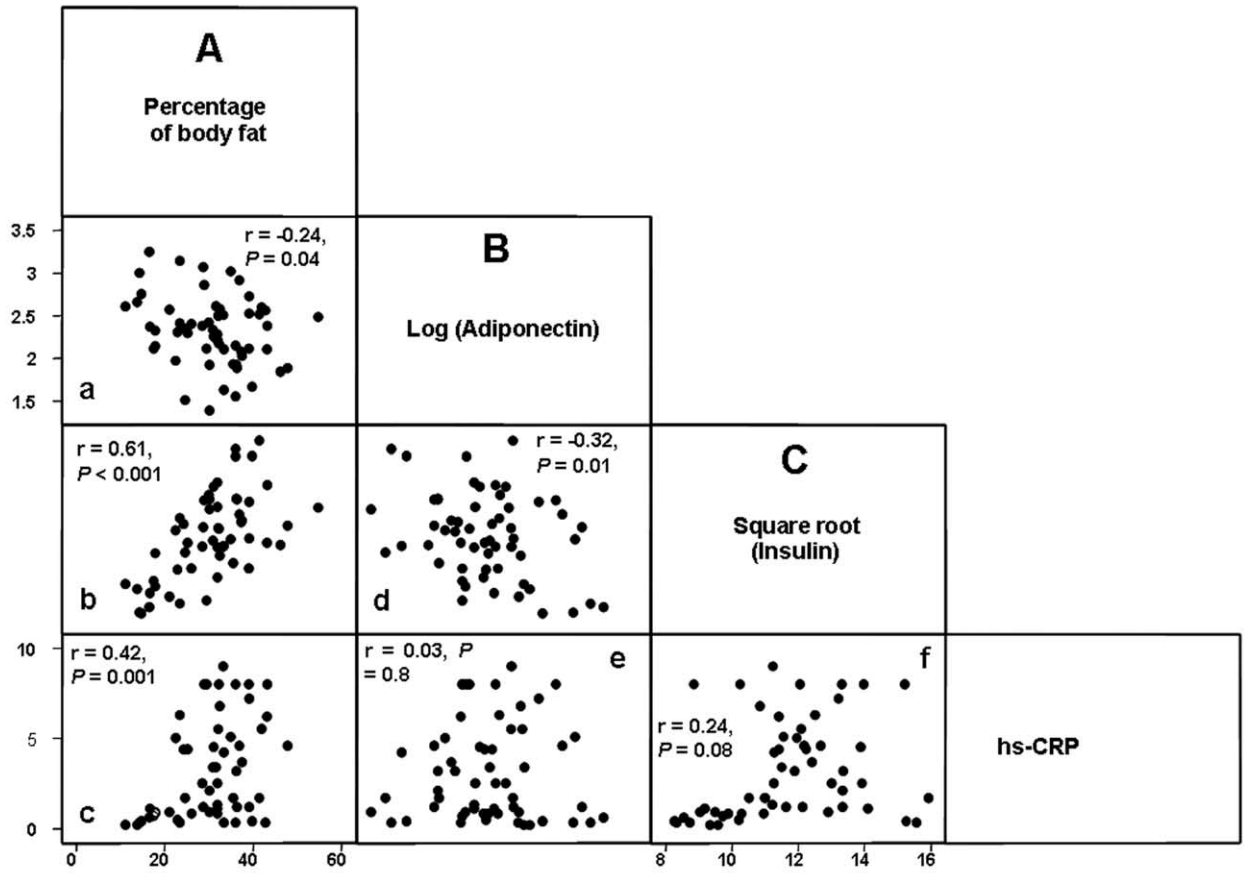

Fig 1. Inter-relationship of adiponectin, fasting insulin, CRP, and %BF. (A) Correlations of percentage of body fat with (a) Log(adiponectin), (b) square root (fasting insulin), and (c) hs-CRP. (B) Correlations of Log(adiponectin) with (d) square root (fasting insulin) and (e) hs-CRP. (C) Correlation of (f) square root (fasting insulin) with hs-CRP. For correlation analysis, logarithmic transformation was done for adiponectin. For fasting insulin, square root transformation was done before correlation analysis, because logarithmic transformed values significantly departed from normal distribution. %BF followed normal distribution and was plotted in straight values. The correlations of hs-CRP with various parameters did not differ significantly even after transformation; hence, nontransformed values were used in correlation analysis. Values of adiponectin in μg/mL; fasting serum insulin in pmol/L; and CRP in mg/L.

levels with the adiponectin-to-%BF ratio, independent of fasting insulin, emerged, which suggests that %BF may affect the adiponectin–TG association in a similar fashion as it affects the adiponectin–insulin sensitivity association in Asian Indian adolescents.

Higher CRP levels have been reported in adult Asian Indians than Caucasians and may contribute to their inordinately high cardiovascular risk.[16] Although, similar to our previous study,[16] hs-CRP levels correlated positively with measures of generalized as well as abdominal obesity, we found no association of hs-CRP levels with low adiponectin levels, in contrast to the finding in adults.[19,27] This variation could be due to the small sample size of the subjects investigated or could represent ethnic variation. Furthermore, the CRP–adiponectin relationship in Asian Indians residing in India may be ambiguous due to recurrent and often persistent elevation of CRP levels due to recurrent and/or persistent infections. Interestingly, our findings suggest dichotomous relationships of adiponectin and hs-CRP with measures of adiposity, the former with generalized obesity and the latter with abdominal adiposity. Whether these findings would also be applicable to postpubertal females and adults remains to be investigated.

This study could be improved by including more subjects, using better methods to assess insulin sensitivity (eg, hyperinsulinemic euglycemic clamp), and more predictors of subclinical inflammation (eg, TNF-α, adhesion molecules).

Table 3. Partial Correlations of Adiponectin With Fasting Insulin and HOMA-IR

Variables Adjusted for	Fasting Insulin	HOMA-IR
Age	$r = -0.32$, $P = .01$	$r = -0.31$, $P = .01$
Age and BMI	$r = -0.24$, $P = .07$	$r = -0.23$, $P = .08$
Age and %BF	$r = -0.22$, $P = .11$	$r = -0.21$, $P = .12$
Age and WC	$r = -0.26$, $P = .05$	$r = -0.24$, $P = .06$
Age and W–HR	$r = -0.30$, $P = .02$	$r = -0.29$, $P = .02$

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