NJPPP National Journal of Physiology, Pharmacy & Pharmacology

DOI: 10.5455/njppp.2015.5.050720141
http://www.njppp.com/

RESEARCH ARTICLE

EFFECT OF TOTAL AND CENTRAL ADIPOSITY ON THE BLOOD GLUCOSE LEVEL OF GUJARATI INDIAN ADOLESCENTS

Minal Chimanlal Patel, Wasim A Shaikh, Sushilkumar Singh

Department of Physiology, Pramukh Swami Medical College, Karamsad, Gujarat, India

Correspondence
Minal Chimanlal Patel
(minalcp@charutarhealth.org)

Received
07.05.2014
Accepted
05.07.2014
Key Words
Waist Circumference; Adiposity; Blood Glucose Level; Adolescents; Guajarati

Background: There is an emerging epidemic of type 2 diabetes mellitus (DM) among children and adolescents due to increased prevalence of obesity. Indians have a genetic phenotype characterized by low body mass index (BMI), high upper body adiposity, and high body fat percentage (BF%) leading to an increased prevalence of metabolic perturbations and DM.
Aims & Objective: To determine the effect of body fat mass on the blood glucose level of Gujarati Indian adolescents during early, middle, and late adolescence.
Materials and Methods: A cross-sectional study was conducted on 468 Gujarati Indian adolescents of age group 13–20 years. Adiposity was assessed using BMI, BF%, fat mass, fat mass index (FMI), and waist circumference (WC). The blood glucose level was measured in the fasting state. Pearson's correlation coefficient was determined to assess the correlation of adiposity markers with fasting blood glucose level.
Results: During early adolescence, in boys, BF% and FMI and in girls, BMI has the strongest positive correlation with fasting blood sugar (FBS), whereas during late adolescence WC shows the strongest significant positive correlation as compared to other parameters in both genders.
Conclusion: This study indicates that central adiposity influences the blood glucose level more strongly than total adiposity in late adolescent phase in Gujarati Indian adolescents.

INTRODUCTION

Obesity is a major risk factor for type 2 diabetes mellitus (T2DM) among children and adolescents. This occurs due to insulin resistance (IR) developed as a result of various metabolic alterations that occur in obesity.[1] Increased release of fatty acids (FAs) due to expansion of fat mass (FM) in obesity may play an important role in impairing glucose uptake and storage in the muscle.[2] Increased adiposity leads to IR by affecting insulin signaling. FAs and potentially several metabolites including acyl-CoAs activate protein kinases such as protein kinase C, which then impair insulin signaling. Adipose tissue releases many adipokines, such as resistin, tumor necrosis factor-α, and interleukin 6, which either modulate or inhibit insulin signaling.[3] Obesity also leads to IR because of ectopic fat deposition in other organs, especially in the viscera. The visceral fat cells secrete a number of inflammatory cytokines that lead to IR.[4]

Indians have a genetic phenotype characterized by low BMI, but with high upper body adiposity, high body fat percentage (BF%), and high IR level. This phenotype predisposes the Indian population to an increased risk of metabolic syndrome and T2DM.[5] Recent epidemiological studies indicate that the prevalence of T2DM is increasing among Indian children and adolescents.[6] Increase in adiposity as indicated by BMI, waist circumference (WC), and waist to hip ratio is considered to be a significant risk factor for T2DM in childhood and adolescence. However, not much has been reported about the effect of body fat mass on the blood glucose level and the effect of growing age during adolescence on the association of fat with the blood glucose level in the Indian population. This study was thus conducted to understand the association of body fat with the blood glucose level with increasing age among adolescents.

MATERIALS AND METHODS

A cross-sectional study was conducted on 468 Gujarati Indian adolescents (262 boys and 206 girls)

of age group 13–20 years. It was carried out after the approval of the institutional Human Research Ethics Committee and after obtaining the informed consent from the participants or the guardian. The study participants were recruited into the study on voluntary basis from schools and colleges in the Anand district using simple random sampling. The study included adolescent boys and girls who were in the age group 13–20 years and had Gujarati language as the mother tongue. The study excluded those participants who were smokers, were athletes, and were suffering from any chronic illness or were on any medical therapy.

Assessment of Adiposity: Adiposity was assessed in terms of body mass index (BMI), BF%, FM, fat mass index (FMI), and WC. The body weight (Wt) was recorded bare footed to the nearest 0.5 kg using a calibrated weighing machine in a standardized state of clothing. The height was measured using meter scale without footwear to the nearest 0.5 cm. BMI was calculated as the weight in kilograms (kg) divided by the height in meters squared (m^2). WC was measured at the midpoint between the lower costal margin and the iliac crest to the nearest 0.5 cm at the end of normal expiration. BF% and FM were assessed by bioelectrical impedance technique using Omron body fat monitor (model HBF-302). FMI was calculated as the FM in kilograms (kg) divided by the height in meters squared (m^2).[7]

Assessment of Blood Glucose Level: The blood glucose level was measured in the fasting state (where the subjects were asked *not* to eat or drink for 8 hours) using the capillary blood by the glucometer Accu-Check Active (accuracy is 0.989 and standard deviation (SD) is 2.7 mg/dl).[7]

Statistical Analysis: Mean and SD were calculated for independent and dependent variables after grouping the study participants into early adolescence (<13 years), middle adolescence (14–16 years), and late adolescence (17–20 years) groups. One-way analysis of variance and *post hoc* test were used to assess if significant (*P*-value < 0.05) differences existed in the dependent and independent variables between the groups. Pearson's correlation coefficient was determined to assess the correlation adiposity markers with fasting blood glucose level.

RESULTS

For boys, BMI and WC were found to be significantly higher in late adolescent phase as compared to early adolescent phase (Table 1), whereas for girls, WC was found to be significantly lower in late adolescent phase as compared to early adolescent phase (Table 2).

Table 1: Subject characteristics of boys

Study Variables	Early Adolescence[a]	Middle Adolescence[b]	Late Adolescence[c]
Weight (kg)	45.9 ± 11.9	45.9 ± 12.7	60.0 ± 12.3****
BMI (kg/m^2)	18.3 ± 3.7	18.2 ± 4.0	20.3 ± 3.4*
BF%	19.9 ± 5.9	18.5 ± 5.5	17.4 ± 6.5
FM (kg)	9.6 ± 4.9	9.5 ± 7.8	11.1 ± 6.3
FMI (kg/m^2)	3.8 ± 1.8	3.8 ± 3.2	3.7 ± 2.0
WC (cm)	63.8 ± 8.5	62.8 ± 9.0	72.8 ± 10.9****
FBS (mg/dl)	84.6 ± 7.7	84.3 ± 6.9	86.5 ± 8.9

*Data presented are mean ± SD. [a] Early adolescence: age ≤ 13 years, N = 27; [b] Middle adolescence: age, 14–16 years, N = 138; [c] Late adolescence: age, 17–20 years, N = 97. Difference between early adolescent phase and late adolescent phase: *P < 0.05, **P < 0.01, ***P < 0.005, ****P < 0.001.*

Table 2: Subject characteristics of girls

Study Variables	Early Adolescence[a]	Middle Adolescence[b]	Late Adolescence[c]
Weight (kg)	43.0 ± 6.4	43.8 ± 9.0	47.1 ± 6.8
BMI (kg/m^2)	19.7 ± 3.1	19.0 ± 3.7	19.6 ± 2.4
BF%	23.4 ± 7.6	23.2 ± 7.3	24.1 ± 5.2
FM (kg)	10.5 ± 4.5	10.7 ± 5.5	13.0 ± 15.5
FMI (kg/m^2)	4.7 ± 2.1	4.6 ± 2.4	4.8 ± 1.6
WC (cm)	59.8 ± 3.9	61.5 ± 6.1	35.6 ± 18.0****
FBS (mg/dl)	84.0 ± 6.4	87.4 ± 8.4	85.3 ± 8.5

*Data presented are mean ± SD. [a] Early adolescence: age ≤ 13 years, N = 27; [b] Middle adolescence: age, 14–16 years, N = 138; [c] Late adolescence: age, 17–20 years, N = 97. Difference between early adolescent phase and late adolescent phase: *P < 0.05, **P < 0.01, ***P < 0.005, ****P < 0.001.*

Table 3: Correlation of adiposity with fasting blood glucose level in boys

Study Variables	Early Adolescence[a]	Middle Adolescence[b]	Late Adolescence[c]
Weight (kg)	0.43*	0.00	0.19
BMI (kg/m^2)	0.58***	0.03	0.27**
BF%	0.68****	0.05	0.26**
FM (kg)	0.62****	0.00	0.26**
FMI (kg/m^2)	0.65****	0.01	0.28***
WC (cm)	0.59****	0.08	0.37****

*Values indicate Pearson's correlation coefficient (r).. [a] Early adolescence: age ≤ 13 years, N = 27; [b] Middle adolescence: age, 14–16 years, N = 138; [c] Late adolescence: age, 17–20 years, N = 97; *P < 0.05, **P < 0.01, ***P < 0.005, ****P < 0.001.*

Table 4: Correlation of adiposity with fasting blood glucose level in girls

Study Variables	Early Adolescence[a]	Middle Adolescence[b]	Late Adolescence[c]
Weight (kg)	0.66*	0.09	0.36****
BMI (kg/m^2)	0.73**	0.12	0.37****
BF%	0.45	0.14	0.37****
FM (kg)	0.57*	0.12	0.19*
FMI (kg/m^2)	0.58*	0.13	0.40****
WC (cm)	0.42	0.08	0.40****

*Values indicate Pearson's correlation coefficient (r).. [a] Early adolescence: age ≤ 13 years, N = 27; [b] Middle adolescence: age, 14–16 years, N = 138; [c] Late adolescence: age, 17–20 years, N = 97; *P < 0.05, **P < 0.01, ***P < 0.005, ****P < 0.001.*

As shown in Table 3, boys showed the strongest positive correlation of fasting blood sugar (FBS) with

BF% and FMI during early adolescence and the strongest positive correlation of FBS with WC during late adolescence. As shown in Table 4, girls showed the strongest positive correlation of FBS with BMI during early adolescence and the strongest positive correlation of FBS with WC and FMI during late adolescence. No correlation was found to exist between blood glucose and adiposity markers during the middle adolescence in both genders (Tables 3 and 4).

DISCUSSION

The study by Young et al.[8] showed that obesity is associated with high fasting glucose and fasting insulin levels in children recognized to be at high risk for T2DM. Body fat, blood pressure, and lipids are all affected by puberty. There are large differences in body composition in boys and girls, with boys having more lean body mass and girls having more body fat. Fat distribution also differs with gender, with boys having a relatively more central fat distribution. These differences begin early in life and become more apparent in puberty due to changes in sex hormone levels.[9] The clearest factor contributing to increased risk of T2DM and cardiovascular disease in children and adolescents is increased body fat, and possibly specific depots of body fat. Some of the earliest evidence for this came from the Bogalusa Heart study that showed weak but significant correlations (r = 0.3–0.4) in children between central body fat (measured by skin folds) and fasting insulin.[10] Later work using more precise measures of body fat found higher correlations in 7- to 11-year-old children between BF% and fasting insulin (r = 0.78).[11] Additional studies using other measures, in addition to fasting insulin, showed that insulin and the insulin-to-glucose ratio were significantly higher in obese versus control group of boys during an oral glucose tolerance test.[12] The study by Gower et al.[13] in 1999 showed that visceral fat has unique metabolic effects on fasting insulin but not on insulin sensitivity and that this effect was independent of other fat compartments and also has shown high inverse correlations between insulin sensitivity and body FM across the spectrum of lean and obese prepubertal boys and girls. Another study was conducted by Goran et al.[14] to examine whether total body fat in general or visceral fat in particular was associated with greater metabolic risk in Caucasian and African-American children. The effect of total body fat and visceral fat on insulin parameters was examined by comparing subgroups of children with high or low fat versus high or low visceral fat and showed that body fat in general is the predominant factor influencing insulin sensitivity, but visceral fat may have additional effects on fasting insulin. Similar effects were shown in a later longitudinal study.[15] These data tend to support the hypothesis that in children, total body FM may influence insulin sensitivity, whereas visceral fat may influence fasting insulin. So, our study is in line with previous studies that show total adiposity is highly correlated with fasting glucose than central adiposity in early adolescent phase in both genders, whereas in late adolescent phase central adiposity shows high positive correlation with fasting glucose in both genders. Fat distribution starts early in life at early adolescent phase, but becomes more marked at late adolescent phase due to changes in sex hormone levels. So, this study indicates that central adiposity affects the blood glucose level more strongly than total adiposity in the late adolescent phase in Gujarati Indian adolescents of age group 13–20 years.

Another important finding in this study was that weight, BMI, and WC significantly increase from early adolescent phase to late adolescent phase in boys, which may be due to changes in sex hormone levels,[9] but WC decreases significantly from early adolescent phase to late adolescent phase in girls. However, WC increased at a similar rate in both genders and large portion of this increase is driven by gains in body weight. We have observed contradictory results in our study, which may be either due to the small number of subjects of early age group involved or due to female sex hormone, estrogen. Estrogen exerts many physiological effects and one of them is to stimulate lipolysis in the abdominal fat depots and promote use of lipid as a fuel by muscles, and the net effect of which is reduction in central adiposity.[9]

CONCLUSION

Both total and visceral adiposities affect the blood glucose level in Gujarati Indian adolescents. However, during early adolescence, total adiposity affects the blood glucose level more strongly than central adiposity, whereas during late adolescence, central adiposity affects more strongly.

LIMITATIONS AND FUTURE PERSPECTIVES

The major limitation of this work is the lack of the

study of sex hormones and their influence on the study variables for which further research is recommended. Another limitation is that we have not measured insulin levels. In a future study insulin levels could be measured to understand if adiposity is affecting insulin secretion or insulin sensitivity. Yet another limitation is the less number of subjects of early age group. Thus, in a future study more number of subjects of early age group could be included.

ACKNOWLEDGMENT

We acknowledge the support of our technical and assistant staff, especially Shri Rameshbhai Rathod, Shri Shailesh Chakravarti, and Shri Rajubhai Khemani, for the research work. We are also grateful to Charutar Arogya Mandal Trust for supporting the research work by providing the necessary infrastructure and equipment.

REFERENCES

1. Stumvoll M, Tataranni PA, Stefan N, Vozarova B, Bogardus C. Perspectives in diabetes: glucose allostasis. Diabetes. 2003;52:903–9.
2. Boden G. Role of fatty acids in the pathogenesis of insulin resistance and NIDDM. Diabetes. 1997;46:3–10.
3. Qatanani M, Lazar MA. Mechanisms of obesity-associated insulin resistance: many choices on the menu. Genes Dev. 2007;21:1443–55.
4. Kim SH, Reaven G. Obesity and insulin resistance: an ongoing saga. Diabetes. 2010;59:2105–6.
5. Ramchandran A. Epidemiology of diabetes in India—three decades of research. J Assoc Physicians India. 2005;53:34–8.
6. Misra A, Misra R, Wijesuriya M, Banerjee D. The metabolic syndrome in South Asians: continuing escalation & possible solutions. Indian J Med Res. 2007;125:345–54.
7. Patel MC, Shaikh WA, Singh SK. Association of sleep duration with blood glucose level of Gujarati Indian adolescents. Indian J Physiol Pharmacol. 2012;56(3):229–33.
8. Young TK, Dean HJ, Flett B, Wood-Steiman P. Childhood obesity in a population at high risk for type 2 diabetes. J Pediatr. 2000;136:365–9.
9. Moran A, Jacobs DR Jr, Steinberger J, Steffen LM, Pankow JS, Hong CP, et al. Changes in insulin resistance and cardiovascular risk during adolescence. Circulation. 2008;117:2361–8.
10. Freedman DS, Srinivasan SR, Burke GL, Shear CL, Smoak CG, Harsha DW, et al. Relation of body fat distribution to hyperinsulinemia in children and adolescents: the Bogalusa Heart Study. Am J Clin Nutr. 1987;46:403–10.
11. Gutin B, Islam S, Manos T, Cucuzzo N, Smith C, Stachura ME. Relation of percentage of body fat and maximal aerobic capacity to risk factors for atherosclerosis and diabetes in black and white seven- to eleven-year-old children. J Pediatr. 1994;125:847–52.
12. Legido A, Sarria A, Bueno M, Garagorri J, Fleta J, Ramos F, et al. Relationship of body fat distribution to metabolic complications in obese prepubertal boys: gender related differences. Acta Paediatr Scand. 1989;78:440–6.
13. Gower BA, Nagy TR, Goran MI. Visceral fat, insulin sensitivity, and lipids in prepubertal children. Diabetes. 1999;48:1515–21.
14. Goran MI, Bergman RN, Gower BA. Influence of total vs. visceral fat on insulin action and secretion in African American and white children. Obes Res. 2001;9:423–31.
15. Huang TK, Johnson MS, Gower BA, Goran MI. Effect of changes in fat distribution on the rates of change of insulin response in children. Obes Res. 2002;10:978–84.

Cite this article as: Patel MC, Shaikh WA, Singh SK. Effect of total and central adiposity on the blood glucose level of Gujarati Indian adolescents. Natl J Physiol Pharm Pharmacol 2015;5:14-17.
Source of Support: Nil
Conflict of interest: None declared