Indian J Med Res 131, May 2010, pp 629-635

Visceral & subcutaneous abdominal fat in relation to insulin resistance & metabolic syndrome in non-diabetic south Indians

S. Sandeep, K. Gokulakrishnan, K. Velmurugan, M. Deepa & V. Mohan

Dr Mohans' Diabetes Specialities Centre & Madras Diabetes Research Foundation, WHO Collaborating Centre for Non-communicable Diseases Prevention & Control, IDF Centre of Education, Chennai, India

Received October 1, 2008

***Background & objectives*: The objective of the study was to determine whether visceral or subcutaneous component of abdominal fat was associated with insulin resistance and metabolic syndrome in non-diabetic Asian Indians.**

***Method*: This cross-sectional study had on 120 individuals with normal glucose tolerance (49 males and 71 females). A single slice CT scan at L4- L5 was done for measurement of visceral and subcutaneous abdominal fat. Metabolic syndrome was defined according to the South Asian Modified National Cholesterol Education Program Adult Treatment Panel III criteria (SAM-NCEP) criteria. Insulin Sensitivity Index (ISI-Matsuda) was used to assess insulin sensitivity/resistance.**

***Results*: Linear regression analysis revealed that visceral, but not subcutaneous fat was associated with serum triglycerides (R^2=0.457, β= 0.34; *P*=0.006), HDL cholesterol (R^2=0.430, β= -0.051; *P*=0.018) and ISI-Matsuda (R^2=0.437, β= -0.05; *P*=0.039) after adjusting for age, gender and BMI. Visceral fat showed significant association with metabolic syndrome (OR: 1.013, 95% CI: 1.001- 1.025; *P*=0.041) even after adjusting for age, gender, body mass index and glycated haemoglobin whereas subcutaneous fat did not show such an association.**

***Interpretation & conclusions*: These results indicate that in non-diabetic Asian Indians, visceral, but not subcutaneous component of abdominal fat is associated with insulin resistance, cardiovascular risk factors and metabolic syndrome.**

Key words Cardiovascular risk factors - diabetes - insulin resistance - insulin sensitivity - metabolic syndrome - south Indians - subcutaneous fat - visceral fat

Central or abdominal obesity has been shown to be an important predictor for increased morbidity and mortality from diabetes and coronary heart disease[1-3]. Abdominal obesity, defined as increased waist circumference is one of the components of the constellation of metabolic abnormalities collectively called as the metabolic syndrome (MS). The latest definition of MS by the International Diabetes Federation (IDF) has included abdominal obesity as one of the essential components[4]. However, it is still unclear whether the visceral (intra-abdominal) or the subcutaneous component of abdominal fat is more

deleterious from the metabolic point of view. There are studies reporting that visceral fat is associated with diabetes and the metabolic syndrome[5,6] and others that subcutaneous fat is associated with insulin resistance[7,8].

Asian Indians are a high risk ethnic group for type 2 diabetes, metabolic syndrome and coronary artery disease and have a unique phenotype called as the "Asian Indian phenotype"[9,10]. This phenotype refers to the fact that despite relatively lower prevalence rates of generalized obesity, they tend to have a greater degree of central body obesity and increased body fat, particularly increased visceral fat, higher plasma insulin levels, insulin resistance and lower adiponectin levels[10-12].

Although studies in other ethnic groups have shown that visceral adipose tissue was a major determinant of MS[13,14], there are none from India that have examined the association of visceral and subcutaneous components of abdominal fat with MS. As India already has the largest number of people with diabetes in the world[15] and the prevalence of MS is also high[16], such studies are of great significance.

This study reports on the association between visceral and subcutaneous component of abdominal fat with insulin resistance, cardiovascular risk factors and MS in non-diabetic Asian Indians

Material & Methods

The study subjects were recruited from the Chennai Urban Rural Epidemiology Study (CURES), an ongoing epidemiological study conducted on a representative population (aged ≥ 20 yr) of Chennai (formerly Madras), the fourth largest city in India. The methodology of the study has been published elsewhere[17]. Briefly, in Phase 1 of the urban component of CURES, 26,001 individuals were recruited based on a systematic random sampling technique. Fasting capillary blood glucose was determined using a One Touch Basic glucose meter (Lifescan, Johnson & Johnson, Milpitas, California, USA) in all subjects. Subjects were classified as 'known diabetic subjects' if they stated that they had diabetes and were on the treatment.

In Phase 2 of CURES, of the known diabetic subjects (n=1529) invited to the centre for detailed studies on vascular complications, 1382 responded (response rate 90.3%). In addition, 10 per cent of newly diagnosed diabetic subjects (n=320, response rate 98.8%), 15 per cent of subjects with impaired fasting glucose (n=866, response rate 99.1%), and 10 per cent of subjects with normal fasting glucose (n=1494, response rate 97.0%) were randomly recruited. The subjects recruited for the Phase 2 of the study underwent detailed anthropometric and biochemical investigations which included Oral Glucose Tolerance Test (OGTT) in non-diabetic subjects. Informed consent was obtained from every participant to undergo this phase of the study. Fasting and 2 h blood glucose measurements were done after a 75 g glucose load. Those who were confirmed by OGTT to have 2 h plasma glucose value ≥ 200 mg/dl based on WHO consulting group criteria[18] were labelled as 'newly detected diabetic subjects', those with 2 h post glucose value ≥ 140 and < 200 mg/dl as impaired glucose tolerance (IGT) and those with 2 h post glucose value < 140 mg/dl as normal glucose tolerance (NGT). Data for the current study had been extracted from the CURES study in the year 2003. Data from subjects with NGT (n=120) were analyzed for this study.

Blood pressure was recorded to the nearest 2 mmHg in the sitting position in the right arm with a mercury sphygmomanometer (Diamond Deluxe BP apparatus; Industrial Electronic and allied products, Pune, India). A trained observer, who was unaware of the clinical status of the subjects, recorded the blood pressure. The first and the fifth Korotkoff's sounds were used to define systolic and diastolic blood pressure, respectively. Two readings were taken 5 min apart, and the mean of the two was calculated. Variations in blood pressure measurements were minimized by (*i*) ensuring 10-min rest before the recording, (*ii*) using appropriate adult cuffs for lean and overweight individuals, and (*iii*) having the same observer record blood pressure.

Fasting plasma glucose (glucose oxidase-peroxidase method) was measured on Hitachi 912 Autoanalyzer (Hitachi, Mannheim, Germany) using kits supplied by Roche Diagnostics (Mannheim, Germany). Serum cholesterol (cholesterol oxidase-peroxidase-amidopyrine method) serum triglycerides (glycerol phosphate oxidase-peroxidase-amidopyrine method) and HDL cholesterol (direct method-polyethylene glycol-pretreated enzymes) were measured using Hitachi-912 Autoanalyser (Hitachi, Mannheim, Germany). The intra and inter assay co-efficient of variation for the biochemical assays ranged between 3.1 to 7.6 per cent. Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald formula[19]. Glycated haemoglobin (HbA1C) was estimated by high-pressure liquid chromatography

using the Variant machine (Bio-Rad, Hercules, Calif., USA). The intra and inter assay co-efficient of variation of HbA1C was <10 per cent.

Serum insulin concentration was estimated using enzyme-linked immunosorbent assay (Dako, Glostrup, Denmark). The intra-assay and the inter-assay co-efficient of variation for insulin assay were 5.7 and 8.9 per cent respectively and the lower detection limit was 0.5 µIU/ml.

Assessment of insulin sensitivity/resistance: The Insulin Sensitivity Index (ISI) derived by Matsuda and DeFronzo (ISI-Matsuda) was used to assess whole body insulin sensitivity using the formula: 10,000/ square root of [fasting glucose (mg/dl) x fasting insulin (µU/ml) x mean glucose (mg/dl) x mean insulin (mg/dl)][20]. Insulin resistance was also calculated using the Homeostasis assessment (HOMA IR) Model using the formula: fasting insulin (µIU/ml) X fasting plasma glucose (mmol/l) / 22.5[21].

MS was diagnosed based on the South Asian Modified NCEP (SAM-NCEP)[22] criteria if 3 or more of the following were present: abdominal obesity (definition of abdominal obesity was modified using Asia Pacific WHO guidelines as waist circumference ≥90 cm for males and ≥80 cm for females, hypertension [subjects who were on antihypertensive medication and/or had systolic blood pressure (SBP) ≥130 mmHg and/or diastolic blood pressure (DBP) ≥85 mmHg]; glucose intolerance (fasting plasma glucose ≥100 mg/dl); hypertriglyceridemia (fasting triglycerides ≥ 150 mg/dl), or low HDL cholesterol (HDL cholesterol: <40 mg/dl for males and <50 mg/dl for females).

CT scan procedure: The scan was done at the Bharat Scans, Chennai, a specialized center for imaging and radiological studies. The observer and the radiologist who interpreted the scans were unaware of the clinical status of the study subjects. Subcutaneous and visceral fat were measured using a Helical CT scan (General Electric, Milwaukee, WI). The scans were done at 120 kV, 200–250 mAs. Subjects were requested to lie in the supine position with their arms above their head and legs elevated with a cushion. A single scan (10 mm) of the abdomen was done at the level of L4-L5 vertebrae and analyzed for a cross sectional area of adipose tissue, which was expressed in centimeters squared. Areas were calculated by multiplying the number of pixels of a given tissue type by the pixel number (pixel density). The external contour of the waist was determined using a threshold of 160 HU (Hounsfield Unit), and the external bone contours were derived at 30 HU. The parameters studied included visceral and subcutaneous fat. Visceral fat was distinguished from subcutaneous abdominal fat by tracing along the fascial plane defining the internal abdominal wall. CT scan was repeated in 10 individuals after a period of one week. Test-retest variability for body fat measurements was less than 5 per cent.

Statistical analysis: Student's 't' test was used to compare groups for continuous variables. Pearson correlation analysis was done to determine the correlation between the visceral and subcutaneous fat and other risk variables. Linear regression analysis was done using visceral and subcutaneous fat as dependent variables and systolic blood pressure, diastolic blood pressure, ISI-Matsuda, total cholesterol, LDL cholesterol, HDL cholesterol, and Serum triglycerides as independent variables and adjusted for confounding factors namely age, gender and BMI. Multiple logistic regression analysis was done using metabolic syndrome as the dependent variable and visceral and subcutaneous fat as independent variables and adjusted for confounding variables like age, gender and BMI. All analyses were done using Windows-based SPSS Statistical Package (version 10.0; SPSS, Chicago, IL), and *P* values <0.05 were considered significant. Complete clinical and biochemical information was available for all the participants.

Results

General characteristics of the study group are given in Table I. Females had significantly higher HDL cholesterol levels (P=0.001) and subcutaneous fat (P<0.001) compared to males. No significant differences were observed in any of the other parameters. Twenty eight subjects (11 males and 17 females) had metabolic syndrome using the SAM-NCEP criteria.

Pearson correlation analysis was used to determine the correlation of visceral and subcutaneous fat with cardiovascular risk factors in both males and females. In both males and females, age (P<0.01), BMI (P<0.001), waist circumference (P<0.001), systolic blood pressure (P<0.001), glycated hemoglobin (P<0.001), serum triglycerides (P<0.001), HDL cholesterol (P<0.05), ISI (P<0.001) and HOMA IR (P<0.01). In males, visceral fat is significantly correlated with diastolic blood pressure (P<0.01), total cholesterol (P<0.01), and LDL cholesterol (P=0.008). Subcutaneous fat showed a significant correlation with BMI (P<0.01), waist

circumference (P<0.001) in both males and females. In males, subcutaneous fat is significantly correlated with HOMA IR (P<0.001).

Table II shows the linear regression analysis using visceral and subcutaneous fat as dependent variables and cardiovascular risk factors as independent variables. Visceral fat was found to be associated with serum triglycerides (P=0.006), HDL cholesterol (P=0.018) and ISI-Matsuda (P=0.039) even after adjusting for confounding variables namely age, gender and BMI whereas subcutaneous fat did not show any association with these variables.

Table III shows the multiple logistic regression analysis with MS as dependent variable and visceral and subcutaneous fat as independent variables. Visceral fat showed significant association with metabolic syndrome (P=0.035) even after adjusting for confounding variables namely age, gender and body mass index whereas subcutaneous fat did not show such an association.

Table I. General Characteristics of the study group

Parameter	Males (n= 49)	Females (n= 71)	*P* value
Age(yr)	42 ± 12	41 ± 9	0.449
Body mass index(kg/m^2)	23 ± 4	24 ± 5	0.070
Waist circumference (cm)	87 ± 10	85 ± 11	0.520
Systolic blood pressure (mm Hg)	120 ± 14	120 ± 16	0.719
Diastolic blood pressure (mm Hg)	78 ± 10	75 ± 10	0.070
Fasting plasma glucose (mg/dl)	87 ± 9	85 ± 10	0.293
2 h plasma glucosa (mg/dl)	95 ± 21	103 ± 26	0.067
Glycated haemoglobin (%)	5.6 ± 0.4	5.6 ± 0.5	0.731
Total cholesterol (mg/dl)	176 ± 37	177 ± 36	0.866
Serum triglycerides (mg/dl)	118 ± 70	106 ± 44	0.254
HDL cholesterol (mg/dl)	42 ± 8	48 ± 11	0.001
LDL cholesterol (mg/dl)	112 ± 29	109 ± 31	0.491
ISI-Matsuda	15.1 ± 12.1	12.3 ± 10.2	0.173
HOMA-IR	1.7 ± 1.4	1.6 ± 0.9	0.811
Visceral fat (cm^2)	122 ± 53	110 ± 52	0.267
Subcutaneous abdominal fat (cm^2)	165 ± 81	235 ± 102	<0.001

Discussion

This study shows that in non-diabetic Asian Indians, visceral, but not subcutaneous fat is significantly associated with metabolic syndrome and that this association is independent of age, gender, generalized obesity and glycemic control. When the individual cardiovascular risk factors were examined in relation to visceral and subcutaneous fat, insulin sensitivity [R^2=0.437, β=-0.05; P=0.039], triglyceride levels [R^2=0.457, β= 0.34; P=0.006], and low HDL cholesterol [R^2=0.430, β= -0.051; P=0.018] showed a significant association with visceral fat even after adjusting for age, gender and BMI. We had earlier reported that visceral, but not subcutaneous abdominal fat is associated with type 2 diabetes in

Table II. Linear regression analysis of visceral and subcutaneous abdominal fat [dependent variable] with cardiovascular risk factors as independent variable

Parameter	R^2, SE, β_1, *P* value	R^2, SE, β_2, *P* value	R^2, SE, β_3, *P* value
Visceral fat – Dependent variable			
Systolic blood pressure	0.09, 0.31, 0.107, 0.002	0.163, 0.66, 0.068, 0.069	0.424, 0.28, 0.078, 0.128
Diastolic blood pressure	0.07, 0.46, 0.051, 0.004	0.164, 0.46, 0.036, 0.049	0.421, 0.39, 0.021, 0.344
Total cholesterol	0.037, 0.12, 0.134, 0.036	0.139, 0.13, 0.044, 0.494	0.419, 0.11, 0.054, 0.490
Serum triglycerides	0.130, 0.08, 0.386, <0.001	0.232, 0.07, 0.380, <0.001	0.454, 0.06, 0.340, 0.006
HDL cholesterol	0.045, 0.45, - 0.054, 0.002	0.192, 0.45, - 0.067, <0.001	0.430, 0.39, - 0.051, 0.018
LDL cholesterol	0.030, 0.15, 0.099, 0.061	0.136, 0.16, 0.019, 0.726	0.417, 0.13, 0.022, 0.736
ISI-Matsuda	0.132, 0.40, - 0.082, <0.001	0.259, 0.38, - 0.087, <0.001	0.437, 0.37, - 0.050, 0.039
Subcutaneous abdominal fat - Dependent variable			
Systolic blood pressure	0.011, 0.62, 0.016, 0.245	0.132, 0.65, 0.017, 0.200	0.444, 0.53, 0.006, 0.693
Diastolic blood pressure	0.025, 0.90, 0.016, 0.086	0.171, 0.88, 0.025, 0.001	0.457, 0.72, 0.020, 0.086
Total cholesterol	0.007, 0.24, 0.030, 0.379	0.127, 0.25, 0.033, 0.322	0.449, 0.20, 0.046, 0.277
Serum triglycerides	0.018, 0.16, 0.076, 0.142	0.150, 0.15, 0.110, 0.044	0.445, 0.12, 0.042, 0.530
HDL cholesterol	0.001, 0.88, - 0.005, 0.633	0.129, 0.89, - 0.016, 0.088	0.444, 0.74, 0.002, 0.842
LDL cholesterol	0.001, 0.30, 0.012, 0.679	0.124, 0.31, 0.022, 0.427	0.447, 0.25, 0.031, 0.369
ISI-Matsuda	0.042, 0.81, -0.006, 0.009	0.146, 0.85, -0.006, 0.029	0.449, 0.69, 0.001, 0.693

β_1, unadjusted, , β_2, adjusted for age and gender, β_3, adjusted for age, gender and BMI; SE, standard error

Table III. Multiple logistic regression analysis using metabolic syndrome as dependent variable and visceral fat and subcutaneous abdominal fat as independent variable

Metabolic syndrome – Dependent variable	SE, odds ratio (95% CI)	*P* value
Visceral fat:		
Unadjusted	0.005, 1.015 (1.006 - 1.025)	0.001
Adjusted for age, gender	0.006, 1.019 (1.009 - 1.030)	0.001
Adjusted for age, gender & BMI	0.006, 1.013 (1.009 - 1.025)	0.035
Subcutaneous abdominal fat:		
Unadjusted	0.002, 1.006 (1.002 - 1.011)	0.007
Adjusted for age, gender	0.002, 1.007 (1.002 - 1.011)	0.009
Adjusted for age, gender & BMI	0.003, 1.002 (0.996 - 1.008)	0.527

SE, standard error; 95% CI, 95% confidence interval

Asian Indians[23]. The findings of the current study are consistent with the findings of earlier studies that visceral component of abdominal fat is more strongly related to cardiovascular risk factors in other ethnic groups[5,6].The findings of this study also confirms the findings of a study done on migrant Indians that increased visceral fat was associated with insulin resistance and dyslipidemia[24].

Recently, a prospective study on Japanese Americans has reported that visceral adiposity is a predictor of future insulin resistance[25]. The results of Framingham Heart Study also support the hypothesis that visceral fat is more strongly associated with an adverse metabolic risk profile[26]. To our knowledge, this is the first report from India to show that the visceral component of abdominal fat is associated with metabolic syndrome in Asian Indians, an ethnic group known to have a higher predilection for cardiovascular disease.

Although the exact molecular mechanisms behind the association of visceral fat with increased cardiovascular risk are unknown, the effect could be due to either anatomical location of the fat within the abdomen or due to the differences in the metabolic properties. The anatomical proximity to the portal venous system leads to the direct drainage of metabolites and secretory products like free fatty acids to the liver resulting in hepatic insulin resistance which in turn may lead to increased hepatic gluconeogenesis[27]. The other hypothesis states that the more active lipolytic feature of visceral adipocytes compared to the subcutaneous adipocytes[28,29] could make visceral fat deposition more deleterious compared to the subcutaneous abdominal deposition. Some studies[30,31] have shown that visceral adipose tissue specifically secretes several biologically active peptides like visfatin and omentin that may modulate glucose and lipid metabolism[31]. We had earlier reported[32] that serum visfatin levels were significantly associated with visceral fat in Asian Indians. However, we did not observe any association of visfatin with insulin resistance or cardiovascular risk factors[32]. Hence novel peptides have to be examined for their potential roles as the links between visceral obesity and adverse metabolic risk profile.

The significant outcome of the study is that an association between increased visceral fat and cardiovascular risk in Asian Indians would help in the early identification of at-risk individuals. Selective reduction of visceral fat induced greater beneficial effects on the parameters of the metabolic syndrome than subcutaneous fat reduction[33]. Another study reported that reduction in visceral adipose tissue area was significantly related to changes in fasting plasma glucose, triglycerides and HOMA score[34].

There are some limitations to our study. Being a cross-sectional study, no cause/effect inferences can be drawn. Secondly, only a single slice CT at L4- L5 level was done to estimate visceral fat area. Finally, we have not used the gold-standard measurement of insulin resistance, the euglycemic clamp technique as it is extremely labour intensive and expensive. However, the ISI-Matsuda derived from OGTT has been shown to be highly correlated with the euglycemic clamp test[20].

In conclusion, this study shows that in non-diabetic Asian Indians, visceral, but not subcutaneous, component of abdominal fat is associated with cardiovascular risk factors like insulin resistance, high triglycerides, low HDL cholesterol and metabolic syndrome. Further studies are needed to elucidate the role of visceral fat in the development of insulin resistance and cardiovascular diseases.

Acknowledgment

We thank the Department of Science and Technology, Government of India, for funding this project, Dr R. Emmanuel of Bharat Scans Chennai for help with the CT scans. This is the thirty seventh report from the Chennai Urban Rural Epidemiology Study (CURES-37) that was supported by the Chennai Willingdon Corporate Foundation, Chennai, India.

References

1. Montague CT, O'Rahilly S. The perils of portliness: causes and consequences of visceral adiposity. *Diabetes* 2000; *49* : 883-8.
2. Kissebah AH, Vydelingum N, Murray R, Evans DJ, Hartz AJ, Kalkhoff RK, *et al*. Relation of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab* 1982; *54* : 254-60.
3. Kannel WB, Cupples LA, Ramaswami R, Stokes J 3rd, Kreger BE, Higgins M. Regional obesity and risk of cardiovascular disease; the Framingham Study. *J Clin Epidemio* 1991; *44* : 183-90.
4. Alberti KG, Zimmet P, Shaw J. IDF Epidemiology Task Force Consensus Group.: The metabolic syndrome—a new worldwide definition. *Lancet* 2005; *366* : 1059-62.
5. Fujioka S, Matsuzawa Y, Tokunaga K, Tarui S. Contribution of intra-abdominal fat accumulation to the impairment of glucose and lipid metabolism in human obesity. *Metabolism* 1987; *36* : 54-9.
6. Pouliot MC, Després JP, Nadeau A, Moorjani S, Prud'Homme D, Lupien PJ, *et al*. Visceral obesity in men. Associations with glucose tolerance, plasma insulin and lipoprotein levels. *Diabetes* 1992; *41* : 826-34.
7. Abate N, Garg A, Peshock RM, Stray-Gundersen J, Grundy SM. Relationships of generalized and regional adiposity to insulin sensitivity in men. *J Clin Invest* 1995; *96* : 88-98.
8. Goodpaster BH, Thaete FL, Simoneau JA, Kelley DE. Subcutaneous abdominal fat and thigh muscle composition predict insulin sensitivity independently of visceral fat. *Diabetes* 1997; *46* : 1579-85.
9. Joshi R. Metabolic syndrome - Emerging clusters of the Indian phenotype. *J Assoc Physicians India* 2003; *51* : 445-6.
10. Deepa R, Sandeep S, Mohan V. Abdominal obesity, visceral fat and type 2 diabetes- "Asian Indian phenotype. In: Mohan V, Rao GHR, editors. *Type 2 diabetes in South Asians:Epidemiology, risk factors and prevention.* New Delhi : Jaypee Brothers Medical Publishers (P) Ltd; 2006. p. 138-52.
11. Sharp PS, Mohan V, Levy JC, Mather HM, Kohner EM. Insulin resistance in patients of Asian Indian and European origin with non-insulin dependent diabetes. *Horm Metab Res* 1987; *19* : 84-5.
12. Raji A, Seely EW, Arky RA, Simonson DC. Body fat distribution and insulin resistance in healthy Asian Indians and Caucasians. *J Clin Endocrinol Metab* 2001 *86* : 5366-71.
13. Carr DB, Utzschneider KM, Hull RL, Kodama K, Retzlaff BM, Brunzell JD, *et al*. Intra-abdominal fat is a major determinant of the National Cholesterol Education Program Adult Treatment Panel III criteria for the metabolic syndrome. *Diabetes* 2004; *53* : 2087-94.
14. Tong J, Boyko EJ, Utzschneider KM, McNeely MJ, Hayashi T, Carr DB, *et al*. Intra-abdominal fat accumulation predicts the development of the metabolic syndrome in non-diabetic Japanese-Americans. *Diabetologia* 2007; *50* : 1156-60.
15. Sicree R, Shaw J, Zimmet P. Diabetes and impaired glucose tolerance. In: Gan D, editor. *Diabetes atlas. International Diabetes Federation*, 3rd ed. Belgium: International Diabetes Federation; 2006. p. 15-103.
16. Deepa M, Farooq S, Datta M, Deepa R, Mohan V. Prevalence of metabolic syndrome using WHO, ATPIII and IDF definitions in Asian Indians: the Chennai Urban Rural Epidemiology Study (CURES-34). *Diabetes Metab Res Rev* 2007; *23* : 127-34.
17. Deepa M, Pradeepa R, Rema M, Mohan A, Deepa R, Shanthirani S, *et al*. The Chennai Urban Rural Epidemiology Study (CURES) – Study design and Methodology (Urban Component) (CURES – 1). *J Assoc Physicians India* 2003; *51* : 863-70.
18. Alberti KG, Zimmet PZ. Definition diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus, provisional report of a WHO Consultation. *Diabet Med* 1998; *15* : 539-53.
19. Friedewald WT, Levy RI, Fredrickson DS. Estimation of low-density lipoprotein cholesterol without the use of the preparative ultracentrifuge. *Clin Chem* 1972; *18* : 499-502.
20. Matsuda M, DeFronzo RA. Insulin sensitivity indices obtained from oral glucose tolerance testing: comparison with the euglycemic insulin clamp. *Diabetes Care* 1999; *22* : 1462–70.
21. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985; *28* : 412-9.
22. Enas EA, Mohan V, Deepa M, Farooq S, Pazhoor S, Chennikkara H. The metabolic syndrome and dyslipidemia among Asian Indians: a population with high rates of diabetes and premature coronary artery disease. *J Cardiometab Syndr* 2007; *2* : 267-75.
23. Anjana M, Sandeep S, Deepa R, Vimaleswaran KS, Farooq S, Mohan V. Visceral and central abdominal fat and anthropometry in relation to diabetes in Asian Indians. *Diabetes Care* 2004; *27* : 2948-53.
24. Banerji MA, Faridi N, Atluri R, Chaiken RL, Lebovitz HE. Body composition, visceral fat, leptin, and insulin resistance in Asian Indian men. *J Clin Endocrinol Metab* 1999; *84* : 137-44.
25. Hayashi T, Boyko EJ, McNeely MJ, Leonetti DL, Kahn SE, Fujimoto WY. Visceral adiposity, not abdominal subcutaneous fat area, is associated with an increase in future insulin resistance in Japanese Americans. *Diabetes* 2008; *57* : 1269-75.
26. Fox CS, Massaro JM, Hoffmann U, Pou KM, Maurovich-Horvat P, Liu CY, *et al*. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. *Circulation* 2007; *116* : 39-48.
27. Gastaldelli A, Cusi K, Pettiti M, Hardies J, Miyazaki Y, Berria R, *et al*. Relationship between hepatic/visceral fat and hepatic insulin resistance in nondiabetic and type 2 diabetic subjects. *Gastroenterology* 2007; *133* : 496-506.
28. Hellmer J, Marcus C, Sonnenfeld T, Arner P. Mechanisms for differences in lipolysis between human subcutaneous and omental fat cells. *J Clin Endocrinol Metab* 1992; *75* : 15-20.

29. Rebuffe-Scrive M, Andersson B, Olbe L, Bjorntorp P. Metabolism of adipose tissue in intraabdominal depots of nonobese men and women. *Metabolism* 1989; *38* : 453-8.

30. Araki S, Dobashi K, Kubo K, Kawagoe R, Yamamoto Y, Kawada Y, *et al*. A Plasma visfatin concentration as a surrogate marker for visceral fat accumulation in obese children. *Obesity* (*Silver Spring*). 2008; *16* : 384-8.

31. Yang RZ, Lee MJ, Hu H, Pray J, Wu HB, Hansen BC, *et al*. Identification of omentin as a novel depot-specific adipokine in human adipose tissue: possible role in modulating insulin action. *Am J Physiol Endocrinol Metab* 2006; *290* : E1253-61.

32. Sandeep S, Velmurugan K, Deepa R, Mohan V. Serum visfatin in relation to visceral fat, obesity, and type 2 diabetes mellitus in Asian Indians. *Metabolism* 2007; *56* : 565-70.

33. Fujioka S, Matsuzawa Y, Tokunaga K, Kawamoto T, Kobatake T, Keno Y, *et al* Improvement of glucose and lipid metabolism associated with selective reduction of intra-abdominal visceral fat in premenopausal women with visceral fat obesity. *Int J Obes* 1991; *15* : 853-9.

34. Park HS, Lee K. Greater beneficial effects of visceral fat reduction compared with subcutaneous fat reduction on parameters of the metabolic syndrome: a study of weight reduction programmes in subjects with visceral and subcutaneous obesity. *Diabet Med* 2005; *22* : 266-72.

Reprint requests: Dr V. Mohan, Chairman & Chief of Diabetology, Dr Mohans' Diabetes Specialities Centre & Madras Diabetes Research Foundation, WHO Collaborating Centre for Non-Communicable Diseases Prevention & Control, 4, Conran Smith Road Gopalapuram, Chennai 600 086, India
e-mail: drmohans@vsnl.net