PGMJ Online First, published on February 25, 2016 as 10.1136/postgradmedj-2015-133363

Association of adiposity, measured by skinfold thickness, with parental history of diabetes in a South Indian population: data from CURES-114

J Surendar, K Indulekha, M Deepa, V Mohan, R Pradeepa

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

Correspondence to
Dr V Mohan, Madras Diabetes Research Foundation and Dr. Mohan's Diabetes Specialities Centre, WHO Collaborating Centre for Non-communicable Diseases Prevention & Control and IDF Centre of Education, Gopalapuram, Chennai 600 086, India;
drmohans@diabetes.ind.in

Received 5 March 2015
Revised 23 December 2015
Accepted 4 January 2016

ABSTRACT

Purpose of the study To look at the association of central and peripheral skinfold thickness with parental history of diabetes in subjects without diabetes.

Methods Subjects with no parental history of diabetes (n=1132), subjects with one parent with diabetes (n=271) and subjects with both parents with diabetes (n=51) were recruited from the Chennai Urban Rural Epidemiological Study (CURES) conducted between 2001 and 2003. Biceps, triceps, medial calf, mid-thigh, chest, abdomen, mid-axillary, suprailiac and subscapsular sites were measured with Lange skinfold callipers.

Results Trunk fat measurements, such as chest (p=0.020), mid-axillary (p=0.005), suprailiac (p=0.014), subscapsular (p<0.001) and abdomen (p=0.010) skinfolds, were highest in subjects with both parents with diabetes followed by those with one parent with diabetes, and lowest in those with no parental history of diabetes. However, the peripheral fat measurements, ie, biceps, triceps, medial calf and mid-thigh, were not significantly different between the study groups. Total truncal and peripheral fat skinfold thicknesses showed a significant positive association with other indices of obesity such as body mass index (BMI) and waist circumference in relation to trunk fat (BMI: r=0.748, p<0.001; waist: r=0.776, p<0.001) and peripheral fat (BMI: r=0.681, p<0.001; waist: r=0.569, p<0.001).

Conclusions A significant association was observed between truncal and peripheral fat, assessed by skinfold thickness, and parental history of diabetes among subjects without diabetes in this urban South Indian population.

INTRODUCTION

Adiposity, which refers to the presence of increased adipose tissue, can cause adverse health outcomes by increasing the risk of type 2 diabetes and cardiovascular disease and other non-communicable diseases.[1] Also, the distribution of body fat has been known to exert a strong influence on the development of insulin resistance and type 2 diabetes.[2] Insulin resistance has been shown to be associated with the visceral fat depot of abdominal fat rather than subcutaneous fat.[3] Although the relative contribution of visceral and subcutaneous fat to the occurrence of type 2 diabetes is yet to be clarified, visceral fat is considered to be a stronger determinant of insulin resistance.[4]

Body mass index (BMI) has been used most commonly as a surrogate measure of adiposity.[5] However, BMI does not provide information about the distribution of body fat,[6] and some studies have shown that it is an unreliable measure of body fat.[7 8] Although dual-energy X-ray absorptiometry (DEXA) is the most reliable and accurate way of measuring body fat mass, cost and logistics limit its use in large epidemiological studies.[9] Hence clinicians and researchers use other measurements such as waist circumference, waist-to-hip ratio[2] or skinfold thickness to measure body fat.

Skinfold thickness is widely used, as it measures fat in the subcutaneous region of the body, which accounts for about 40–60% of total body fat.[10] Skinfold thickness can be easily measured using handheld callipers, which are portable, inexpensive and non-invasive. It is a useful method of measuring body fat distribution in large epidemiological studies.[11]

Skinfold thickness has been shown to have a good agreement with DEXA in providing estimates of body fat and is considered to be superior to bioelectrical impedance.[12 13] The association of skinfold thickness with diabetes was shown in a report that looked at the predictability of BMI, waist-to-hip ratio, and subscapular skinfold thickness for screening type 2 diabetes.[14] Some of the measurement sites for skinfold thickness are the biceps, triceps, chest, subscapular, mid-axillary, suprailiac, abdomen and mid-thigh. Total trunk fat is calculated as the sum of chest, mid-axillary, suprailiac, subscapular and abdomen skinfold thicknesses. Total peripheral fat thickness is calculated as the sum of biceps, triceps, medial calf and mid-thigh skinfold thicknesses.

Another major risk factor for type 2 diabetes is family history of diabetes, and the presence of a parental history of diabetes has been shown to be associated with impairments in insulin secretion and sensitivity.[15] Several studies have looked at the association between obesity and parental history of diabetes. However, there are few data on the association of fat distribution, measured by skinfold thickness, with parental history of diabetes, and none in Asian Indians.

Chennai Urban Rural Epidemiology Study (CURES) is a large cross-sectional study of representative samples of the whole of Chennai and surrounding villages. The study was started in 2001 with the objective of comparing the prevalence of type 2 diabetes and associated risk factors in an urban and rural South Indian population and also to assess the prevalence of diabetes-associated complications, particularly retinopathy and disorders such as glaucoma and cataract in patients with type 2 diabetes. Chennai is the largest city in Southern India and the fourth largest in India and has a population of 4.2 million. The whole of

To cite: Surendar J, Indulekha K, Deepa M, *et al*. *Postgrad Med J* Published Online First: [*please include* Day Month Year] doi:10.1136/postgradmedj-2015-133363

Chennai is divided into 10 zones and 155 wards, and, of these, 46 wards were selected to represent all the 10 zones using a systematic random sampling method. The total sample size of 26 001 individuals was selected as per sample size calculation, and the study was divided into three phases.[16]

The objective of the present study was to look at the association of trunk and peripheral fat measurements, assessed by skinfold thickness, with parental history of diabetes among Asian Indian subjects without diabetes.

SUBJECTS AND METHODS

Subject selection

Study subjects were recruited from CURES, an ongoing epidemiological study conducted on a representative population (aged ≥20 years of age) of Chennai, India. The detailed methodology is published elsewhere.[16] In brief, 26 001 individuals were recruited for phase I of the urban component of CURES conducted between 2001 and 2003, using a systematic sampling technique; subjects with self-reported diabetes receiving drug treatment for diabetes were classified as 'known diabetes subjects'. Details of sampling are given on our website (http://www.drmohansdiabetes.com, under the link 'Publications'). Brief details of the study are presented in figure 1.

For this study, from phase I of CURES we included a total of 1454 subjects with normal glucose tolerance who had both family history and skinfold measurement data and categorised them as follows:

- No parental history of diabetes (n=1132)
- One parent diabetic (n=271)
- Both parents diabetic (n=51)

Parental history of diabetes

On the basis of the parental history of diabetes, the subjects were categorised as no parental history of diabetes, one parent diabetic or both parents diabetic groups.

Skinfold measurements

All measurements were taken in duplicate on the right side of the body with the subject lightly clothed. Measurements were taken to the nearest millimetre using Lange skinfold callipers (Cambridge Scientific Industries, Cambridge, Maryland, USA). The sites of measurement were biceps, triceps, subscapular, chest, suprailiac, mid-axillary, abdomen and mid-thigh using standard techniques. Two well-trained observers took the measurements for male and female subjects separately. The intraobserver mean error was 0.08 mm (p=0.307) for skinfold measurements, and the interobserver mean error was 0.09 mm (p=0.411), which were within the acceptable limits.

The skin was firmly grasped between the thumb and index finger of the left hand about 1 cm or 1/2 inch proximal to the skinfold site and was pulled away from the body. The calliper held in the right hand was placed perpendicular to the long axis of the skin with the calliper's dial facing up so that it was easily readable. The calliper tips were placed on the site of measurement about 1 cm or 1/2 inch distal to the fingers holding the skin, so that the measured value was not affected by pressure from the fingers.[16]

Triceps

The triceps skinfold thickness was measured on the posterior aspect of the right arm, midway from the lateral projection of the acromion process of the scapula and the inferior margin of the olecranon process of the ulna and over the muscle of the triceps.

Biceps

The biceps skinfold is a vertical fold on the anterior arm, directly opposite the triceps skinfold site and over the bump of the biceps muscle.

Chest

The chest or pectoral skinfold measurements were made using a skinfold with its long axis running from the top of the anterior axillary fold to the nipple. The skinfold thickness of the fat fold was measured 1 cm or 1/2 inch below the fingers along the axis.

Subscapular

The subscapular thickness was measured 1 cm below the inferior angle of the scapula, which was identified by gentle palpation of the lower end of the scapula with the subject standing with their arms by their side.

Mid-axillary

This was measured at the level of the right mid-axillary line where it is met by a horizontal line from the xiphisternum (at the bottom of the sternum where the xiphoid process starts). The subject was asked to stand erect with right arm slightly abducted and flexed (bent posteriorly).

Suprailiac

The region immediately above the iliac crest in the mid-axillary line was measured as the suprailiac skinfold.

Abdomen

The study subjects were asked to stand erect with their body weight evenly distributed on both feet. They were then asked to breathe evenly and relax their abdominal muscles. A horizontal fold of skin 1 cm below the umbilicus and 3 cm laterally (to the right) was then measured.

Thigh

The vertical skinfold in the anterior aspect of the thigh midway between the inguinal crease and the upper border of the patella in the midline was measured.

The inguinal crease was located by flexing the hip of the subject.

Medial calf

The point of maximum calf circumference was marked at the medial (inner) aspect of the calf. A vertical skinfold was grasped about 1 cm proximal to the marked site and measured at that site.

Trunk fat

Total trunk fat was calculated as the sum of chest, mid-axillary, suprailiac, subscapular and abdomen skinfold thicknesses.

Peripheral fat

Total peripheral fat thickness was calculated as the sum of biceps, triceps, medial calf and mid-thigh skinfold thicknesses.

Clinical data

Anthropometric measurements, including height, weight and waist, were obtained using standardised techniques. The BMI was calculated using the formula: weight (kg)/height (m^2). Blood pressure (BP) was recorded in the right arm in the sitting position to the nearest 2 mm Hg with a mercury sphygmomanometer (Diamond Deluxe BP Apparatus, Pune). The average of

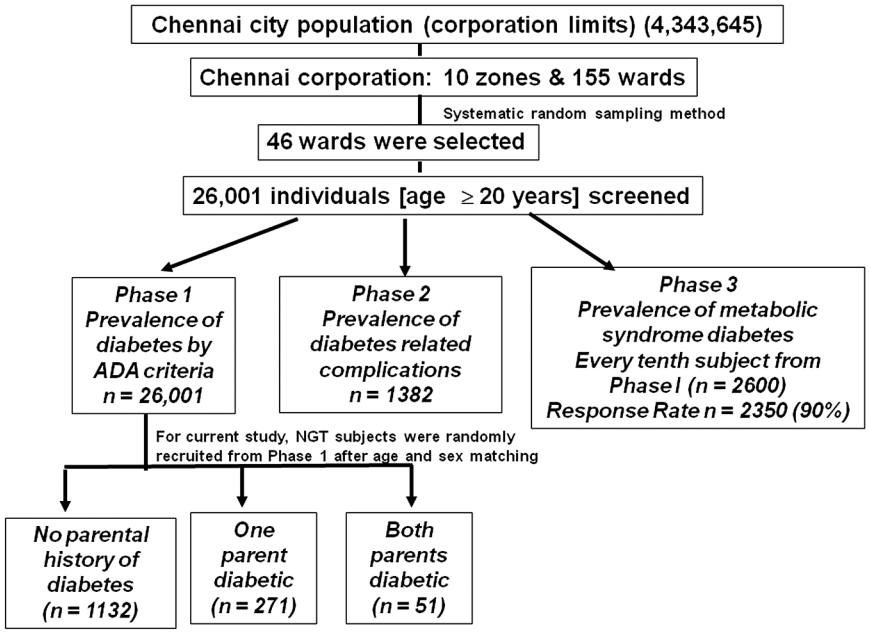

Figure 1 Schematic representation of subject selection. ADA, American Diabetes Association; NGT, normal glucose tolerance.

two consecutive readings taken at an interval of 5 min was used as the BP.

Biochemical data

A fasting blood sample was taken for estimation of plasma glucose and serum lipids after an overnight fast of 8 h. Fasting plasma glucose (glucose oxidase–peroxidase method), serum cholesterol (cholesterol oxidase–peroxidase amidopyrine method), serum triglycerides (glycerol phosphate oxidase–peroxidase–amidopyrine method) and high-density lipoprotein (HDL) cholesterol (direct method–polyethylene glycol-pretreated enzymes) were measured using the Hitachi-912 Autoanalyzer (Roche Diagnostics/Hitachi, Mannheim, Germany). The intra- and inter-assay coefficients of variation for the biochemical assays ranged between 3.1% and 7.6%. Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald formula. Glycated haemoglobin was estimated by high-pressure liquid chromatography using a Variant Chromatograph (Bio-Rad, Hercules, California, USA). The intra- and inter-assay coefficients of variation of glycated haemoglobin were less than 10%. Insulin resistance was calculated using the homoeostasis assessment model (HOMA-IR) using the formula: fasting insulin (μU/mL) fasting glucose (mmol/L)/22.5.[17]

Data analysis

Details pertaining to demography and parental history of diabetes were elicited using a structured, pretested and validated interviewer-administered questionnaire. Total trunk and peripheral fat were derived, and their association with parental history of diabetes was analysed using statistical tests outlined below.

Statistical analysis

Summary measures were means for continuous variables and percentages for categorical variables. Analysis of variance for continuous variables and χ^2 for categorical variables were used to identify statistical significance. For variables that were not normally distributed (such as HOMA-IR), the non-parametric Kruskal–Wallis test was used to test the statistical significance. Logistic regression was used to present the point estimates and CIs for association of individual variables with parental history of diabetes, using no parents with diabetes as the reference group. Linear regression models were used to assess the association of fat measurements with parental history of diabetes using fat measurement as the dependent variable and parental history of diabetes as a continuous, independent variable. The levels of parents having diabetes were treated as categorical predictors for table 1 and a continuous predictor (for the test for trend) in table 2. Relationships between adiposity measures and skinfold measurements were explored by partial correlations (Pearson product moment). $p<0.05$ was considered significant. All data were analysed using SPSS V.15.0 software.

Ethics committee permission

All procedures performed were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments. Institutional ethics committee approval from the Madras Diabetes Research Foundation was obtained before the start of the study, and written informed consent was obtained from all the study subjects.

RESULTS

Clinical and biochemical characteristics

Age- and gender-matched subjects were used for this study, with the result that 8.4% of subjects were excluded. The mean (SD) of the various clinical variables of the excluded subjects did not differ from that of those included. BMI ($p=0.023$) and waist circumference ($p=0.003$) were higher in the group in which both parents were diabetic compared with those with one diabetic parent and those with no parental history of diabetes (table 1).

Trunk fat measurements are increased in subjects with parental history of diabetes

There was a linear increase in truncal (central) fat measurements according to parental history of diabetes. The mean values of chest (no parental diabetes: 13.6 mm; one parent diabetic: 14.3 mm; both parents diabetic:15.3 mm; p for trend 0.020), mid-axillary (19.2 mm; 20.5 mm; 21.6 mm; p for trend 0.005),

Table 1 Clinical and biochemical characteristics of the study group

Variable	No parental history of diabetes (n=1132)	One parent diabetic (n=271)	Both parents diabetic (n=51)	p Value	No vs one parent diabetic	No vs both parents diabetic
Gender, male	411 (36.3%)	95 (35.1%)	18 (35.3%)	–	0.95 (0.72–1.25), p=0.700	0.96 (0.53–0.72), p=0.883
Age (years)	34.5 (8.5)	33.3 (8.2)	34.7 (8.7)	0.112	0.98 (0.97–0.99), p=0.040	1.00 (0.97–1.04), p=0.823
Body mass index (kg/m^2)	23.2 (4.8)	23.9 (4.5)	24.4 (3.7)	0.023	1.03 (1.00–1.06), p=0.028	1.05 (0.99–1.11), p=0.750
Waist circumference (cm)	81.7 (12.1)	83.9 (11.8)	85.6 (10.5)	0.003	1.02 (1.00–1.03), p=0.006	1.03 (1.00–1.05), p=0.024
Systolic blood pressure (mm Hg)	114 (15)	115 (14)	117 (17)	0.504	1.02 (0.99–1.01), p=0.884	1.01 (0.99–1.03), p=0.249
Diastolic blood pressure (mm Hg)	72 (11)	73 (10)	75 (10)	0.146	1.01 (0.99–1.02), p=0.381	1.02 (0.99–1.03), p=0.074
Fasting plasma glucose (mg/dL)	83.9 (7.9)	84.2 (7.7)	84.0 (8.4)	0.920	1.00 (0.99–1.02), p=0.683	1.00 (0.97–1.04), p=0.984
Glycated haemoglobin (%)	5.5 (0.6)	5.5 (0.5)	5.6 (0.5)	0.432	1.01 (0.81–1.25), p=0.935	1.17 (0.90–1.53), p=0.239
Total cholesterol (mg/dL)	170 (32)	171 (31)	174 (33)	0.624	1.00 (0.99–1.01), p=0.517	1.00 (0.99–1.01), p=0.443
Serum triglycerides (mg/dL)	106 (60)	111 (51)	101 (40)	0.340	1.00 (0.99–1.00), p=0.187	0.99 (0.99–1.00), p=0.627
HDL cholesterol (mg/dL)	43 (10)	42 (9)	44 (11)	0.098	0.99 (0.97–0.99), p=0.034	1.00 (0.97–1.03), p=0.805
LDL cholesterol (mg/dL)	106 (27)	107 (27)	110 (27)	0.446	1.00 (0.99–1.01), p=0.385	1.01 (0.99–1.02), p=0.316
HOMA-IR	1.28 (1.24)	1.43 (1.25)	1.50 (1.46)	0.379	1.05 (0.95–1.17), p=0.321	1.11 (0.92–1.35), p=0.287

Values are presented as mean (SD) except for gender, which is given as n (%).
Comparison of groups was made using analysis of variance for normal data and the Kruskal–Wallis test for non-normal data. The comparisons are OR (95% CI).
HDL, high-density lipoprotein; HOMA-IR, homoeostasis model assessment of insulin resistance; LDL, low-density lipoprotein.

suprailiac (22.3 mm; 23.6 mm; 24.9 mm p for trend 0.014), subscapular (22.4 mm; 24.4 mm; 25.9 mm; p for trend <0.001) and abdomen (25.4 mm; 26.7 mm; 28.3 mm; p for trend 0.010) measurements were lowest in the no parent diabetic group followed by one parent diabetic, and highest in the both parents diabetic group (table 2).

Peripheral fat measurements are not associated with parental history of diabetes

The levels of peripheral fat —that is, biceps, triceps, medial calf and mid-thigh regions—were measured in the three study groups. The fat measurements in the biceps (p for trend 0.191), triceps (p=0.100), medial calf (p=0.164) and mid-thigh (p=0.178) were not significantly different between the study groups (table 3).

Total trunk fat but not peripheral fat is associated with parental history of diabetes

Total trunk and peripheral fat measurements were derived by summing the individual measurements. There was a linear increase in total trunk fat in the no parental diabetes group, followed by the one parent diabetic and both parents diabetic groups (no parental diabetes: mean 103 mm; one parent diabetic: mean 109.5 mm; both parents diabetic: mean 115.9 mm; p for trend 0.001). No such trend was observed in the case of peripheral fat measurements (p=0.136).

Association of total trunk fat with BMI and waist

Total trunk fat showed a positive association with BMI (r=0.748, p<0.001) and waist circumference (r=0.776, p<0.001). Peripheral fat also showed a positive association with BMI (r=0.681, p<0.001) and waist circumference (r=0.569, p<0.001) (figure 2).

DISCUSSION

The major findings of the study are as follows. In non-diabetic Asian Indians, trunk fat, but not peripheral fat, is associated with a parental history of diabetes. Trunk and peripheral fat are also associated with other indices of obesity such as BMI and waist circumference.

Our findings thus show the value of measurements of skinfold thickness as an additional tool in clinical practice, as they provide a simple measure of trunk and peripheral fat distribution. Hughes *et al*[18] have suggested that, owing to its high sensitivity, skinfold thickness should be used to monitor obesity in children. Sosenko *et al*[14] have shown that subscapular skinfold thickness is more accurate than BMI in screening for type 2 diabetes. They also showed that subscapular skinfold thickness

Table 2 Trunk fat measurements in the study groups

Trunk fat measurement site	Fat measurement			p for trend	Linear regression coefficient (95% CI), p value
	No parental history of diabetes (n=1132)	One parent diabetic (n=271)	Both parents diabetic (n=51)		
Chest	13.6 (5.5)	14.3 (5.5)	15.3 (5.2)	0.020	0.78 (0.23 to 1.33), p=0.005
Mid-axillary	19.2 (7.3)	20.5 (7.2)	21.6 (6.9)	0.005	1.21 (0.49 to 1.94), p=0.001
Suprailiac	22.3 (8.4)	23.6 (8.6)	24.9 (7.8)	0.014	1.27 (0.42 to 2.12), p=0.004
Subscapular	22.4 (8.7)	24.4 (8.4)	25.9 (8.2)	<0.001	1.87 (1.00 to 2.74), p<0.001
Abdomen	25.4 (9.0)	26.7 (8.0)	28.3 (7.9)	0.010	1.37 (0.48 to 2.25), p=0.002

Fat measurements are in mm presented as mean (SD). Linear regression model was used to assess the association of fat measurements with parental history of diabetes; values are presented as coefficient and 95% CI.

Table 3 Peripheral fat measurements in the study groups

Peripheral fat measurement site	Fat measurement			p for trend	Linear regression coefficient (95% CI), p value
	No parental history of diabetes (n=1132)	One parent diabetic (n=271)	Both parents diabetic (n=51)		
Biceps	12.3 (6.3)	13.1 (6.5)	12.9 (5.9)	0.191	0.52 (−0.11 to 1.16), p=0.105
Triceps	16.6 (6.6)	17.5 (7.0)	17.3 (6.1)	0.100	0.67 (−0.004 to 1.33), p=0.051
Mid-calf	18.4 (8.2)	19.2 (7.5)	19.8 (6.8)	0.164	0.78 (−0.03 to 1.58), p=0.058
Mid-thigh	25.0 (9.80	25.4 (9.8)	27.5 (9.0)	0.178	0.79 (−0.19 to 1.77), p=0.115

Fat measurements are in mm presented as mean (SD). Linear regression model was used to assess the association of fat measurements with parental history of diabetes; values are presented as coefficient and 95% CI.

along with postprandial blood glucose levels can predict impaired glucose tolerance better than BMI and waist-to-hip ratio. Furthermore, they highlighted that a clear demarcation between transient and persistent glucose intolerance could be made by the addition of subscapular skinfold thickness measurement to the standard oral glucose tolerance test (OGTT) measurement.[14] The Amsterdam Growth and Health Longitudinal Study has shown that high body fat in middle age can be better predicted using skinfold thickness than BMI measured at baseline during adolescence.[19] One of the disadvantages of skinfold thickness measurement is that exposure and touching of the skin can produce some degree of discomfort in some subjects. The need for well-trained anthropometrists and careful fat measurement using callipers are some of the inherent disadvantages of the use of the technique in a busy clinical practice. A Japanese study by Kagawa *et al*[20] also showed that reliable patterns of fat distribution can be obtained if skinfold measurements are taken by trained technicians.

Earlier studies have shown that the association between regional fat deposits, total adiposity and type 2 diabetes is influenced by family history of diabetes.[21] Some studies have established that the association between family history of diabetes and the risk of type 2 diabetes can be largely explained by excess adiposity.[22] Further, a synergistic association between family history of diabetes and abnormal waist-to-hip ratio and the incidence of type 2 diabetes has been reported in a Chinese study.[23] Hence we sought to determine the association of family history of diabetes with adiposity measurements using skinfold thickness; total trunk and peripheral fat measurements were derived by calculating the sum of trunk and peripheral skinfolds. It was found that trunk fat increased linearly according to diabetes status and a positive parental history of diabetes, whereas peripheral fat did not show such a trend. This corroborates our previous findings that visceral fat measured using CT increased with increasing degree of glucose intolerance, whereas subcutaneous fat did not.[24]

Our study results also demonstrated a good correlation of trunk fat and peripheral fat, measured by skinfold thickness, with BMI and waist circumference. Addo *et al*[25] showed that fat estimations using skinfold measurements such as

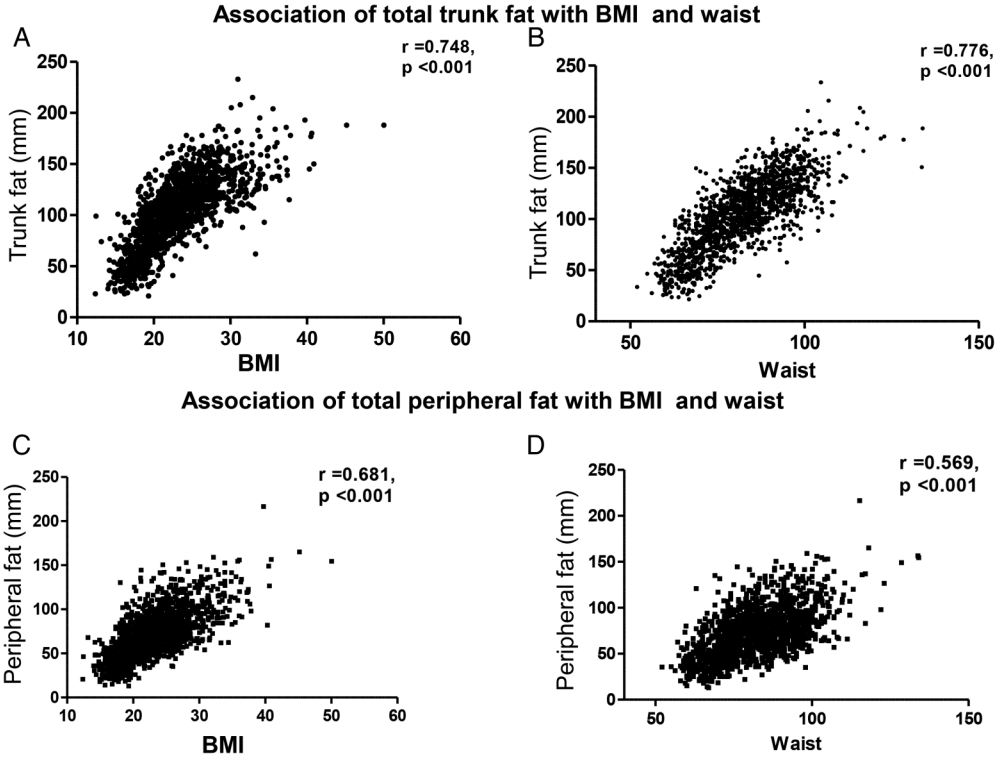

Figure 2 (A) Association of total trunk fat with body mass index (BMI) and waist circumference. (B) Association of total peripheral fat with BMI and waist circumference.

subscapular and triceps thickness correlated fairly well with estimates of body fat obtained using DEXA. These findings are in agreement with studies using other indices of central and peripheral obesity that have shown a significant association between family history of diabetes and adiposity.[26] In a 20-year longitudinal study, it was found that excess adiposity could explain a large part of the association between the risk of type 2 diabetes and family history.[22] In a population of Hispanic children with a family history of diabetes, visceral fat was found to be associated with an increased risk of type 2 diabetes.[27] The mechanistic links connecting family history and adiposity in subjects with type 2 diabetes are largely unclear. However, there are some reports that implicate the role of some genetic variations that connect family history of diabetes with adiposity.[28] It is also entirely possible that environmental factors—for example, decreased physical activity and/or unhealthy diet—may be familial and thus explain the link between family history of diabetes and adiposity index.

The study has several limitations. First, because of its cross-sectional nature, causality cannot be established. Second, while the skinfold measures show a good correlation with BMI and waist circumference in this study, we did not use gold standard measures such as DEXA. It is possible that, due to multiple testing, type 1 and type 2 errors might have resulted. However, it is unlikely that these affected the results or conclusions drawn. The strength of the study is that it includes a large sample representing urban India and that there are no such studies in our population.

Future work should focus on the association between skinfold thickness and other measurements of fat including DEXA and MRI. The skinfold measures should also be further validated separately in male and female subjects as the sexes tend to have distinct distributions of body fat.

In conclusion, a robust association between trunk fat, measured by skinfold thickness, and parental history of diabetes is reported, but no association with peripheral fat was found.

Main messages

- Trunk fat is associated with a parental history of diabetes.
- Peripheral fat is not associated with parental history of diabetes.
- Both trunk and peripheral fat are associated with other indices of obesity including body mass index and waist circumference.

Current research questions

- What is the association between skinfold thickness and gold standard measurements of fat (DEXA and MRI)?
- Is there a link between parental diabetes and abnormalities of glucose and lipid metabolism among the offspring?
- What is the place of skinfold measurements of fat in clinical practice?

Acknowledgements We thank the epidemiology team members for conducting the CURES field studies. This is the 114th publication from CURES (CURES-114).

Contributors VM conceived and designed the study. JS, KI and MD analysed the data. JS, KI and RP wrote the manuscript. VM helped revise the manuscript critically for important intellectual content and read and approved the final manuscript.

Competing interests None declared.

Ethics approval Institutional Ethics Committee, Madras Diabetes Research Foundation.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Després JP. Body fat distribution and risk of cardiovascular disease: an update. *Circulation* 2012;126:1301–13.
2 Van Pelt RE, Evans EM, Schechtman KB, *et al*. Waist circumference vs body mass index for prediction of disease risk in postmenopausal women. *Int J Obes Relat Metab Disord* 2001;25:1183–8.
3 DeNino WF, Tchernof A, Dionne IJ, *et al*. Contribution of abdominal adiposity to age-related differences in insulin sensitivity and plasma lipids in healthy nonobese women. *Diabetes Care* 2001;24:925–32.
4 Ross R, Aru J, Freeman J, *et al*. Abdominal adiposity and insulin resistance in obese men. *Am J Physiol Endocrinol Metab* 2002;282:E657–63.
5 Shah NR, Braverman ER. Measuring adiposity in patients: the utility of body mass index (BMI), percent body fat, and leptin. *PLoS ONE* 2012;7:e33308.
6 Chan DC, Watts GF, Barrett PH, *et al*. Waist circumference, waist-to-hip ratio and body mass index as predictors of adipose tissue compartments in men. *QJM* 2003;96:441–7.
7 Lohman TG. *Advances in body composition assessment, current issues in exercise science series*. Champaign, IL: Human Kinetics Publishers, 1992.
8 Smalley KJ, Knerr AN, Kendrick ZV, *et al*. Reassessment of body mass indices. *Am J Clin Nutr* 1990;52:405–8.
9 Paradisi G, Smith L, Burtner C, *et al*. Dual energy X-ray absorptiometry assessment of fat mass distribution and its association with the insulin resistance syndrome. *Diabetes Care* 1999;22:1310–17.
10 Wang J, Thornton JC, Kolesnik S, *et al*. Anthropometry in body composition. An overview. *Ann N Y Acad Sci* 2000;904:317–26.
11 Jackson AS, Pollock ML. Practical assessment of body composition. *Phys Sports Med* 1985;13:76–90.
12 Wattanapenpaiboon N, Lukito W, Strauss BJ, *et al*. Agreement of skinfold measurement and bioelectrical impedance analysis (BIA) methods with dual energy X-ray absorptiometry (DEXA) in estimating total body fat in Anglo-Celtic Australians. *Int J Obes Relat Metab Disord* 1998;22:854–60.
13 Kamimura MA, Avesani CM, Cendoroglo M, *et al*. Comparison of skinfold thicknesses and bioelectrical impedance analysis with dual-energy X-ray absorptiometry for the assessment of body fat in patients on long-term haemodialysis therapy. *Nephrol Dial Transplant* 2003;18:101–5.
14 Sosenko JM, Kato M, Soto R, *et al*. A comparison of adiposity measures for screening non-insulin dependent diabetes mellitus. *Int J Obes Relat Metab Disord* 1993;17:441–4.
15 Kelly LA, Lane CJ, Weigensberg MJ, *et al*. Parental history and risk of type 2 diabetes in overweight Latino adolescents: a longitudinal analysis. *Diabetes Care* 2007;30:2700–5.
16 Deepa M, Pradeepa R, Rema M, *et al*. The Chennai Urban Rural Epidemiology Study (CURES)—study design and methodology (urban component) (CURES-I). *J Assoc Physicians India* 2003;51:863–70.
17 Deepa M, Farooq S, Datta M, *et al*. 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.
18 Hughes JM, Li L, Chinn S, *et al*. Trends in growth in England and Scotland, 1972 to 1994. *Arch Dis Child* 1997;76:182–9.
19 Nooyens AC, Koppes LL, Visscher TL, *et al*. Adolescent skinfold thickness is a better predictor of high body fatness in adults than is body mass index: the Amsterdam Growth and Health Longitudinal Study. *Am J Clin Nutr* 2007;85:1533–9.
20 Kagawa M, Uenishi K, Mori M, *et al*. Obesity screening for young Japanese males and females using skin fold measurements: the classification revisited. *Asia Pac J Clin Nutr* 2010;19:289–93.
21 Fujimoto WY, Leonetti DL, Newell-Morris L, *et al*. Relationship of absence or presence of a family history of diabetes to body weight and body fat distribution in type 2 diabetes. *Int J Obes* 1991;15:111–20.
22 van 't Riet E, Dekker JM, Sun Q, *et al*. Role of adiposity and lifestyle in the relationship between family history of diabetes and 20-year incidence of type 2 diabetes in U.S. women. *Diabetes Care* 2010;33:763–7.
23 Liu L, Pang Z, Wang S, *et al*. Synergistic effect of diabetes family history and abnormal waist-to-hip ratio on the incidence of type 2 diabetes. *Wei Sheng Yan Jiu* 2012;41:308–10.
24 Indulekha K, Anjana RM, Surendar J, *et al*. Association of visceral and subcutaneous fat with glucose intolerance, insulin resistance, adipocytokines and inflammatory markers in Asian Indians (CURES-113). *Clin Biochem* 2011;44:281–7.

25 Addo OY, Pereira MA, Himes JH. Is skinfold thickness as good as DXA when measuring adiposity contributions to insulin resistance in adolescents? *Am J Hum Biol* 2012;24:806–11.
26 Neeland IJ, Turer AT, Ayers CR, *et al*. Dysfunctional adiposity and the risk of prediabetes and type 2 diabetes in obese adults. *JAMA* 2012;308:1150–9.
27 Cruz ML, Bergman RN, Goran MI. Unique effect of visceral fat on insulin sensitivity in obese Hispanic children with a family history of type 2 diabetes. *Diabetes Care* 2002;25:1631–6.
28 Suchindran S, Vana AM, Shaffer RA, *et al*. Racial differences in the interaction between family history and risk factors associated with diabetes in the National Health and Nutritional Examination Survey, 1999–2004. *Genet Med* 2009;11:542–7.

Association of adiposity, measured by skinfold thickness, with parental history of diabetes in a South Indian population: data from CURES-114

J Surendar, K Indulekha, M Deepa, V Mohan and R Pradeepa

Postgrad Med J published online February 25, 2016