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ORIGINAL ARTICLE

The effect of abdominal obesity on insulin sensitivity and serum lipid and cytokine concentrations in African women

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Summary

Objectives Studies have shown clear associations of abdominal obesity with lipid and glucose metabolism and cytokine levels in a number of different population groups. However, no such studies have been performed in an African population in which visceral adipose tissue levels have been shown to be lower than in European subjects.

Design and patients Cross-sectional analysis in 124 African women.

Measurements Fasting serum samples were taken from all subjects and anthropometric measurements obtained. Blood levels of glucose, insulin, total cholesterol, high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol, triglyceride, interleukin (IL)-6, IL-8 and IL-18 were measured. Subjects were separated into normal and abnormal glucose tolerant groups and into tertiles according to waist circumference (WC). Insulin resistance was assessed using the homeostasis model assessment (HOMA).

Results Abnormal glucose-tolerant subjects had higher WC, glucose and HOMA levels than the normal glucose-tolerant group. Increased WC was associated with higher triglyceride, insulin and HOMA levels and lower HDL levels. Multiple regression analyses showed that WC associated positively with HOMA and serum triglyceride levels and negatively with HDL levels. IL18 was a positive but weak determinant of the HOMA level and BMI correlated positively with serum IL-6 concentrations.

Conclusions Although previous studies have shown that African subjects have a lower visceral adipose depot size than European subjects, abdominal obesity is still associated with insulin resistance and dyslipidaemia. The association between abdominal obesity and metabolic dysfunction within this population is not dependent upon IL-6, IL-8 or IL-18.

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Introduction

Abdominal obesity has a positive relationship with insulin resistance and serum lipid levels.[1] This relationship is thought to be as a result of the metabolic effects of the visceral adipose depot which secretes a number of factors that are known to influence insulin function and lipid metabolism.[2] In particular, visceral fat releases free fatty acids (FFAs) into the portal vein and this may lead to increased hepatic lipid and glucose output and reduced insulin clearance.[3–5] However, the visceral adipose depot seems to have differential effects on insulin sensitivity in various ethnic groups. Thus, studies conducted in both South Africa and the United States have shown that Black patients have less visceral fat than body mass index (BMI)-matched White patients and yet are more insulin resistant.[6–8] It has been suggested that this demonstrates that either the visceral fat depot in Black patients is far more effective at inducing insulin resistance than in White patients or that visceral fat plays no part in the aetiology of insulin resistance.[9] Black patients also have less atherogenic lipid profiles than White patients[10–12] and it is possible that this is a direct reflection of their smaller visceral adipose depot size.

The visceral adipose depot may affect insulin sensitivity and lipid metabolism by the secretion of products other than FFAs. It is known that adipocytes secrete a number of pro-inflammatory [e.g. interleukin (IL)-6 and tumour necrosis factor alpha (TNF-α)] and anti-inflammatory (e.g. adiponectin) cytokines that demonstrate a negative and positive relationship with insulin sensitivity, respectively.[13] A number of these cytokines have been shown to correlate positively with measures of visceral adiposity[14–16] and to be secreted in larger quantities from visceral adipocytes than adipocytes isolated from other fat depots.[17,18] Recently, IL-8 and IL-18 have been added to this list of adipocyte-derived cytokines. The serum levels of both these cytokines have been shown to correlate positively with abdominal obesity and negatively with insulin sensitivity.[15,16,19]

The purpose of the present study was to investigate the relationship of a simple measure of abdominal obesity, i.e. waist circumference (WC) to insulin sensitivity and serum lipid concentrations in

African women; only women were studied because a previous investigation has demonstrated that Black South African women have less visceral fat than White women and yet are more insulin resistant.[8] Additionally, the relationship between WC and systemic levels of the cytokines IL-6, IL-8 and IL-18 was analysed to determine whether concentrations of these cytokines are influenced by abdominal obesity within this population group. The relationship of serum concentrations of these three molecules with insulin sensitivity was also determined.

Materials and methods

A group of 124 African women volunteered to take part in this study. The subjects were attending the Mamelodi hypertension clinic, close to Pretoria. The subjects represented 124 consecutive patients, who agreed to take part in the study. Patients with known diabetes mellitus were excluded from the study. The study was approved by the Human Ethics Committee of the University of Pretoria and each subject gave informed consent prior to entry into the study.

Weight was measured to the nearest 0·1 kg with subjects standing barefoot in light clothing, using a calibrated balance-beam scale. Height was measured to the nearest 0·1 cm. Waist to hip ratio (WHR) was measured by taking WC as the smallest diameter between the xyphoid and the umbilicus and hip circumference as the widest circumference of the buttock. Two measurements were taken and if there was more than a 2-cm difference between the two, a third was taken. The mean of the two measurements or the closest two of three were used for calculating the WHR. Blood pressure was measured using a Baumanometer with the subjects sitting after at least 5 min rest. Two measurements were taken, 1 min between each. If the readings were more than 5 mmHg different, then a third was taken and the mean of the closest two readings was used. Information regarding medications taken by the patient was obtained verbally and by consulting the patient's medical records.

Blood samples were taken from a cubital fossa vein following an overnight fast. Blood was centrifuged within 2 h and immediately assayed for glucose, insulin, total cholesterol, HDL cholesterol, LDL cholesterol and triglyceride. Serum was stored at – 80 °C and assayed for IL-6, IL-8 and IL-18. Insulin resistance was measured using the homeostasis model assessment method (HOMA).[20]

Glucose was measured using a Synchron CX delta autoanalyser (Beckman, Fullerton, CA, USA) via an oxygen rate method (Beckman). Insulin levels were assessed using a radioimmunoassay (Pharmacia, New York, NY, USA) that cross-reacts 41% with proinsulin. Total cholesterol was assayed using an automated Technicon DAX analyser. The HDL cholesterol concentration was assayed using a heparin-manganese precipitation method (Merck, Whitehouse Station, NJ, USA), whereas LDL cholesterol was measured using a differential precipitation method (Merck). Triglyceride levels were measured using the Synchron CX system. IL levels were assayed using commercial kits from R & D Systems (Minneapolis, MN): Human IL-8 Quantiglo high-sensitivity ELISA; Human IL-6 Quantikine high sensitivity enzyme-linked immunosorbent assay (ELISA); Human IL-18 ELISA. The coefficients of variation (CVs) for the cytokine assays were 5·4%, 7·8% and 4·9%, respectively.

Patients were diagnosed as diabetic or as having impaired fasting glucose levels using the WHO criteria, i.e. fasting glucose ≥ 7·0 mmol/l and ≥ 6·1 mmol/l, respectively.[21] Individuals diagnosed with type 2 diabetes and impaired fasting glucose were grouped together as abnormal glucose-tolerant group and compared with normal glucose-tolerant subjects. Menopausal status was defined by using an age cut-off of 50 years.[22]

The total population of 124 women were split into tertiles according to WHR and WC. Cytokine levels were measured in the top and bottom WHR tertiles only but there was not sufficient serum to measure cytokine concentrations in all these subjects. Thus, IL-18 was measured in 81 subjects, whereas IL-8 and IL-6 were measured in 78 and 70, respectively, of the subjects in whom IL-18 was measured. All other blood analytes were measured in all study participants.

Statistical analysis

Nonparametric data were log transformed to normality and is expressed in text and tables as median [interquartile range], whereas data displaying a normal distribution are expressed as mean ± standard deviation. Variables were compared between normal and abnormal glucose-tolerant groups using ANCOVA adjusted for age (Table 1), whereas differences between WC tertiles were analysed using ANCOVA adjusted for age and BMI (Table 2). Nonparametric data was log transformed before ANCOVA or multiple regression analyses were carried out. Multiple regression analyses were performed and nonstandardized β coefficients are given in the text and in Table 3.

The study population was split into tertiles of WC and WHR. The data in Table 2 show results for subjects in WC tertiles only. This method of data presentation was used in preference to WHR tertiles because WC is thought to be a better method of assessing visceral fat mass than WHR.[23–26]

Sample size calculation was performed on the cytokine data assuming a power of 80% and an α of 0·05, to determine differences of 1 standard deviation on log-transformed variables in two or three subject groups with standard deviations (SD) between 0·8 and 1·5. The resulting sample sizes varied between 17 and 22 subjects per group.

Results

The total cohort of 124 subjects had a median BMI of 32·9 with minimum and maximum values of 17·1 and 59·2, respectively. The median value and range for WC were 90·8 and 64·5–121 cm, respectively, whereas for HOMA the median and range were 2·60 and 0·98–13·00, respectively.

All subjects in this study were hypertensive and were receiving antihypertensive treatment. Thus, 97·6% of subjects were taking thiazide diuretics and 39·5% were taking angiotensinogen-converting enzyme (ACE) inhibitors. This latter group had similar anthropometric and insulin, glucose and IL measurements when compared to the nontreated group, but had higher LDL ($3{\cdot}92 \pm 1{\cdot}21$ *vs.* $3{\cdot}36 \pm 0{\cdot}92$ mmol/l; $P < 0{\cdot}01$) and lower HDL ($1{\cdot}10 \pm 0{\cdot}28$ *vs.* $1{\cdot}22 \pm 0{\cdot}33$ mmol/l; $P < 0{\cdot}05$) blood levels than the non-ACE inhibitor treated subjects. Adjusting for ACE inhibitor treatment or the daily dose of thiazide diuretic had no significant effect on the results obtained from ANCOVA or multiple regression analyses. Neither the

Table 1. Anthropometric and metabolic characteristics of abnormal and normal glucose tolerant subjects

Variables	Normal glucose tolerance group ($N = 94$)	Abnormal glucose tolerance group ($N = 27$)
Age (years)	56 ± 11	59 ± 10
BMI	33 ± 8	37 ± 6
Waist (cm)	90 ± 11	95 ± 9*
WHR	0·80 ± 0·07	0·82 ± 0·07
Total cholesterol (mmol/l)	5·24 ± 1·05	5·20 ± 1·10
LDL (mmol/l)	3·57 ± 1·09	3·51 ± 1·03
HDL (mmol/l)	1·17 ± 0·34	1·16 ± 0·26
Triglycerides (mmol/l)	1·39 [0·72]	1·44 [1·18]
Glucose (mmol/l)	5·20 [0·60]	6·60 [1·20]**
Insulin (pmol/l)	76 [34]	71 [42]
HOMA	2·51 [1·15]	3·56 [2·06]**
IL-6 (pg/ml)†	2·03 [2·23]	2·41 [2·32]
IL-8 (pg/ml)†	60 [39]	50 [37]
IL-18 (pg/ml)†	46 [37]	47 [30]

Data are expressed as mean ± SD or median [interquartile range]. †N numbers for IL-6, IL-8 and IL-18 in normal glucose tolerant group are 51, 58 and 59, respectively, and in abnormal glucose tolerant group are 18, 20 and 20, respectively; *$P < 0·05$, **$P < 0·0001$ (ANCOVA adjusted for age).

Table 2. Anthropometric and metabolic variables for each waist circumference tertile

Variables	Tertile 1 ($N = 38$–42)†	Tertile 2 ($N = 41$)†	Tertile 3 ($N = 40$–41)†
Age (years)	55 ± 11	59 ± 10	56 ± 10
BMI	27 ± 5	33 ± 4	41 ± 8***
Waist (cm)	79 ± 5	91 ± 3	103 ± 7***
WHR	0·77 ± 0·07	0·81 ± 0·06	0·84 ± 0·14**
Total cholesterol (mmol/l)	5·22 ± 1·22	5·31 ± 1·00	5·24 ± 0·95
LDL (mmol/l)	3·43 ± 1·22	3·77 ± 1·03	3·55 ± 0·95
HDL (mmol/l)	1·29 ± 0·38	1·09 ± 0·25	1·11 ± 0·27*
Triglycerides (mmol/l)	1·07 [0·53]	1·55 [0·49]	1·44 [0·73]**
Glucose (mmol/l)	5·40 [0·90]	5·50 [1·20]	5·50 [1·10]
Insulin (pmol/l)	65 [35]	77 [26]	78 [29]*
HOMA	1·97 [1·44]	2·81 [1·02]	2·66 [1·29]*
IL-6 (pg/ml)	2·19 [2·04]	2·11 [2·10]	2·87 [2·92]
IL-8 (pg/ml)	68 [31]	71 [34]	57 [39]
IL-18 (pg/ml)	44 [33]	49 [29]	68 [67]

Data are expressed as mean ± SD or median [interquartile range]; †N numbers for interleukins are different and are given in the text; *$P < 0·05$, **$P < 0·0005$, ***$P < 0·0001$ for trends (ANCOVA adjusted for age and BMI).

thiazide dose nor the number of subjects receiving ACE inhibitors differed across the tertiles of WC or WHR. Furthermore, ANOVA showed no relationship between the daily thiazide dose and any of the metabolic variables (data not shown). Neither systolic nor diastolic blood pressures were found to correlate with any of the measured blood analytes. None of the study participants were receiving lipid-lowering agents.

Fourteen subjects (11·3%) were receiving oestrogen replacement therapy, which showed no effect on any of the measured anthropometric or metabolic variables. The study included 95 (76·6%) post-menopausal and 29 (23·4%) premenopausal subjects, with menopausal status assigned using an age cut-off of 50 years. Total cholesterol (5·41 ± 1·07 *vs.* 4·75 ± 0·84 mmol/l; $P < 0·005$), LDL (3·71 ± 1·11 *vs.* 3·11 ± 0·79 mmol/l; $P < 0·01$) and glucose (5·50 [0·95] *vs.* 5·30 [1·00] mmol/l; $P < 0·05$) levels were higher in the post-menopausal group. Age was therefore included as an independent variable in all ANCOVA and multiple regression analyses but its inclusion had very little effect on the outcomes.

The subjects in whom one or more cytokines were measured ($N = 81$) did not differ significantly ($P > 0·05$) from those in whom cytokines were not measured ($N = 43$) in terms of age, BMI, WC, HOMA, HDL, LDL, total cholesterol and triglyceride levels. When subjects were divided into groups based on WC tertiles, tertile 1 (lowest tertile) contained 20 subjects in whom IL-6 was measured, 24 in

Table 3. Multiple regression analyses

Model number	Dependent variable	Independent variables	β coefficient (*P* value)	Adjusted R^2 (*P* value)
1	HOMA	Age	0·003 (0·10)	0·14 (< 0·0001)
(*N* = 120)		Waist	0·006 (< 0·0001)	
2	HOMA	Age	–0·00 (0·61)	0·15 (0·002)
(*N* = 79)		Waist	0·005 (0·004)	
		IL-18	0·19 (0·02)	
3	Triglyceride	Age	0·002 (0·11)	0·05 (0·02)
(*N* = 122)		Waist	0·003 (0·02)	
4	HDL	Age	0·004 (0·09)	0·07 (0·004)
(*N* = 124)		Waist	–0·007 (0·004)	
5	IL-6	Age	0·006 (0·10)	0·10 (0·009)
(*N* = 70)		BMI	0·45 (0·01)	

BMI, HOMA, triglyceride, IL-18 and IL-6-values were log-transformed to normality.

whom IL-8 was measured and 26 in whom IL-18 was measured. In tertile 2 the numbers were 23, 24 and 24, respectively, and 27, 30 and 31, respectively in tertile 3. When subjects were divided into WHR tertiles, cytokine levels were measured only in subjects in tertiles 1 and 3 (see Methods section).

The study group was found to include 11 patients with undiagnosed type 2 diabetes and 16 subjects with impaired fasting glucose. These two patient groups differed only in terms of fasting glucose (8·98 [1·5] *vs.* 6·39 [0·25] mmol/l, respectively; $P < 0·001$) and HOMA levels (5·74 [4·83] *vs.* 3·13 [1·46], respectively; $P < 0·005$) and were grouped together as being abnormal glucose tolerant and compared with the normal glucose-tolerant subjects (Table 1). These two subject groups differed with regard to fasting glucose and HOMA levels and WC but not WHR (see Table 1). There were three subjects for whom fasting glucose levels were not available.

Five newly diagnosed diabetic subjects were found in the top WC tertile, five more in tertile 2 and one in tertile 1. Sixteen subjects with impaired fasting glucose were found, 3 in tertile 1, 7 in tertile 2 and 6 in tertile 3. Thus, in tertile 1, 4 of 39 (10·3%) subjects had abnormal glucose tolerance whereas in tertiles 2 and 3 combined, 23 of 82 subjects (28·0%; $P = 0·028$ *vs.* tertile 1) had abnormal glucose tolerance. Six newly diagnosed diabetic subjects were found in the top WHR tertile, 2 more in tertile 2 and 3 in tertile 1. Six subjects with impaired fasting glucose were found in tertile 1, 4 in tertile 2 and 6 in tertile 3. No significant difference was found for the incidence of abnormal glucose tolerance in WHR tertile 1 (21·9%) when compared to WHR tertiles 2 and 3 combined (22·5%).

Comparisons between the WC tertiles (Table 2) demonstrated a positive trend for insulin, HOMA and triglyceride levels, whereas serum HDL cholesterol concentrations displayed a negative trend. No differences between the WC tertiles were noted for any of the cytokines ($P = 0·61$ for log IL-6, 0·45 for log IL-8, 0·81 for log IL-18). Similar results were observed when data were analysed in the WHR tertiles, the only difference being a trend for glucose levels to increase with increasing WHR ($P = 0·03$ for trend). Removing the abnormal glucose-tolerant subjects from the statistical analyses comparing means between the tertiles (WC and WHR) did not affect the outcomes.

Multiple regression analyses showed that WC correlated with HOMA (model 1, Table 3). Removal of WC from the multiple regression model and analysis of the fall in the adjusted R^2 value demonstrated that WC explained 12·2%, whereas BMI explained 4·9% of the variance in HOMA. A similar analysis showed that WHR explained 6·5% of the variance in HOMA. IL-18 also influenced HOMA levels (model 2, Table 3), explaining 4·8% of its variance. However, this relationship was influenced by two outlying IL-18 values (269 pg/ml and 324 pg/ml) which, when removed from the regression model, caused the β-coefficient for IL-18 to drop from 0·19 ($P = 0·02$) to 0·12 ($P = 0·18$). WC was also a determinant of triglyceride (model 3, Table 3) and HDL (model 4, Table 3) levels explaining 3·6% and 5·9% of their variation, respectively. Similar results were obtained for WHR. IL-6 did not correlate with HOMA, WC or WHR, but did correlate with BMI (model 5, Table 3), which explained 7·8% of the variation in IL-6 levels.

Discussion

Studies performed in North America and South Africa have shown that Black patients are more insulin resistant and yet have less visceral fat than BMI-matched White patients.[6–8,27] Furthermore, in BMI-matched Black and White subjects with similar WC[8] or WHR,[28] insulin resistance was higher in the former group. The present study demonstrates that within Black women, increased abdominal obesity leads to higher levels of insulin resistance. Therefore, although abdominal adiposity is lower in Africans than in Europeans, it still influences insulin sensitivity.

This study used WC as a measure of abdominal obesity and consequently, it is not possible to conclusively demonstrate that the visceral fat depot contributes to insulin resistance or any of the other metabolic parameters that were analysed. However, a number of studies have shown that visceral fat depot size is correlated to the level of insulin resistance[1–3] and a recent study has shown that removal of subcutaneous abdominal adipose tissue via liposuction has no effect on insulin sensitivity.[29] Studies have also shown that WC is a good measure of visceral fat depot size[23–26] and furthermore, WC and visceral adiposity are lower in Black, compared to White South Africans, but subcutaneous abdominal fat levels are equivalent.[30]

Subjects with abnormal glucose tolerance were found to have a higher WC, but not WHR than normal glucose-tolerant subjects and were found at a higher frequency in the top two than the bottom tertiles for WC. This was not the case for WHR tertiles. WC correlated more strongly with HOMA than did WHR. This confirms results from studies showing that increased WC is a risk factor for type 2 diabetes[31,32] and studies showing that patients with type 2 diabetes have a higher visceral adipose depot size than nondiabetic patients.[9,33,34] This data also suggest that WC is a better anthropometric marker than WHR for type 2 diabetes and insulin resistance. This may be related to WC being a better indicator of visceral fat mass than WHR.[23–26]

No differences were observed between the normal and abnormal glucose-tolerant groups for fasting cytokine levels, confirming data for IL-6 and IL-8 from previous studies.[35,36] However, other studies have shown that IL-8[37,38] and IL-18[38,39] concentrations are higher in

patients with type 2 diabetes than in nondiabetic patients. In the present study, although the sample sizes for IL-6, IL-8 and IL-18 were 18, 20 and 20, respectively, for the abnormal glucose-tolerant subjects, these sample sizes were still adequate to detect at least a 1-SD difference between mean values. Additionally, the normal glucose-tolerant group had a mean BMI of 33 ± 8, suggesting some degree of insulin resistance. This cannot explain the lack of difference in cytokine levels between the two subject groups as the abnormal glucose tolerant group did have higher WC and HOMA than the normal glucose-tolerant group but in the absence of differences in cytokine levels.

Insulin resistance, as measured using HOMA, correlated positively and more strongly with WC than with BMI. This confirms data from other studies showing that WC is a strong predictor of insulin resistance, independently of BMI.[40–42] Visceral adiposity may explain the relationship between WC and insulin sensitivity; however, the process involved is not fully understood. One possible mechanism may be the high release of FFA into the portal circulation by visceral adipocytes[3–5] and also the secretion of adipokines that modify insulin sensitivity. Thus, studies have shown that visceral adipocytes secrete higher levels of IL-6 and IL-8 than subcutaneous adipocytes[17,18] and both these factors have been shown to negatively influence insulin sensitivity.[15,19] Furthermore, serum levels of IL-6 have been shown to be predictive of the future development of type 2 diabetes.[43] IL-18 has also been shown to correlate with the level of insulin resistance and is positively associated with increased visceral fat deposition.[16,44,45] However, the present study could find no correlation between these cytokines and WC, but a marginal, positive association was observed between IL-18 levels and HOMA. It is interesting to note that although earlier studies suggested IL-6 may influence insulin sensitivity, a more recent investigation has shown that this may not be the case.[36] In the latter study, serum IL-6 levels correlated only with BMI and were not higher in subjects with type 2 diabetes. The present study confirms these findings.

Although the median BMI value for this population was high, the range of BMIs was extensive. This was also true for WC and HOMA levels. This suggests that the low correlation between BMI and HOMA was not the result of a narrow range of values and likewise for the poor correlations of BMI and WC with cytokine concentrations. The cohort of subjects studied was not atypical in regard to anthropometric variables when compared to the Black, urban female population in general. Thus, a national survey has shown that the highest prevalence of obesity is found within middle-aged Black South African women[46] and two studies performed in Johannesburg using randomly selected Black women demonstrated mean BMIs of 33·0 ± 7·2 ($N = 93$)[47] and 31·8 ± 7·1 ($N = 66$).[48]

Insulin resistance was measured in this study using HOMA. This method provides a measure of insulin sensitivity that correlates well with the hyperinsulinaemic, euglycaemic clamp technique,[20,49] and has been used to assess insulin resistance in a wide variety of population groups. Furthermore, a previous study using the hyperinsulinaemic, euglycaemic clamp has shown that Black South African subjects are more insulin resistant than White subjects,[8] and this has been confirmed in a subsequent study using HOMA.[28]

In this study, WC correlated positively with serum triglyceride levels and there was a tendency for HDL cholesterol concentrations to fall with increasing WC. These findings are similar to those from other studies[1,3,50] and suggest that abdominal obesity may negatively influence hepatic lipid metabolism in this population group.

All subjects in this study were hypertensive and were receiving therapy for raised blood pressure. Certain antihypertensive agents are known to lead to increased lipid levels[51] and within our study, subjects taking ACE inhibitors did have higher LDL and lower HDL serum levels than subjects not receiving this therapy. However, adjusting for ACE inhibitor use did not affect any of the results obtained from multiple group comparisons or regression analyses. One study has shown that 12-week treatment with hydrochlorothiazide has no effect on fasting insulin or glucose levels, leads to a small but insignificant increase in postprandial insulin and glucose concentrations, but does cause a small, but significant ($P < 0{\cdot}01$) increase in haemoglobin A1c levels.[52] In the present investigation, statistical adjustments for the daily dose of diuretics had no significant effect on the study results and no relationship was observed between diuretic dose and any of the metabolic variables. Thus, antihypertensive therapy did not confound the results of this investigation.

Angiotensin-II does have pro-inflammatory actions and it has therefore been suggested that ACE inhibitors may be anti-inflammatory agents.[53] However, the present study did not observe any differences in blood cytokine levels between subjects receiving or not receiving ACE inhibitor therapy. Thiazide diuretics have also been studied for their effect on the inflammatory process, but investigations have not been able to demonstrate any significant effects.[54,55] Thus, the antihypertensive drugs used in the present investigation did not affect the cytokine levels of the study subjects and adjusting for the dosage levels of thiazides or for the use of ACE inhibitors did not affect results from any of the statistical analyses.

A recent study has demonstrated that subjects with high IL-6 serum levels have an increased risk of hypertension.[56] Investigations have also shown that hypertensive patients are more insulin resistant than normotensive subjects.[57,58] It is therefore possible that our cohort of subjects represents a population with abnormal metabolic function and that the data obtained from this study cannot be used to make conclusions in normotensive subjects. However, the results from the present investigation confirm those from many other studies,[1,9,16,33–36,50] suggesting that our results are not confined to hypertensive patients.

The present study demonstrates that abdominal obesity influences insulin sensitivity in African women, despite the reported lower level of visceral fat in Black compared to White subjects.[6,7,27] However, the cytokines IL-6, IL-8 and IL-18 were found not to correlate with any measure of abdominal obesity and only IL-18 was found to be associated with insulin sensitivity. IL-6 was observed to correlate with BMI. Abdominal obesity in these subjects was also associated with higher fasting triglyceride levels and lower HDL levels. Thus, abdominal obesity in Black women is associated with insulin resistance and dyslipidaemia, but these relationships do not depend upon the serum levels of IL-6, IL-8 or IL-18.

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