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Central and peripheral fat and subclinical vascular damage in older women

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Abstract

Objective: the aim of this study was to evaluate the relationship between fat distribution and arterial compliance in a group of elderly women, in particular to test a possible independent role of abdominal fat mass and peripheral fat mass on subclinical vascular damage, defined by a pulse wave velocity (PWV) >12 m/s.

Methods: in 96 women with age range 60–80 years (68.65 ± 4.98 years) and BMI range from 18.8 to 41.2 kg/m² (27.07 ± 4.61 kg/m²), we evaluated the body mass index, waist circumference, systolic and diastolic blood pressure, fasting glucose, cholesterol, LDL and HDL cholesterol, triglycerides and body composition by dual energy X-ray absorptiometry (DXA). Arterial stiffness was assessed by carotid-femoral (PWVcf) and carotid-radial pulse wave velocity (PWVcr).

Results: significant associations were found between PWVcf, age, waist circumference, BMI and trunk fat assessed by DXA, as well as TG and HDL cholesterol. After adjustment for the total fat mass a negative statistically significant association between PWVcf and leg fat mass was shown. In multiple regression analyses the mean arterial pressure, trunk fat

359
mass and leg fat mass were significant predictors of vascular damage with OR, respectively, of 1.06 (CI: 1.01–1.11), 1.25 (CI: 1.06–1.48) and 0.73 (CI: 0.53–0.99).

**Conclusions:** The results of this study show, in a sample of apparently healthy elderly women, that central and peripheral adiposity are independent predictors, with an opposite effect on subclinical vascular damage, confirming and strengthening the protective role of the gluteal-femoral fat on cardiovascular risk even in elderly.

**Keywords:** fat distribution, subclinical vascular damage, pulse wave velocity, elderly, older people

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**Introduction**

Ageing is associated with a progressive increase in the fat mass, in particular visceral adipose tissue, and a decrease in the peripheral subcutaneous adipose tissue [1, 2].

Traditional cardiovascular risk factors are able to affect the structural and functional characteristics of arteries by reducing their compliance [3, 4]. Several studies showed a significant association between visceral adiposity and arterial stiffness [5]. The relationship between lean body mass, fat mass and central and peripheral atherosclerosis was investigated in the ‘Amsterdam Growth and Health Longitudinal Study (AGAHLS)’ in 336 young, healthy and not obese adults [6]. The authors showed that visceral fat was negatively correlated with carotid and femoral artery distensibility and with femoral artery compliance and that both the peripheral fat mass and the lean body mass were inversely associated with carotid-femoral pulse wave velocity (PWV).

From a clinical point of view the peripheral fat mass would play a protective role on cardiovascular risk and haemodynamic-metabolic obesity would play a protective role on cardiovascular risk and haemodynamic-metabolic obesity’s complications.

Only few studies investigated the relations between arterial stiffness and peripheral fat mass in older persons. Snijder et al. [7] in the HOORN study, studying a sample of healthy elderly, observed a positive correlation between indices of peripheral stiffness and fat mass of the trunk. The lean mass and the leg fat mass, instead, were negatively related to arterial stiffness.

The aim of this study was to evaluate the relationship between fat distribution and arterial compliance in a group of women between 60 and 80 years old. In addition, we investigated a possible independent role of the abdominal fat mass and the peripheral fat mass in determining subclinical vascular damage, defined by a PWV >12 m/s, as stated in the ESH/ESC guidelines for hypertension [8].

**Methods**

**Subjects**

Ninety-six women with age range 60–80 years (68.65 ± 4.98 years) and BMI range from 18.8 to 41.2 kg/m² (27.07 ± 4.61 kg/m²) took part in the study.

Subjects were recruited from the lists of a few general practitioners in Verona and were eligible if they were post-menopausal women free of known CV diseases and did not assume any medications affecting the arterial compliance, such as antihypertensive, statin treatment and hormone replacement therapy.

All subjects had stable weight during the past 6 months. None of the subjects was affected by diabetes, cancer, or liver, renal or thyroid disease, and had no evidence of cardiovascular disease (CVD) but hypertension diagnosed after our visit but not known at recruitment. Patients with renal impairment (serum-creatinine >120 µmol/l) were excluded. All subjects were non-smokers. The study was approved by the ethical committee of the University of Verona. All subjects underwent a careful clinical assessment before the study. Twenty-one per cent of the study population was hypertensive, 27% had hypercholesterolaemia.

**Anthropometric variables**

With the subject barefoot and wearing light indoor clothing, body weight was measured to the nearest 0.1 kg (Salus scale, Milan, Italy), and height to the nearest 0.5 cm using a stadiometer (Salus stadiometer, Milan, Italy). The BMI was calculated as body weight adjusted by stature (kg/m²). Waist circumference was obtained with a measuring tape at the level of the narrowest part of the torso as viewed anteriorly.

**Blood pressure and arterial stiffness measurements**

Brachial blood pressure (BP) was measured three times in a time frame of 15 min using a mercury sphygmomanometer in the left arm of the subject, in the supine position. The mean of three readings was considered as the subject’s BP. The BP was recorded immediately prior to tonometric recording. Subject conditions were standardised according to established guidelines: all subjects were fasting, and all subjects had refrained from consumption of alcohol or caffeine for the preceding 24 h. The mean arterial pressure (MAP) was calculated by adding one-third of the pulse pressure (systolic minus diastolic) to the diastolic pressure.

**Arterial stiffness measurements**

PWV, as a measure of arterial stiffness, was measured with PulsePen (Diatecne, Milan, Italy), a non-invasive portable device [9]. Its software provides central aortic pressure...
values, an assessment of arterial pulse wave contours, an estimation of reflection waves and measurements of PWV. PulsePen determines the PWV by a single probe placed at two sites in rapid succession, using the ECG trace as a reference. The operator starts by positioning the probe at the common carotid artery, the central detection site, while simultaneously performing ECG. Then, the same procedure is followed for the femoral artery, in aortic PWV, and for the radial artery in the carotid-radial pulse wave velocity. When the difference between heart rate recorded during the carotid measurement and that recorded during the femoral or radial measurement was ≥10%, the PWV measurement was repeated. The difference in the heart rate is indicated by the PulsePen software.

The PWV was calculated as distance between the measurement sites divided by a transit time delay between femoral and carotid pulse wave and expressed in meters per second.

The distance of the pulse wave transit was the difference between the distance from the suprasternal notch to femoral point of application of the tonometer and the distance from carotid point of tonometer application and the suprasternal notch. The time delay was measured between the feet of the peripheral artery (femoral or radial) and carotid waveforms. The foot of the wave is defined at the end of diastole, when the steep rise of the waveform begins. The duration of the examination was generally of 10–15 min for each patient.

Coefficient of variation for carotid-femoral pulse wave velocity (PWVcf) measured by PulsePen device in our laboratory was 6.7%. It was obtained by double evaluation in 25 participants.

Biochemical analyses

Venous blood samples for all metabolic assessments were obtained after the subjects fasted overnight. Plasma glucose was measured with a glucose analyzer (Beckman Instruments, Inc., Palo Alto, CA, USA). The intra-assay CV was 1.5%. Cholesterol and triglycerides concentrations were determined with an automated enzymatic method (Auto analyzer; Technicon, Tarrytown, NY, USA) [10, 11]. High-density lipoprotein (HDL) cholesterol was measured by using the method of Wärnich and Albers [12]. LDL cholesterol was calculated using the Friedwald formula [13]. HbA1c was measured by high-performance liquid chromatography (Bio-Rad Diagnostics Group, Hercules, CA, USA).

Dual energy X-ray absorptiometry

Total body fat, trunk and appendicular, leg and total fat free mass (FFM) were determined using dual energy X-ray absorptiometry (DXA) Hologic QDR 4500 fan beam densitometer with software version 8.21. The characteristics and physical concepts of DXA measurements have been described elsewhere [14]. All metal objects (jewellery, snaps and belts) were removed. Measurements were taken with the subject positioned supine on the scanning table. Radiation exposure was <8 mSv and the mean measurement time was 6 min. Daily quality-assurance tests were performed according to the manufacturer’s instructions. All scans were subsequently analysed by a single-trained investigator. The percentage of fat was calculated as the fat mass (kg) measured by DXA divided by body weight (kg) measured by scale. The fat mass of the trunk and the sum of right and left (leg fat mass) legs were obtained by using subregions option of the software. The trunk region consists of the area bordered by a horizontal line below the chin, vertical borders lateral to the ribs and oblique lines passing through the femoral necks.

Statistical analyses

Results are shown as mean ± SD. Variables not normally distributed were log-transformed before analysis. Pearson’s correlations were used to test the relationship between the variables. The study population was divided into four groups according to body fat distribution. Group 1 included subjects with the leg fat mass higher than 50th percentile and the trunk fat mass lower than the 50th percentile, Group 2 included subjects with the fat mass of the leg and trunk lower than 50th percentile. Group 3 included subjects with the fat mass of both trunk and leg higher than the 50th percentile, Group 4 included people with trunk fat mass higher than the 50th percentile and leg fat mass lower than the 50th percentile. The analysis of variance (ANOVA) was used to compare mean values of PWVcf of the four groups. A LSD post hoc analysis was used to evaluate the differences between four groups. To adjust for MAP PWVcf values, the covariance analysis (ANCOVA) was used.

A stepwise multiple regression model was used to evaluate the joint effect of independent variables on PWVcf. Age, MAP total fat mass, fat mass of the trunk, fat mass of the leg, HDL cholesterol and triglycerides were considered independent variables, PWVcf. A binary logistic regression model was used to evaluate the joint effect of independent variables on subclinical cardiovascular damage, defined as values PWVcf >12 m/s. In this model age, trunk fat mass, leg fat mass and MAP were considered independent variables.

A P-value <0.05 was considered significant. All analyses were performed using SPSS statistical program (version 16.0 for Windows).

Results

The main characteristics of the study population are shown in Table 1. Ninety-six women, mean age 68.65 ± 4.98 and mean BMI of 27.07 ± 4.61 kg/m², were evaluated. Twenty-one per cent of subjects were hypertensive (mean BP >140/90 mmHg), 27.1% hypercholesterolaemic (total
compared with those in Group 1 (respectively, 118.94 ± 44.4 and 56.60 ± 13.75 versus 68.78 ± 13.51, HDL cholesterol was lower in subjects of Groups 3 and 4.

jects in Group 1 than in those in Groups 3 and 4 (respect-

Figure 1 shows the mean values of PWVcf stratiﬁed by

Table 1. Characteristics of the study sample

<table>
<thead>
<tr>
<th></th>
<th>n = 96</th>
<th>Mean ± SD</th>
<th>Range</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>68.65 ± 4.98</td>
<td>60–80</td>
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<tr>
<td>Weight (kg)</td>
<td>66.89 ± 11.34</td>
<td>37–103.20</td>
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<tr>
<td>BMI (kg/m²)</td>
<td>27.07 ± 4.61</td>
<td>18.83–41.19</td>
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<tr>
<td>Waist circumference (cm)</td>
<td>84.78 ± 10.46</td>
<td>68–111</td>
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<td>FFM (kg)</td>
<td>39.64 ± 50.84</td>
<td>22.52–59.15</td>
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<tr>
<td>FM total (kg)</td>
<td>24.21 ± 68.43</td>
<td>11.36–39.53</td>
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<tr>
<td>FM trunk (kg)</td>
<td>11.63 ± 4.46</td>
<td>4.66–30.89</td>
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<tr>
<td>Leg FM (kg)</td>
<td>8.55 ± 2.56</td>
<td>3.69–15.63</td>
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<tr>
<td>Glucose (mg/dl)</td>
<td>90.48 ± 11.37</td>
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<tr>
<td>Total cholesterol (mg/dl)</td>
<td>223.38 ± 35.80</td>
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<td>HDL cholesterol (mg/dl)</td>
<td>61.05 ± 16.86</td>
<td>32–135</td>
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<td>LDL cholesterol (mg/dl)</td>
<td>140.74 ± 32.15</td>
<td>70–221</td>
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<td>Triglycerides (mg/dl)</td>
<td>108.29 ± 52.62</td>
<td>31–322</td>
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<td>HbA1c (%)</td>
<td>5.77 ± 0.32</td>
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<td>SBP (mmHg)</td>
<td>143.62 ± 20.62</td>
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<td>MAP (mmHg)</td>
<td>102.98 ± 11.87</td>
<td>74.33–136.33</td>
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<td>DBP (mmHg)</td>
<td>82.28 ± 9.16</td>
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<tr>
<td>PWVcf (m/s)</td>
<td>11.00 ± 2.71</td>
<td>6.22–22.56</td>
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<tr>
<td>PWVcr (m/s)</td>
<td>10.52 ± 2.38</td>
<td>6.22–22.56</td>
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Discussion

Our study showed that the high-trunk fat mass and the leg fat mass were, respectively, positively and negatively associated with subclival vascular damage as indicated by PWVcf >12 m/s in older women, suggesting a protective role of peripheral fat on subclival vascular damage.

It has been shown that ageing is associated with body fat distribution changes, i.e. visceral fat increase and subcutaneous fat decrease (in particular in thighs and calves). Accumulation of intra-abdominal fat even without body weight increase seems to persist even in old stable weight men and women [15].

Age-related body fat distribution has been shown to be linked to a higher risk of metabolic syndrome, diabetes and CVDs [1,16].

Scuteri et al. [17] reported increased arterial stiffness, as assessed by the stiffness index, in subjects with metabolic syndrome across all age groups; Schillaci et al. [18] showed an increased PWVcf in adult hypertensive subjects with metabolic syndrome.

Association between central adiposity, in particular visceral fat, and arterial stiffness has been reported in young [6] and in old subjects [19,20].

We previously reported signiﬁcant association between trunk fat, evaluated by DXA, and PWVcf in a group of elderly men and women with and without metabolic syndrome [20].

A few studies showed, respectively, positive and negative association between trunk fat and PWVcf.

Ferreira et al. showed in a young population (mean age 36 years) a positive association between arterial stiffness and trunk fat and a negative relation between peripheral fat, lean mass and arterial stiffness evaluated by carotid-femoral PWV. Snijder et al., in a sample of healthy elderly, showed a positive correlation between indices of peripheral stiffness, such as distensibility coefﬁcient of femoral, carotid and brachial arteries, but not of central arterial stiffness (systemic arterial compliance) and fat mass of the trunk, and a negative correlation between them and fat mass of the legs.
Table 2. Matrix correlation between the main study variables

<table>
<thead>
<tr>
<th></th>
<th>Age (anni)</th>
<th>Weight (kg)</th>
<th>BMI (kg/m²)</th>
<th>Waist circumf. (cm)</th>
<th>FFM (kg)</th>
<th>FM tot. (kg)</th>
<th>FM trunk (kg)</th>
<th>FM leg (kg)</th>
<th>Glucose (mg/dl)</th>
<th>Tot. chol. (mg/dl)</th>
<th>HDL chol. (mg/dl)</th>
<th>LDL chol. (mg/dl)</th>
<th>log-TG (mg/dl)</th>
<th>HbA1c (%)</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
<th>PWVcf (m/s)</th>
<th>PWVcr (m/s)</th>
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<tr>
<td>Weight (kg)</td>
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<tr>
<td>BMI</td>
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<tr>
<td>Waist circ.</td>
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<td>0.84**</td>
<td>0.90**</td>
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<td>FFM (kg)</td>
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<td>0.91**</td>
<td>0.71**</td>
<td>0.73**</td>
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<td>FM tot. (kg)</td>
<td>0.16</td>
<td>0.95**</td>
<td>0.92**</td>
<td>0.86**</td>
<td>0.73**</td>
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<td>FM trunk (kg)</td>
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<td>0.91**</td>
<td>0.90**</td>
<td>0.71**</td>
<td>0.96**</td>
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<td>FM leg (kg)</td>
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<td>0.76**</td>
<td>0.73**</td>
<td>0.50**</td>
<td>0.59**</td>
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<tr>
<td>Glucose</td>
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<td>0.34**</td>
<td>0.37**</td>
<td>0.35**</td>
<td>0.31**</td>
<td>0.32**</td>
<td>0.18</td>
<td>0.12</td>
<td>0.23*</td>
<td>0.96**</td>
<td>0.71**</td>
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<td>-0.18</td>
<td>-0.00</td>
<td>-0.31**</td>
<td>-0.23*</td>
<td>-0.06</td>
<td>-0.10</td>
<td>0.04</td>
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<td>-0.39**</td>
<td>-0.41**</td>
<td>-0.23*</td>
<td>-0.12</td>
<td>0.29**</td>
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<td>-0.08</td>
<td>0.07</td>
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<td>-0.13</td>
<td>0.02</td>
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<td>0.91**</td>
<td>-0.04</td>
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<tr>
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<td>0.30**</td>
<td>0.35**</td>
<td>0.27**</td>
<td>0.27**</td>
<td>0.37**</td>
<td>0.03</td>
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<td>0.40**</td>
<td>0.43**</td>
<td>0.44**</td>
<td>0.40**</td>
<td>0.40**</td>
<td>0.30**</td>
<td>0.53**</td>
<td>0.26**</td>
<td>-0.24*</td>
<td>0.24*</td>
<td>1.00</td>
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<td>0.09</td>
<td>0.12</td>
<td>0.07</td>
<td>0.07</td>
<td>0.20</td>
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<td>-0.17</td>
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<td>0.02</td>
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<td>DBP (mmHg)</td>
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<td>0.03</td>
<td>0.01</td>
<td>0.14</td>
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<td>-0.12</td>
<td>-0.02</td>
<td>0.25*</td>
<td>0.14</td>
<td>0.66**</td>
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<tr>
<td>PWVcf (m/s)</td>
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<td>0.22*</td>
<td>0.28**</td>
<td>0.27**</td>
<td>0.19</td>
<td>0.25*</td>
<td>0.33**</td>
<td>0.02</td>
<td>0.02</td>
<td>0.01</td>
<td>-0.21*</td>
<td>0.04</td>
<td>0.24*</td>
<td>0.05</td>
<td>0.39**</td>
<td>0.23*</td>
<td>1.00</td>
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<tr>
<td>PWVcr (m/s)</td>
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<td>0.06</td>
<td>0.15</td>
<td>0.05</td>
<td>0.03</td>
<td>0.08</td>
<td>0.05</td>
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BMI, body mass index; FFM, fat free mass; FM tot., total fat mass; FM trunk, truncular fat mass; FM leg, sum of right and left fat mass; Chol. tot., total cholesterol; HDL chol., HDL cholesterol; LDL chol., cholesterol LDL; log-TG, logarithm of triglycerides; HbA1c, glyco-haemoglobin; SBP, systolic blood pressure; DBP, diastolic blood pressure; PWVcf, carotid-femoral pulse wave velocity; PWVcr, carotid-radial pulse wave velocity.

*P < 0.05.

**P < 0.01.
Lean mass, instead, was inversely related to both central and peripheral arterial stiffness [19]. However, our study confirms and complements these findings because our findings were observed in a sample of elderly women, without known CV diseases, with high range of BMI and because comprehensive measures of both peripheral and central arterial stiffness were determined.

In our study sample the binary logistic regression showed that MAP, the trunk fat mass and the leg fat mass were significant predictors of vascular damage. For every 1 mmHg of increase in MAP and for every kilogram of increase in the trunk fat mass, there was, respectively, a 6% (OR: 1.06) and 25% (OR: 1.25) risk to develop subclinical vascular damage. On the contrary for an increase of 1 kg of peripheral fat, there is a 27% (OR: 0.73) reduction in the risk of developing subclinical vascular damage.

Interestingly, when we stratified the study population considering the body fat distribution, the lowest mean PWVcf (9.7 ± 1.98 m/s) was observed in the group with a combined high-leg fat mass/low trunk mass, whereas the highest was in the group with a combined high-trunk fat mass/low leg fat mass, showing a PWVcf of 12.12 ± 3.69 m/s.

These findings are consistent with the concept that peripheral adipose tissue has a protective role on cardiovascular risk and particularly on arterial stiffness in terms of subclinical vascular damage [21].

Subcutaneous peripheral fat depots are metabolically different from abdominal visceral fat [22]. Peripheral adipose tissue is known to have higher lipoprotein lipase activity and low fatty acid turnover compared with visceral adipose tissue, which shows high lipolytic activity and elevated FFA flow and metabolism [23].

Furthermore, central adipose tissue is known to have a higher secretion of pro-inflammatory markers, such as leptin and IL 6, advanced glycation and lipoxidation products, whereas gluteo-femoral adipose tissue shows an increased secretion of anti-inflammatory adipokines, such as adiponectin, which has a known protective effect on arterial stiffness too [24].

Some limitations of the study should be recognised. First, we did not measure markers of inflammation, whose assessment could have provided more information about possible mechanisms by which the distribution of body fat affects subclinical vascular damage. Secondly, our study sample is small and limited only to apparently healthy older women, thus these findings cannot be generalised to a normal elderly population. In particular, this study was performed just in women, thus the present findings are not applicable in men. More importantly, we did not measure visceral fat, but rather its surrogates such as trunk fat, assessed by DXA. Thus, it was not possible to distinguish abdominal subcutaneous and visceral adipose tissue.

Thirdly, this study is cross-sectional and therefore cause–effect relationships cannot be determined.

Finally, in this study we used a cut-off of 12 m/s PWVcf, as an index of subclinical vascular damage; it has been suggested that this value should be modified in the elderly [25].

However, Alecu et al. [25] have previously suggested a threshold value of 13 m/s for the PulsePen device, even in elderly subjects without hypertension. It should also be noted that when the binary logistic analysis was performed using a threshold value of PWV of 13 m/s, the results did not change substantially.

In conclusion, the results of this study show in a sample of apparently healthy elderly women that central and peripheral adiposity together with MAP are independent predictors, with an opposite effect on subclinical vascular damage.

In particular, our findings show that in older ages, a high amount of trunk fat together with a low amount of peripheral fat is associated with a higher risk of developing subclinical vascular damage.

Our study seems to show that keeping the peripheral fat stable throughout ageing should be relevant to reduce CV risk in older women and to stress the need of measure surrogates of fat distribution even in older ages.
Key points

- Central and peripheral adiposity are independent predictors, with an opposite effect on subclinical vascular damage.
- This study shows a protective role of the gluteal-femoral fat on cardiovascular risk even in elderly.
- The study confirms the harmful role of the central fat mass on arteries in the elderly.

Conflicts of interest

None declared.

References


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