Peri-coronary epicardial adipose tissue is related to cardiovascular risk factors and coronary artery calcification in post-menopausal women

Alexander M. de Vos1,2, Mathias Prokop2, Cornelis J. Roos3, Matthijs F.L. Meijs1, Yvonne T. van der Schouw3, Annemarieke Rutten2, Petra M. Gorter4, Maarten-Jan Cramer1, Pieter A. Doevendans1, Benno J. Rensing5, Marie-Louise Bartelink3, Birgitta K. Velthuis2, Arend Mosterd1,3,6, and Michiel L. Bots3*

1Department of Cardiology, University Medical Centre Utrecht, Heidelberglaan 100, 3584 CX Utrecht, The Netherlands; 2Department of Radiology, University Medical Centre Utrecht, Utrecht, The Netherlands; 3Julius Centre for Health Sciences and Primary Care, University Medical Centre Utrecht, Huispost: str. 6.131, Heidelberglaan 10, 3584 CX, Utrecht, The Netherlands; 4Department of Vascular Medicine, University Medical Centre Utrecht, Heidelberglaan 100, 3584 CX Utrecht, The Netherlands; 5Department of Cardiology, Antonius Hospital Nieuwegein, Nieuwegein, The Netherlands; 6Department of Cardiology, Meander Medical Centre, Amersfoort, The Netherlands

Received 8 May 2007; revised 29 October 2007; accepted 13 November 2007; online publish-ahead-of-print 20 December 2007

See page 695 for the editorial comment on this article (doi:10.1093/eurheartj/ehm643)

Aims
To determine whether peri-coronary epicardial adipose tissue (EAT) is associated with vascular risk factors and coronary atherosclerosis.

Methods and results
In this study, 573 healthy post-menopausal women underwent a cardiac CT scan to assess coronary calcification. Peri-coronary EAT thickness was measured in the areas of right coronary artery (RCA), left anterior descending (LAD) artery, and left circumflex (LCX) coronary artery. Average EAT thickness was 16.5 ± 4.3 mm (range 5.9–34.6) in the RCA area, 6.4 ± 2.2 mm (range 2.0–14.0) in the LAD area, and 10.8 ± 3.0 mm (range 2.8–29.1) in the LCX area. Overall average thickness was 11.2 ± 2.2 mm (range 5.4–19.1). EAT was positively related to age (P = 0.002). In age-adjusted linear regression models, EAT was positively related to weight (P < 0.001), waist circumference (P < 0.001), waist-to-hip ratio (P < 0.001), body mass index (P < 0.001), glucose (P < 0.001), triglycerides (P = 0.001), use of anti-hypertensive drugs (P = 0.007), and systolic blood pressure (P = 0.034), and inversely to HDL cholesterol (P = 0.005). In multivariable models, age, weight, waist circumference, smoking, and glucose were the main determinants of EAT. EAT showed a graded relation with coronary calcification (P = 0.026).

Conclusion
EAT is strongly related to vascular risk factors and coronary calcification. Our findings support the hypothesis that EAT affects coronary atherosclerosis and possibly coronary risk.

Keywords
Epidemiology • CT and MRI • Risk factors • Imaging • Lipid • Lipoprotein metabolism

Introduction
Visceral adipose tissue is an important indicator of cardiovascular risk. Abdominal obesity has been shown to be a stronger predictor of cardiovascular risk than increased body mass index (BMI). This was found to be true for men and women, in the young and the old, and across populations of different ethnic origins. Epicardial adipose tissue (EAT) is a layer of visceral fat between the myocardium and the pericardium (Figure 1) and takes up ~22% of total heart weight. EAT, as well as intra-abdominal fat, appears...
Coronary arterial calcification (CAC) is a known marker of coronary atherosclerosis, which is considered an excessive inflammatory and proliferative process inside the vascular wall. There is growing evidence that the presence of inflammatory mediators in the tissues surrounding the epicardial coronary arteries plays an important role in this process. It is therefore conceivable that EAT contributes to the local development of atherosclerosis and the occurrence of cardiovascular events.

The few studies to date that assessed EAT predominantly used echocardiography, demonstrating that right ventricular EAT is significantly related to waist circumference, diastolic blood pressure, left ventricular mass, and high levels of insulin. Chaowalit et al. were not able to confirm these findings. It has also been reported that there is a significant association of average maximal EAT thickness on the right ventricle and whole-body glucose uptake, even when adjusted for BMI and waist circumference. In addition, the amount of all the (peri- and epicardial) adipose tissue surrounding the left anterior descending coronary artery area can be done perpendicularly to the heart to originate from the same brown adipose tissue of infancy and is a rich source of bioactive molecules directly surrounding the coronary arteries.

Coronary arterial calcification (CAC) is a known marker of coronary atherosclerosis, which is considered an excessive inflammatory and proliferative process inside the vascular wall. There is growing evidence that the presence of inflammatory mediators in the tissues surrounding the epicardial coronary arteries plays an important role in this process. It is therefore conceivable that EAT contributes to the local development of atherosclerosis and the occurrence of cardiovascular events.

The few studies to date that assessed EAT predominantly used echocardiography, demonstrating that right ventricular EAT is significantly related to waist circumference, diastolic blood pressure, left ventricular mass, and high levels of insulin. Chaowalit et al. were not able to confirm these findings. It has also been reported that there is a significant association of average maximal EAT thickness on the right ventricle and whole-body glucose uptake, even when adjusted for BMI and waist circumference. In addition, the amount of all the (peri- and epicardial) adipose tissue surrounding the heart has been related to the severity of coronary artery disease, assessed by coronary angiography. The EAT and, in particular, the adipose tissue directly surrounding the coronary arteries rather than all the adipose tissue surrounding the coronary arteries plays an important role in this process. It is therefore conceivable that EAT contributes to the local development of atherosclerosis and the occurrence of cardiovascular events.

The few studies to date that assessed EAT predominantly used echocardiography, demonstrating that right ventricular EAT is significantly related to waist circumference, diastolic blood pressure, left ventricular mass, and high levels of insulin. Chaowalit et al. were not able to confirm these findings. It has also been reported that there is a significant association of average maximal EAT thickness on the right ventricle and whole-body glucose uptake, even when adjusted for BMI and waist circumference. In addition, the amount of all the (peri- and epicardial) adipose tissue surrounding the heart has been related to the severity of coronary artery disease, assessed by coronary angiography. The EAT and, in particular, the adipose tissue directly surrounding the coronary arteries rather than all the adipose tissue surrounding the coronary arteries plays an important role in this process. It is therefore conceivable that EAT contributes to the local development of atherosclerosis and the occurrence of cardiovascular events.

The current study addressed whether peri-coronary EAT is associated with cardiovascular risk factors and coronary artery calcification.

**Methods**

**Study population**

We used data from a cross-sectional study among 573 healthy post-menopausal women. These women were selected from participants of PROSPECT study, one of the two Dutch cohorts participating in the European Prospective Investigation into Cancer and Nutrition (EPIC). In PROSPECT, 17 357 healthy participants of a nationwide population-based breast cancer screening programme, aged 49–70 years, living in Utrecht and surroundings, were enrolled between 1993 and 1997. Between October 2002 and April 2004, a re-examination was planned in a sample to investigate the prognostic value of age at menopause on CVD risk. For this purpose, 6612 women of the total of 17 357 were excluded because of death, further participation in PROSPECT or in other studies, absence of written informed consent, or emigration. Other reasons for exclusion were pre-menopausal state (n = 1309), missing data on menopausal status (n = 2105), or use of oral contraceptives or post-menopausal hormone therapy in the year before or after the last menstruation (n = 1487), as this precludes accurate estimation of age at menopause. Of 5844 eligible women, a random selection of 1996 women was invited by a personal letter from the principal investigator of PROSPECT, and 1000 (50.1%) answered positively. Of these 1000 women, 573 women were randomly selected for CAC measurement. During the conduct of the study, the possibility of performing CAC measurements came about to study menopausal aspects in relation to coronary atherosclerosis. From a logistical aspect, CAC measurement appeared to be possible in 573 women. There was no a priori sample size estimation for EAT, since the possibility for doing EAT measurements came after the scans had been made.

The study complies with the Declaration of Helsinki, the Medical Ethical Committee of the University Medical Centre Utrecht approved the study, and written informed consent was obtained from all participants.

Current cardiovascular drug use (blood pressure-lowering, lipid-lowering, and glucose-lowering drugs) was assessed by asking women to bring all packages to the study centre. Smoking behaviour, medical history, and cardiovascular family history were assessed by a questionnaire. Height and weight were measured and BMI was calculated as weight divided by height squared (kg/m²). Waist-to-hip ratio was assessed. Systolic and diastolic blood pressures were measured at both arms with an automated and calibrated blood pressure device (DINAMAP™ XL, Critikon, Johnson & Johnson, Tampa, FL, USA), with the subject in supine position. A venous blood sample was drawn after an overnight fasting period of at least 8 h. Plasma total cholesterol, plasma triglycerides, and plasma glucose were determined using an automated enzymatic procedure on a Vitros 250 (Johnson & Johnson, Rochester, New York, USA). Low-density lipid (LDL) and high-density lipid (HDL) cholesterol were measured using a colorimetric assay on a Hitachi 904 (Johnson & Johnson, Rochester).

**Cardiac imaging and calcium measurements**

The amount of calcium in the coronary arteries was assessed with a multidetector-row computed tomography (MDCT) scanner (MX 8000 IDT 16, Philips Medical Systems, Best, The Netherlands). Subjects were positioned within the gantry of the MDCT scanner in a supine position. During a single breath-hold, images of the heart, from the level of the tracheal bifurcation to below the base of the heart, were acquired using prospective ECG triggering at 50–80% of the RR interval, depending on the heart rate. Scan parameters were 16 × 1.5 mm collimation, 205 mm field of view, 0.42 s rotation time, 0.38 s scan time per table position, 120 kVp, and 40–70 mAs (patient weight < 70 kg: 40 mAs; 70–90 kg: 55 mAs; > 90 kg: 70 mAs). Scan duration was ~10 s, depending on heart rate and patient size. From the acquired data, the amount of calcium in the coronary arteries was assessed with a multidetector-row computed tomography calcium scoring device (DINAMAP™ XL, Critikon, Johnson & Johnson, Rochester).
raw data, the whole volume was reconstructed with an intermediate reconstruction algorithm in non-overlapping data sets of 1.5 mm thick sections. Quantification of coronary calcium was performed on a separate workstation with software for calcium scoring (Heartbeat-CS, Extended Brilliance Workspace, Philips Medical Systems, Best, The Netherlands). All regions with a density >130 HU within the coronary arteries were manually identified as potential calcifications. To reduce the influence of noise, the minimum size of a calcified lesion was set at 0.5 mm². The peak density in Hounsfield units and the area in millimeter² of each selected signal anywhere in the lesion.23 The scores of individual lesions were added to obtain the Agatston CAC score for the entire coronary tree. A reproducibility study in which a subgroup of 76 women were scanned twice revealed an intra-class correlation coefficient of 0.98 for the Agatston CAC score.24

**Epicardial adipose tissue measurements**

The amount of EAT surrounding the coronary arteries was quantified on the MDCT scans, using a standardized method. Scans were loaded into a regular Philips CT application (Extended Brilliance Workspace). The researcher adjusted window settings to make the pericardium and EAT visible. Next, the sections were determined where axial cuts are perpendicular to the surface of the heart (in order not to overestimate EAT diameter due to obliquity). This was done separately for the three main coronary territories: right coronary artery (RCA), left anterior descending (LAD) coronary artery, and the left circumflex (LCX) coronary artery. If more than one axial cut was perpendicular to the heart, the one with the most distinct layer of EAT was chosen for the measurements. At each of the three main coronary territories, maximal EAT thickness (mm) was determined perpendicular to the pericardium (Figure 1). A number of 32 scans were evaluated a second time by a different observer in order to determine interobserver variability. This was regarded as sufficient to obtain an adequate estimate of reproducibility of the reading method.

One reader (C.J.R.) read all the images. At the time of reading, he was blinded to the study hypothesis and to the levels of risk factors in participants to limit the potential for bias. Furthermore, CAC measurements were performed by a different person (A.R.).

**Data analysis**

Baseline characteristics were expressed according to the quartile distribution of average peri-coronary EAT thickness. Reproducibility of the EAT measurements was assessed by estimation of the intra-class correlation coefficient and the 95% limits of agreement.25

The analyses were performed using the average thickness of EAT measurements of the RCA, the LAD, and the LCX as continuous variable. Continuous variables were computed as mean and standard deviation, and categorical variables were expressed as percentage.

In order to study whether the 10% random selection of the 5844 women might have severely biased our findings, we compared the baseline characteristics of the 573 participating women with the characteristics of the 5844 women, using regression models. Please note that (positive) selection bias occurs only when the selection process results in oversampling of subjects with both increased levels of exposure of interest and increased levels of the outcome. Extreme bias towards a null finding may occur when extreme oversampling occurs in both exposure and outcome.

First, the mean levels of all risk factors were studied across quartiles of the average EAT measurements. Since EAT was strongly related to age, all the relations with risk factors were initially adjusted for age using linear regression models. Risk factors that were related to EAT with P < 0.10 were entered into a multivariable linear regression model to study the independent relationships. If two factors interfered (for example, weight and BMI), one was left out.

Next, the relation between EAT and coronary calcification was assessed using linear regression models. Vascular risk factors were not added into these models because we thought of traditional risk factors as intermediates in the process of EAT influencing the process of atherosclerosis. CAC was categorized as proposed by Rumberger et al.10 Mean EAT thickness across categories of CAC was evaluated using an age-adjusted model. Model assumptions were checked graphically by qq plots of predicted values vs. residuals. All statistical analyses were performed with the statistical package SPSS (SPSS for Windows, release 12.0.1, 2004; SPSS Inc., Chicago, IL, USA). Associations were considered significant at P < 0.05. All statistical tests were two-sided.

**Results**

The women were between 57 and 81 years of age (average 66.8 ± 5.5). The average thickness of the layer of EAT surrounding the coronaries was 16.5 ± 4.3 mm (range 5.9–34.6 mm) in the RCA area, 6.4 ± 2.2 mm (range 2.0–14.0 mm) in the LAD area, and 10.8 ± 3.0 mm (range 2.8–29.1 mm) in the LCX area. Average thickness over all three territories was 11.2 ± 2.2 mm (range 5.4–19.1). According to visual inspection, average EAT thickness is normally distributed (Figure 2). The intra-class correlation coefficient was 0.76 (95% CI 0.50–0.88). According to Bland–Altman analysis, the 95% limits of agreement for the average EAT thickness measurements were –4.0 and 7.1 mm. In 12 subjects, CT scans could not be evaluated due to poor image quality. These persons were excluded from the analysis.
The baseline characteristics of the 573 participating women showed modestly lower levels of most vascular risk factors and equal distribution of waist-to-hip ratio. Thus, in the participating women, the CAC as well as EAT is expected to be lower since determinants of both show lower levels. Therefore, if anything, our relation is most likely an underestimate of the truth.

Table 1 describes the baseline characteristics of the study cohort according to quartiles of average peri-coronary EAT thickness. Women with the highest EAT values (fourth quartile) were on average older, had a higher weight, a higher BMI, a larger waist circumference and waist-to-hip ratio, had higher systolic blood pressure, more often used antihypertensive medication, a lower HDL levels and higher triglycerides, and a higher glucose level than women with the lowest EAT values (first quartile). Furthermore, CAC levels increased with increasing EAT (Figure 3).

EAT was positively related to age ($P = 0.002$). Table 2 provides results from age-adjusted linear regression analyses, with average EAT thickness over the three coronary arteries. Significant positive relations were found for weight, BMI, waist circumference, waist-to-hip ratio, systolic blood pressure, use of anti-hypertensive drugs, triglycerides, and glucose. HDL cholesterol was inversely related to the layer of peri-coronary EAT. When we entered those risk factors with $P < 0.10$ into a multivariable linear regression model (Table 3, model 1), age, weight, current smoking (borderline significant), and serum glucose were significantly related to the average thickness of EAT around the coronary arteries. As a measure for serum lipid status, we took both HDL and triglycerides into the model. When, however, waist circumference entered the model (Table 3, model 2), all traditional risk factors but age and waist circumference lost their significant relation with EAT. The Spearman correlation between EAT and waist circumference was 0.34 ($P < 0.001$). A correlation of 0.34 does not indicate severe collinearity.

Figure 3 depicts the univariable relation between EAT and CAC. The Spearman correlation between EAT and CAC was 0.016 ($P = 0.003$). A graded association was seen: the higher the amount of EAT, the higher the amount of CAC. This relationship remained statistically significant when adjusted for age in a linear regression model ($P = 0.026$). The relation of EAT and CAC was not significant when stratified according to waist circumference. Age-adjusted regression analyses showed that the diameter of EAT around the LAD and the RCA was strongly correlated to the presence of coronary calcification in the same artery ($P = 0.034$ and $P = 0.03$, respectively). This was not found for the right circumflex coronary artery ($P = 0.285$).

### Table 1 Characteristics of 573 post-menopausal women by epicardial adipose tissue thickness quartiles

<table>
<thead>
<tr>
<th>Quartiles of overall average EAT thickness</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Number of subjects</td>
<td>140</td>
</tr>
<tr>
<td>Age, years</td>
<td>65.6 ± 5.2</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>68.1 ± 12.6</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.64 ± 0.06</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>25.1 ± 4.3</td>
</tr>
<tr>
<td>Hip circumference, cm</td>
<td>99.1 ± 8.2</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>81.4 ± 10.7</td>
</tr>
<tr>
<td>Waist-to-hip ratio</td>
<td>0.82 ± 0.06</td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>131.8 ± 19.1</td>
</tr>
<tr>
<td>Diastolic blood pressure, mmHg</td>
<td>71.1 ± 9.5</td>
</tr>
<tr>
<td>Use of BP-lowering drugs, %</td>
<td>21.4</td>
</tr>
<tr>
<td>Hypertension 140/90 or medication, %</td>
<td>47.1</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>5.9 ± 0.9</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.4 ± 0.4</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>4.1 ± 0.8</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>1.1 ± 0.5</td>
</tr>
<tr>
<td>Current smoking, %</td>
<td>8.6</td>
</tr>
<tr>
<td>Former smoking, %</td>
<td>47.1</td>
</tr>
<tr>
<td>Previous CVD, %</td>
<td>2.9</td>
</tr>
<tr>
<td>Diabetes mellitus, %</td>
<td>5.7</td>
</tr>
<tr>
<td>Glucose, mmol/L</td>
<td>5.5 ± 0.8</td>
</tr>
<tr>
<td>CAC score, Agatston</td>
<td>58.8 ± 151.3</td>
</tr>
</tbody>
</table>

Continuous variables are presented as mean ± SD. The last column shows significance in the age-adjusted linear regression analysis, with average EAT thickness of the three coronary arteries as dependent variable. The cut points of the quartiles are 9.7, 11.2, and 12.5 mm.
Additional analyses, excluding participants with previous cardiovascular disease (acute myocardial infarction, coronary artery stenosis, carotid artery stenosis, and stroke), did not materially change the results.

**Discussion**

The major finding of this population-based study in healthy post-menopausal women is that EAT directly surrounding the coronary arteries is related to a large number of vascular risk factors. Most of these relations are attenuated when an adjustment was made for waist circumference, suggesting that EAT and abdominal adipose tissue are strongly related. Furthermore, peri-coronary EAT is related to CAC, suggesting a role in the development of coronary atherosclerosis.

Previous studies on EAT thickness were mainly based on echocardiography and have measured EAT thickness on the right ventricle only,1,17,18 or combined epicardial and pericardial adipose tissue surrounding the heart.21 Differentiation between epicardial and pericardial fat may be difficult at echocardiography.26 On CT, the pericardium is readily identified, resulting in easy differentiation between epicardial and pericardial fat. In contrast to previous studies, we focused on epicardial fat surrounding the arteries because of the notion that it is the local fat that may drive the development of atherosclerosis. Sample size of the echocardiography studies ranged from 22 to 72 persons. Moreover, studied populations differed from our general population sample.18,20,21 The studies using echocardiography to measure EAT thickness on the right ventricle showed relations with waist circumference, diastolic blood pressure, and left ventricular mass.1,17,18 Our findings for peri-coronary EAT are in agreement with this. We report a strong relation between the peri-coronary EAT and age. Although

---

**Table 2 Age-adjusted relations between epicardial adipose tissue and risk factors**

<table>
<thead>
<tr>
<th>Cardiovascular risk factor</th>
<th>Beta</th>
<th>95% Confidence interval for beta</th>
<th>P-value</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Lower bound</td>
<td>Upper bound</td>
<td></td>
</tr>
<tr>
<td>Weight, kg</td>
<td>0.017</td>
<td>0.010</td>
<td>0.025</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height, m</td>
<td>0.821</td>
<td>-2.234</td>
<td>3.876</td>
<td>0.598</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>0.161</td>
<td>0.121</td>
<td>0.200</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hip circumference, cm</td>
<td>0.008</td>
<td>-0.001</td>
<td>0.017</td>
<td>0.095</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>0.076</td>
<td>0.060</td>
<td>0.092</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Waist-to-hip ratio</td>
<td>9.699</td>
<td>7.380</td>
<td>12.018</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic blood pressure, mmHg</td>
<td>0.010</td>
<td>0.001</td>
<td>0.019</td>
<td>0.034</td>
</tr>
<tr>
<td>Diastolic blood pressure, mmHg</td>
<td>0.018</td>
<td>-0.002</td>
<td>0.037</td>
<td>0.079</td>
</tr>
<tr>
<td>Use of BP-lowering drugs</td>
<td>0.592</td>
<td>0.165</td>
<td>1.019</td>
<td>0.007</td>
</tr>
<tr>
<td>Hypertension 140/90 or medication</td>
<td>0.317</td>
<td>-0.057</td>
<td>0.691</td>
<td>0.096</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>0.038</td>
<td>-0.147</td>
<td>0.223</td>
<td>0.687</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>0.749</td>
<td>-1.267</td>
<td>-0.231</td>
<td>0.005</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>0.098</td>
<td>-0.103</td>
<td>0.300</td>
<td>0.339</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>0.502</td>
<td>0.206</td>
<td>0.798</td>
<td>0.001</td>
</tr>
<tr>
<td>Current smoking</td>
<td>0.500</td>
<td>-0.074</td>
<td>1.074</td>
<td>0.088</td>
</tr>
<tr>
<td>Former smoking</td>
<td>-0.114</td>
<td>-0.482</td>
<td>0.255</td>
<td>0.545</td>
</tr>
<tr>
<td>Previous CVD</td>
<td>0.189</td>
<td>-0.801</td>
<td>1.180</td>
<td>0.707</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>0.540</td>
<td>-0.287</td>
<td>1.368</td>
<td>0.200</td>
</tr>
<tr>
<td>Glucose, mmol/L</td>
<td>0.349</td>
<td>0.153</td>
<td>0.544</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CAC score, Agatston</td>
<td>0.161</td>
<td>0.019</td>
<td>0.303</td>
<td>0.026</td>
</tr>
</tbody>
</table>

Results are presented as linear regression coefficients with corresponding 95% confidence limits, P-values, and the amount of variance explained (R²). Beta means a change in EAT (mm) with an increase of one unit in the risk factor. For example, change of 1 kg in weight leads to a change in EAT of 0.017 mm.
autopsy studies have found a relationship between EAT and age. The fact that we found more EAT in the area of the RCA is in concordance with the literature. Some limitations of the study need to be addressed. We did not collect either CT or ultrasound measurements on abdominal adipose fat, which would have made our study results stronger in elucidating the relation between EAT and abdominal adipose tissue. Also, it is a cross-sectional study, which limits conclusions on cause and consequence. Although our results are based on data obtained in women, we do not know any mechanistic reason why these findings may not also apply to men. Nevertheless, confirmation of these findings in studies among men is recommended to firmly support that notion. The strength of the study, however, is its population-based nature and its fairly large sample size. CT offers excellent spatial resolution of ~0.4–0.6 mm, which is superior to competing imaging modalities (Figure 1).

The present study is not able to distinguish between peri-coronary EAT having a direct effect on the development of CAC and peri-coronary EAT being merely a reflection of systemic elevated risk factors that lead to the development of CAC. Future studies on this issue are needed, as are studies relating the presence of peri-coronary EAT to the occurrence of cardiovascular events.

In conclusion, our findings in a population-based study of CT scans in post-menopausal women provide evidence that the layer of EAT directly surrounding the coronary arteries is strongly associated with several vascular risk factors and coronary artery calcification.

Conflict of Interest: none declared.

Funding
This study was partly made possible by grant 2100.0078 from the Netherlands Organization for Health Research and Development.

References


