Pericardial Fat, Visceral Abdominal Fat, Cardiovascular Disease Risk Factors, and Vascular Calcification in a Community-Based Sample

The Framingham Heart Study

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Background—Pericardial fat may be an important mediator of metabolic risk. Correlations with cardiovascular disease risk factors and vascular calcification in a community-based sample are lacking. We sought to examine associations between pericardial fat, metabolic risk factors, and vascular calcification.

Methods and Results—Participants free of cardiovascular disease from the Framingham Heart Study (n=11005, mean age 63 years, 54.8% women) who were part of a multidetector computed tomography study underwent quantification of intrathoracic fat, pericardial fat, visceral abdominal fat (VAT), coronary artery calcification, and aortic artery calcification. Intrathoracic and pericardial fat volumes were examined in relation to body mass index, waist circumference, VAT, metabolic risk factors, coronary artery calcification, and abdominal aortic calcification. Intrathoracic and pericardial fat were directly correlated with body mass index (r=0.41 to 0.51, P<0.001), waist circumference (r=0.43 to 0.53, P<0.001), and VAT (r=0.62 to 0.76, P<0.001). Both intrathoracic and pericardial fat were associated with higher triglycerides (P<0.0001), lower high-density lipoprotein (P<0.0001), hypertension (P<0.0001 to 0.01), impaired fasting glucose (P<0.0001 to 0.001), diabetes mellitus (P=0.0005 to 0.009), and metabolic syndrome (P<0.0001) after multivariable adjustment. Associations generally persisted after additional adjustment for body mass index and waist circumference but not after adjustment for VAT (all P<0.05). Pericardial fat, but not intrathoracic fat, was associated with coronary artery calcification after multivariable and VAT adjustment (odds ratio 1.21, 95% confidence interval 1.005 to 1.46, P=0.04), whereas intrathoracic fat, but not pericardial fat, was associated with abdominal aortic calcification (odds ratio 1.32, 95% confidence interval 1.03 to 1.67, P=0.03).

Conclusions—Pericardial fat is correlated with multiple measures of adiposity and cardiovascular disease risk factors, but VAT is a stronger correlate of most metabolic risk factors. However, intrathoracic and pericardial fat are associated with vascular calcification, which suggests that these fat depots may exert local toxic effects on the vasculature. (Circulation. 2008;117:605-613.)

Key Words: pericardium ■ epidemiology ■ risk factors ■ obesity

Different fat depots may be associated with variable metabolic risk. Visceral abdominal tissue (VAT) is associated with cardiovascular disease (CVD) and its risk factors, including diabetes mellitus, insulin resistance, hypertension, and dyslipidemia. In the Framingham Heart Study, the volume of VAT determined by computed tomography (CT) was associated with multiple metabolic risk factors, as well as the metabolic syndrome (MetS). Ectopic fat depots may mediate obesity-related vascular disease. Pericardial fat, an ectopic fat depot, is correlated with body mass index (BMI), VAT, metabolic risk factors, insulin resistance, and coronary artery disease. However, it is not known whether pericardial fat is a marker of overall adiposity or an independent pathogenic factor.
fat depot, because the role of pericardial fat above and beyond that of VAT has not been assessed. In addition, the current understanding of the relationship between pericardial fat and metabolic risk factors may be obscured by methodological issues, including small, selected patient-based samples and measures of pericardial fat thickness instead of actual pericardial fat volume. Thus, the purpose of the present study is to examine the relations of CT measures of pericardial fat volume with multiple measures of adiposity including VAT, as well as CVD risk factors; to assess whether pericardial fat is significantly correlated with metabolic risk factors above and beyond BMI, waist circumference (WC), and VAT; and to assess correlations with atherosclerotic burden measured by coronary and abdominal aortic calcium.

Methods

Study Sample
The original cohort of the Framingham Heart Study was enrolled in 1948 and included 5209 women and men aged 28 to 62 years. In 1971, the offspring and spouses of the original cohort’s offspring were enrolled into the Offspring Study; the study design has been described previously. The present study includes Offspring Study participants from the multidetector CT (MDCT) substudy. Overall, 3539 participants attended the seventh examination. Of these, 1418 (40.1%) underwent CT scanning and assessment for coronary and aortic calcium with MDCT between June 2002 and April 2005; inclusion criteria have been described previously. Of the 1418 participants imaged, 1372 had interpretable pericardial fat measures, and 1212 were free of CVD; of these, 1189 attended the seventh examination cycle (1998–2001), 1188 had at least 1 outcome of interest measured, 1160 had an available measure of VAT, and 1155 had a complete covariate profile. The study protocol was approved by the institutional review boards of the Boston University Medical Center and Massachusetts General Hospital. All subjects provided written informed consent.

MDCT Scan Protocol
Participants underwent radiographic assessment in a supine position with 8-slice MDCT (LightSpeed Ultra, General Electric, Milwaukee, Wis). An average of 48 contiguous 2.5-mm slices of the heart were taken with a prospectively ECG-triggered CT scanning protocol with the image acquisition initiated at 70% of the cardiac cycle to obtain nearly motion-free images of the coronary arteries (120 kVp, 400 mA, temporal resolution 330 ms), and 25 slices 5 mm thick (120 kVp, 400 mA, gantry rotation time 500 ms, table feed 3:1) were obtained that covered a 125-mm segment of the abdomen with the upper edge of S1 used as an anatomic landmark on the topogram. Participants underwent a second thoracic scan after being briefly repositioned on the table.

Intrathoracic and Pericardial Fat Volumes

Data Analysis
Intrathoracic and pericardial fat tissue volumes were measured with a dedicated offline workstation (Aquarius 3D Workstation, TeraRecon Inc, San Mateo, Calif). Absolute Hounsfield unit (HU) values of pixels correspond directly to tissue property. Therefore, a predefined image display setting was used to determine the intrathoracic and pericardial fat tissue volumes (window width −195 to −45 HU; window center −120 HU) to identify pixels that corresponded to adipose tissue. Intrathoracic and pericardial fat were measured across total available imaging volume in cubic centimeters.

Intrathoracic and pericardial fat volumes were measured by a semiautomatic segmentation technique with the above display settings. The reader was required to manually trace the pericardium. Intrathoracic fat volume was defined as any adipose tissue located both within the pericardium and within the thorax from the level of the right pulmonary artery to the diaphragm and from the chest wall to the descending aorta. Pericardial fat volume was defined as any adipose tissue located within the pericardial sac. Segmentation of the overall volume was automatically interpolated with the manually defined tracings. When necessary, the reader made manual adjustments through the scan volumes to account for interpolating errors. On a random sample of 100 participants, interreader reproducibility was excellent for intrathoracic fat (intraclass correlation 0.99) and for pericardial fat (intraclass correlation 0.97). Interreader reproducibility was also excellent (intrathoracic fat intraclass correlation 0.98, pericardial fat 0.95).

Abdominal Adipose Tissue Measurements
VAT volumes were assessed as described previously with the same image display windows as above (Aquarius 3D Workstation, TeraRecon Inc). Intraclass correlations for interreader comparisons were 0.99 for VAT.

Coronary Artery Calcium and Abdominal Aortic Calcium Measurements
MDCT scans were read by an experienced reader for the presence and quantity of coronary artery calcium (CAC) and abdominal aortic calcium (AAC); a dedicated offline workstation was used for this purpose (Aquarius, TeraRecon). A calcified lesion was defined as an area of at least 3 connected pixels with MDCT attenuation >130 HU (applying 3D connectivity criteria [6 points]). The Agatston score was computed by multiplying each lesion (area) by a weighted MDCT attenuation score (in HU) within the lesion. The mean Agatston score from both chest scans was used to calculate CAC, and CAC scores were highly reproducible. The presence of CAC or AAC was based on age- and sex-specific 90th percentile cut points derived from a healthy referent sample.

Risk Factor and Covariate Assessment
Risk factors and covariates were measured at the seventh Framingham Offspring examination (1998–2001). BMI was defined as weight (in kilograms) divided by the square of height (in meters). WC was measured at the umbilicus. Serum triglycerides, total and high-density lipoprotein (HDL) cholesterol, and fasting plasma glucose were measured on fasting morning samples. To define diabetes mellitus, fasting plasma glucose ≥126 mg/dL or treatment with a hypoglycemic agent or insulin was used; impaired fasting glucose was defined as fasting plasma glucose 100 to 125 mg/dL in the absence of treatment for diabetes mellitus. Current smoking status was defined as smoking at least 1 cigarette per day in the past year. Using physician-administered questionnaires, alcohol use was assessed and categorized as more or less than >14 drinks/week (men) or 7 drinks/week (women). If periods stopped for 1 year or more, women were considered postmenopausal. Systolic blood pressure ≥140 mm Hg, diastolic blood pressure ≥90 mm Hg, or antihypertensive treatment was used to define hypertension. Modified National Cholesterol Education Program Adult Treatment Panel III criteria were used to define the MetS.

Statistical Analysis
Intrathoracic and pericardial fat were normally distributed. Age-adjusted Pearson correlations between intrathoracic and pericardial fat were performed with metabolic risk factors and adiposity traits. Multivariable linear and logistic regression was used to assess the significance of covariate-adjusted cross-sectional relations between continuous and dichotomous metabolic risk factors and intrathoracic and pericardial fat. The covariate-adjusted risk factor per 1-SD increase of adipose tissue was estimated for continuous variables; for dichotomous risk factors, the odds ratio of the risk factor prevalence per 1-SD increase in adipose tissue was estimated. Sex interactions were tested. Covariates included age, smoking (current/former/never smoker), alcohol use (>14 drinks/week [men] or >7 drinks/week [women]), menopausal status, and hormone replacement therapy. Lipid treatment, hypertension treatment, and diabetes treatment were included as covariates in models for HDL cholesterol, log triglycer-
Intrathoracic fat, cm³ 194 (76) 285 (107)
Alcohol use, %‡ 16.1 16.7
Hormone replacement therapy, % 36.5
Postmenopausal, % 81.8
Smoking, %
MetS, % 37.6 42.9
Diabetes mellitus, % 7.6 10.7
Impaired fasting glucose, %† 26.0 46.6
Fasting plasma glucose, mg/dL 98 (17) 104 (23)
Hypertension, % 36.2 39.7
Diastolic blood pressure, mm Hg 73 (9) 77 (9)
Systolic blood pressure, mm Hg 124 (19) 127 (16)
Total cholesterol, mg/dL 208 (36) 195 (33)
HDL cholesterol, mg/dL 61 (15) 45 (12)
Triglycerides, mg/dL* 111 (77–162) 116 (78–171)
WC, cm 96 (15) 103 (11)
VAT, cm³ 1631 (876) 2548 (1036)
Pericardial fat, cm³ 110 (41) 137 (53)

Table 1. Study Sample Characteristics

<table>
<thead>
<tr>
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<th>Women (n=633)</th>
<th>Men (n=522)</th>
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<td>Age, y</td>
<td>63 (9)</td>
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<td>Body mass index, kg/m²</td>
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<td>28.8 (4.3)</td>
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<tr>
<td>WC, cm</td>
<td>96 (15)</td>
<td>103 (11)</td>
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<tr>
<td>Triglycerides, mg/dL*</td>
<td>111 (77–162)</td>
<td>116 (78–171)</td>
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<tr>
<td>HDL cholesterol, mg/dL</td>
<td>61 (15)</td>
<td>45 (12)</td>
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<tr>
<td>Total cholesterol, mg/dL</td>
<td>208 (36)</td>
<td>195 (33)</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>124 (19)</td>
<td>127 (16)</td>
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<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>73 (9)</td>
<td>77 (9)</td>
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<td>Hypertension, %</td>
<td>36.2</td>
<td>39.7</td>
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<tr>
<td>Fasting plasma glucose, mg/dL</td>
<td>98 (17)</td>
<td>104 (23)</td>
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<tr>
<td>Impaired fasting glucose, %‡</td>
<td>26.0</td>
<td>46.6</td>
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<tr>
<td>Diabetes mellitus, %</td>
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<tr>
<td>MetS, %</td>
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<td>Smoking, %</td>
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<td>Current</td>
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<tr>
<td>Former</td>
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<td>52.5</td>
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<tr>
<td>Never</td>
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<td>37.9</td>
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<td>Postmenopausal, %</td>
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<td>Hormone replacement therapy, %</td>
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<tr>
<td>Alcohol use, %‡</td>
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<tr>
<td>Intrathoracic fat, cm³</td>
<td>194 (76)</td>
<td>285 (107)</td>
</tr>
<tr>
<td>Pericardial fat, cm³</td>
<td>110 (41)</td>
<td>137 (53)</td>
</tr>
<tr>
<td>VAT, cm³</td>
<td>1631 (876)</td>
<td>2548 (1036)</td>
</tr>
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</table>

Data are presented as mean (SD) or as percentage having that characteristic.
*Median with 25th–75th percentiles.
†Defined as fasting plasma glucose 100 to 125 mg/dL; percentage based on those without diabetes.
‡Defined as >14 drinks per week (men) or >7 drinks per week (women).

Table 2. Age-Adjusted Pearson Correlation Coefficients Between Metabolic Risk Factors and Intrathoracic and Pericardial Fat Volumes

<table>
<thead>
<tr>
<th></th>
<th>Intrathoracic Fat</th>
<th>Pericardial Fat</th>
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<th>Pericardial Fat</th>
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<td></td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
</tr>
<tr>
<td>Age</td>
<td>0.28‡</td>
<td>0.25‡</td>
<td>0.25‡</td>
<td>0.23‡</td>
</tr>
<tr>
<td>BMI</td>
<td>0.49‡</td>
<td>0.41‡</td>
<td>0.51†</td>
<td>0.46§</td>
</tr>
<tr>
<td>WC</td>
<td>0.53‡</td>
<td>0.43‡</td>
<td>0.54‡</td>
<td>0.49§</td>
</tr>
<tr>
<td>VAT</td>
<td>0.76‡</td>
<td>0.62‡</td>
<td>0.76‡</td>
<td>0.63§</td>
</tr>
<tr>
<td>Log triglycerides</td>
<td>0.34‡</td>
<td>0.31‡</td>
<td>0.26‡</td>
<td>0.22§</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>−0.25‡</td>
<td>−0.24‡</td>
<td>−0.21‡</td>
<td>−0.14‡</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>0.06</td>
<td>0.05</td>
<td>0.01</td>
<td>0.04</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>0.19‡</td>
<td>0.15‡</td>
<td>0.12*</td>
<td>0.10*</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>0.17‡</td>
<td>0.12‡</td>
<td>0.10*</td>
<td>0.08</td>
</tr>
<tr>
<td>Blood glucose</td>
<td>0.30‡</td>
<td>0.27‡</td>
<td>0.13†</td>
<td>0.10*</td>
</tr>
<tr>
<td>Physical activity index</td>
<td>−0.09*</td>
<td>−0.06</td>
<td>−0.05</td>
<td>−0.04</td>
</tr>
</tbody>
</table>

*P<0.05; †P<0.01; ‡P<0.001.

Results

The mean age of the study sample was 63 years, and 54.8% were women (Table 1). Mean intrathoracic and pericardial fat volumes were 194 and 110 cm³ in women and 285 and 137 cm³ in men, respectively. Both intrathoracic and pericardial fat volumes were significantly larger in men than women (P<0.0001).

Intrathoracic and pericardial fat were highly correlated in women (r=0.90, P<0.0001) and in men (r=0.90, P<0.0001). Both intrathoracic and pericardial fat increased with age (P<0.001) and were associated with BMI and WC in women and men (Table 2). Intrathoracic fat was more closely correlated with VAT than was pericardial fat in women (r=0.76 versus 0.62) and men (r=0.76 versus 0.63). Intrathoracic and pericardial fat were associated with most of the cardiovascular risk factors with the exception of total cholesterol.

Both intrathoracic and pericardial fat were associated with systolic blood pressure in women but not men (Table 3; P for sex interaction=0.0002 for intrathoracic fat and 0.003 for pericardial fat). Among women, associated mean systolic blood pressure was 3.1 mm Hg higher per 1-SD increase in intrathoracic fat (P<0.0001) and 2.5 mm Hg higher per 1-SD increase in pericardial fat.
increase in pericardial fat ($P=0.0005$). After additional adjustment for BMI and WC, these relations were no longer significant. Among women, VAT but not intrathoracic fat was associated with systolic blood pressure when both adiposity variables were considered in the model together (Table 4); similar results were obtained for pericardial fat (data not shown). Similar results were observed for diastolic blood pressure.

Fasting plasma glucose was associated with both intrathoracic and pericardial fat in women but not men (Table 3); these associations persisted after additional adjustment for BMI and WC. When VAT was added to the model, VAT but not intrathoracic fat was associated with fasting plasma glucose (Table 4). Similar results were obtained for pericardial fat (data not shown).

For every 1-SD increase in intrathoracic fat, HDL was 3.7 mg/dL lower in women ($P<0.0001$) and 3.1 mg/dL lower in men ($P<0.0001$; Table 3); relations remained significant after BMI and WC were added to the model for intrathoracic and pericardial fat in women but only for intrathoracic fat in men. However, when both VAT and intrathoracic fat appeared in the model together, only VAT was significant (Table 4). Similar results were obtained for pericardial fat (data not shown) and for log triglycerides.

Similar patterns were noted for dichotomous traits (Tables 3 and 4). For every 1-SD increase in intrathoracic fat, the OR...
of MetS increased 2.87-fold in women and 2.24-fold in men. These relations remained significant after adjustment for BMI; however, when VAT and intrathoracic fat appeared in the same model, only VAT was significant. Similar results were obtained for pericardial fat (data not shown).

**Sex Interaction**

For nearly every trait examined, we observed a significant sex interaction (Table 3), which suggests that intrathoracic and pericardial fat are associated with more adverse risk factor profiles in women than men.

**Combination of Intrathoracic Fat and VAT**

The prevalence of metabolic risk factors is shown in the Figure by tertiles of VAT and additionally by tertiles of intrathoracic fat. In women and men separately, hypertension, diabetes mellitus, and MetS each increased in a stepwise manner across increasing tertiles of VAT, and within each tertile of VAT, each risk factor increased by increasing tertiles of intrathoracic fat. The probability value for trend of intrathoracic fat tertiles was statistically significant within the upper tertile of VAT for the risk factors of hypertension ($P=0.02$) and impaired fasting glucose ($P=0.009$) in women and for MetS ($P=0.02$) in men, after adjustment for age.

**Relations With Coronary and Abdominal Aortic Calcium**

Overall, 23.6% of the sample had CAC. In minimally adjusted models, significant associations were found for both intrathoracic and pericardial fat with CAC; no evidence was present for sex interactions. The OR for having CAC was similar for intrathoracic and pericardial fat in age- and sex-adjusted models (OR 1.29 and 1.28 per 1-SD increase in intrathoracic fat [$P=0.001$] and pericardial fat [$P=0.0004$] respectively; Table 5). Associations were unchanged after additional adjustment for VAT, and associations with pericardial fat but not intrathoracic fat persisted after additional adjustment for CVD risk factors, BMI, and WC (OR 1.21, $P=0.04$).

Overall, 29.7% of the sample had AAC. Significant sex interactions were not observed. Both intrathoracic and pericardial fat were associated with AAC after adjustment for age, sex, and VAT. After additional adjustment for CVD risk factors, BMI, and WC, only intrathoracic fat (OR 1.32, $P=0.03$), not pericardial fat (OR 1.12, $P=0.25$), was associated with AAC.

When intrathoracic fat was redefined with the exclusion of the pericardial fat compartment, the association with CAC was further attenuated (OR 1.10, 95% CI 0.86 to 1.41, $P=0.44$ in the fully adjusted model). Similarly, the association with AAC was strengthened (OR 1.37, 95% CI 1.07 to 1.75, $P=0.01$).

**Discussion**

Among individuals free of clinical CVD, pericardial fat was correlated with multiple measures of metabolic risk in a community-based setting, and correlations remained significant after adjustment for BMI and WC. However, associations with pericardial fat were attenuated when considered simultaneously with VAT with regard to metabolic risk, suggesting that pericardial fat may be an overall marker of visceral fat accumulation. Lastly, pericardial fat was associated with CAC and AAC even after metabolic risk factors and VAT were accounted for.

We tested 2 different protocols for pericardial fat ascertainment. For metabolic risk factors, effect sizes were consistently higher in magnitude for intrathoracic fat than for pericardial fat. However, for relations with CAC and AAC, we observed differential effects of intrathoracic fat and pericardial fat. Because pericardial fat represents the pericardial fat that is in direct proximity to the coronary vasculature, we hypothesized that associations between pericardial fat and CAC would be stronger than intrathoracic fat and CAC. Conversely, because the intrathoracic fat quantification includes adipose tissue surrounding the aorta, observed associations between intrathoracic fat and AAC may in part be driven by periaortic fat. However, relations between pericardial fat and CAC and AAC were
consistently stronger once VAT entered the model, which suggests potential complex interactions between different fat depots and vascular calcification that require further investigation.

**In the Context of the Current Literature**

Several small studies have reported correlations between pericardial fat, BMI,16 –19 metabolic risk factors,16,22 and VAT.20,21 In a sample of 72 consecutive subjects, Iacobellis et al.16 demonstrated an association between pericardial fat thickness and multiple CVD risk factors, including blood pressure, low-density lipoprotein cholesterol, glucose, and HDL cholesterol, in unadjusted analyses. We extend these findings by demonstrating strong correlations between pericardial fat volume and metabolic risk factors even after accounting for many potential confounders, including age, smoking, alcohol use, and physical activity.

Despite nonsignificant associations with CVD risk factors for pericardial compared with visceral fat when both were considered in the same model, pericardial fat was associated with CAC and AAC even after we accounted for concomitant CVD risk factors. The present findings are consistent with prior findings in selected subjects in studies that have demonstrated unadjusted associations between epicardial fat and coronary artery disease by angiography in primarily patient-based samples. Among 203 patients who underwent cardiac catheterization, epicardial fat thickness was correlated with the severity of angiographic coronary disease.19 Among individuals with human immunodeficiency virus, subepicardial adipose tissue thickness was associated with carotid intima-media thickness, a subclinical measure of atherosclerosis.30 Lastly, among 251 Japanese men who underwent chest-abdominal CT scanning, pericardial fat volume was associated with coronary artery disease by angiography.22 We extend these findings by relating pericardial fat volume among individuals free of clinical CVD and performing adjustment for multiple potential covariates in a large community-based sample of both women and men.
Potential Mechanisms
Epicardial fat is associated with increased expression of inflammatory markers, including monocyte chemotactic protein-1, interleukin-6, and tumor necrosis factor-α.31 CD45 expression is higher in epicardial fat than in subcutaneous or omental fat depots, and adiponectin expression is lower in epicardial fat depots.32 Thus, epicardial fat, located in close proximity to the coronary arteries, may have a locally toxic effect on the vasculature, as hypothesized by Yudkin et al.33 Perivascular fat might contribute directly to perivascular inflammation34 and smooth muscle cell proliferation.35 Perivascular fat has also been shown to decrease vascular contractility in rats,36,37 another mechanism by which epicardial fat may exert locally toxic influences on the coronary arteries. Furthermore, epicardial fat has been shown to be associated with insulin resistance,23 and myocardial fat depots are associated with serum free fatty acid levels.38 Therefore, the present finding that only intrapericardial fat is associated with CAC supports the notion that epicardial fat is an ectopic fat depot located in close proximity to the coronary arteries, which may exert deleterious perivascular effects.

Strengths and Limitations
Strengths of the present study include the community-based sample with participants not enriched for adiposity-related traits. Furthermore, we developed protocols and report reproducibility data for both intrathoracic and pericardial fat. Pericardial fat and VAT were quantified by volumes instead of a measure of thickness. We had a simultaneous measure of VAT, which allowed us to compare the effects of pericardial fat with visceral fat. Among the study limitations, the Framingham Offspring study is primarily a sample of whites, and therefore, generalization to other ethnic groups is uncertain. Furthermore, the present study design was cross-sectional, and we cannot infer causality. The CT measurements were collected on average 4 years after the clinical data were collected. This gap could result in misclassification of risk factors and therefore lead to an underestimation of the magnitude of our reported associations; however, this should not impact the relative association of pericardial fat compared with visceral fat.

Implications
VAT has been shown to be a stronger correlate of cardiovascular risk than systemic adiposity indexes, WC, or subcutaneous abdominal fat.14 Here, we demonstrate that pericardial fat volumes are well correlated with cardiovascular risk factors and that the associations with pericardial fat are independent of BMI, WC, age, smoking, and alcohol consumption. Pericardial fat can be quantified during chest CT without incurring excess abdominal radiation. Therefore, pericardial fat may yield additional information on metabolic risk, which may be particularly useful when abdominal imaging with VAT quantification is not feasible. Our finding that pericardial fat is associated with CAC suggests that further research relating perivascular fat depots to target end-organ damage should be pursued to identify potential mechanisms of obesity-related atherosclerosis.

Conclusions
Pericardial fat is correlated with multiple measures of adiposity and CVD risk factors, but VAT is a stronger correlate of most metabolic risk factors. However, pericardial fat is associated with vascular calcification, which suggests that pericardial fat may exert local vascular effects.
Sources of Funding

This work was supported by the Framingham Heart Study of the National Heart, Lung, and Blood Institute (grant N01-HC-25195). Dr Rosito is supported by a grant from CAPES/Brazil.

Disclosures

None.

References


Clinical Perspective

Pericardial fat may be an important mediator of metabolic risk. Correlations with cardiovascular disease risk factors and vascular calcification in a community-based sample are lacking. We sought to examine associations between pericardial fat, metabolic risk factors, and vascular calcification. Participants from the Framingham Heart Study underwent quantification of intrathoracic fat, pericardial fat, visceral abdominal fat, and coronary artery and aortic artery calcification. Intrathoracic and pericardial fat were directly correlated with body mass index and visceral abdominal fat. Both intrathoracic and pericardial fat were associated with higher triglycerides, lower high-density lipoprotein, hypertension, impaired fasting glucose, diabetes mellitus, and metabolic syndrome after multivariable adjustment. Associations generally persisted after additional adjustment for body mass index and waist circumference but not after adjustment for visceral abdominal fat. Pericardial fat, but not intrathoracic fat, was associated with coronary artery calcification, whereas intrathoracic fat, but not pericardial fat, was associated with aortic artery calcification. Pericardial fat is correlated with multiple measures of adiposity and cardiovascular disease risk factors, but visceral abdominal fat is a stronger correlate of most metabolic risk factors. However, intrathoracic and pericardial fat are associated with vascular calcification, which suggests that these fat depots may exert local toxic effects on the vasculature.
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