The relationship between body composition and structural changes at the knee

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Abstract

Objective. Obesity is an important risk factor for knee OA. Evidence suggests that fat and muscle have differential effects on the pathogenesis of disease. The aim of this study was to examine the relationship between body composition and knee structure, including knee cartilage volume, defects and bone marrow lesions (BMLs).

Methods. A total of 153 subjects aged 25–60 years, 81% females, were recruited across a range of BMI (18–55 kg/m²) for a study examining the relationship between obesity and musculoskeletal disease. MRI was performed of the dominant knee. Cartilage volume, defects and BMLs were measured using validated methods. Body composition was measured using dual X-ray absorptiometry.

Results. There was an 81 (95% CI: 69, 94) mm³ increase in cartilage volume for every 1 kg increase in skeletal muscle mass. Fat mass was not significantly associated with cartilage volume. Fat mass, but not skeletal muscle mass, was a risk factor for cartilage defects and BMLs. For every 1 kg increase in total body fat there was an increased risk of cartilage defects (OR = 1.31, 95% CI: 1.04, 1.64) and BMLs (OR = 1.09, 95% CI: 1.01, 1.18).

Conclusions. In this relatively healthy population, fat mass was associated with increased cartilage defects and BMLs, which are features of early knee OA. In contrast, skeletal muscle mass was positively associated with cartilage volume, which may be due to coinheritance, a commonality of environmental factors associated with cartilage accrual or a protective effect of increased muscle.

Key words: Osteoarthritis, Obesity, MRI, Bone marrow lesions, Cartilage.

Introduction

Obesity is a well-recognized risk factor for OA [1–3], and the burden of this disease is only expected to increase with the world-wide obesity epidemic. Although weight loss assessed by a decrease in BMI has been shown to reduce the risk of knee OA [2], emerging evidence suggests that the mechanism by which obesity increases the risk of OA is more than simply an effect of load on the joint [4, 5]. This has important implications for weight loss strategies, suggesting that simple reduction in weight alone may not be the most efficient strategy to prevent knee OA. Given that obesity is an important modifiable risk factor for OA, understanding the mechanism by which obesity increases the risk of OA is essential.

BMI, a measure that does not discriminate adipose from non-adipose body mass [6, 7] has been most commonly used in studies examining the relationship between obesity and OA [1–3]. Increasing BMI has been associated with early structural changes measured from MRI including the presence, severity [8] and worsening of cartilage defects over a 2-year period [9], as well as the prevalence of bone marrow lesions (BMLs) [10]. However, the relationship between increasing BMI and OA may not simply
be due to weight-related increased load on the joint, since BMI has been associated with the development of OA in non-weight-bearing joints such as the hands [4, 5].

In contrast to BMI per se, there is evidence to suggest that muscle mass protects the knee joint with increased quadriceps strength protecting against incident knee OA [11, 12]. Building on this, we have previously shown that muscle mass was associated with increased tibial cartilage volume [13–15] and reduced cartilage loss over 2 years in healthy, normal weight populations [14]. However, the relationship between specific measures of fat mass and distribution and the risk of knee OA is less clear, with several previous studies showing no significant association [3, 16–18]. We have recently shown that there was a 3- to 4-fold increased risk of primary joint replacement associated with body weight, BMI, fat mass and percentage fat, waist circumference and waist-to-hip ratio [19]. Consistent with this, a recent study identified a negative association between increasing fat mass and cartilage volume, which was dependent on leptin [15]. Taken together, these results suggest that some of the association between obesity and OA may be hormonally mediated.

Despite emerging evidence suggesting that measures of body composition may be better predictors of OA incidence and severity compared with BMI [20], the relative contribution of fat and muscle mass, which may affect the joint differently, has received little attention. No previous study has examined whether the increased risk of cartilage defects and BMLs related to obesity is purely attributable to increased mass, or whether muscle and fat differ in their effect on early structural changes of knee OA. Moreover, the relationship between body composition and knee joint structures measured from MRI has been limited primarily to normal or overweight populations, and has yet to include obese subjects. We hypothesize that muscle mass will have a beneficial effect on knee joint structures, and fat mass will have a detrimental effect. Therefore, the aim of this study was to examine the relationship between body composition measures and knee structure in a relatively healthy population that ranged from normal weight to obese.

Methods

Subjects

A total of 153 subjects aged 25–60 years, who ranged from normal weight to obese, were recruited to take part in a study examining the relationship between obesity and musculoskeletal disease. Participants were recruited through the local media, public, private and community weight loss clinics. Exclusion criteria included physician-diagnosed arthritis, prior surgical intervention to the knee, previous significant knee injury requiring non-weight-bearing therapy, knee pain precluding weight-bearing activity for ≥24 h or prescribed analgesia, malignancy, inability to complete the study or contraindication to MRI. The study was approved by the Alfred Human Research and Ethics Committee, the Monash Standing Research Ethics Committee, the Austin Health HREC and the University of Melbourne Central HREC. Informed consent was obtained from all participants.

Anthropometric and physical activity data

Weight was measured to the nearest 0.1 kg using a single pair of electronic scales. Height was measured to the nearest 0.1 cm using a stadiometer. BMI (weight/height2 kg/m2) was calculated. Strenuous physical activity was assessed by asking ‘On how many days during the last 14 days did you spend at least 20 minutes doing strenuous exercise?’ E.g. bicycling, brisk walking, etc. that was severe enough to raise your pulse rate, cause you to breathe faster’ with frequency options: no days, 1–2 days, 3–5 days, 6–8 days, ≥9 days. Participation in strenuous activity for ≥3 days was categorized as performing strenuous activity.

Body composition

Body composition was measured using DXA (GE Lunar Prodigy; using operating system version 9; Madison, WI, USA). The machine has a weight limit of ~130 kg. Standard regional analyses were used to measure total body fat and lean tissue mass. Total limb lean tissue mass was calculated as the sum of upper limb lean tissue mass and lower limb lean tissue mass, which was then converted to total body skeletal muscle mass using a prediction model developed and validated in adults (BMI < 35) [21]. This measure excludes BMC. Short-term coefficients of variation, assessed in 15 normal young adults was 1.2% for total body fat mass [22].

MRI

MRI of the dominant knee was performed [23]. Knees were imaged in the sagittal plane on a 1.5-T whole-body magnetic resonance unit (Philips Medical Systems, Eindhoven, The Netherlands) using a commercial transmit-receive extremity coil. The weight limit for the machine is 150 kg. The following sequence and parameters were used: T1-weighted fat saturation 3D gradient recall acquisition in the steady state (58 ms/12 ms/55°, repetition time/echo time/flip angle) with a 16 cm field of view, 60 partitions, 512 × 512 matrix and acquisition time 11 min 56 s (one acquisition). Sagittal images were obtained at a partition thickness of 1.5 mm and an in-plane resolution of 0.31 × 0.31 mm (512 × 512 pixels). A coronal fat-saturated, fast spin echo three-dimensional, T2-weighted acquisition (2200 ms, 20/80 ms/90° repetition time/echo time/flip angle) with a slice thickness of 3 mm, a 0.3 inter-slice gap, 1 excitation, a field of view of 13 cm and a matrix of 256 × 192 pixels was also obtained [24].

Cartilage volume

Cartilage volume was determined by manually drawing disarticulation contours around the cartilage boundary, using independent workstation software Osiris (Geneva University Hospital, Geneva, Switzerland). Measurement was done by one trained observer with random cross-checks blindly performed by an independent
Cartilage defects

Cartilage defects were graded on the MR images with a classification system previously described in the medial and lateral tibial and femoral cartilages by a single trained observer, who measured all images in duplicate on separate occasions. The grading was as follows: Grade 0, normal cartilage; Grade 1, focal blistering and intra-cartilaginous low-signal intensity area with an intact surface and bottom; Grade 2, irregularities on the surface or bottom and loss of thickness <50%; Grade 3, deep ulceration with loss of thickness >50%; Grade 4, full-thickness cartilage wear with exposure of subchondral bone. A cartilage defect also had to be present in at least two consecutive slices. The medial and lateral tibiofemoral cartilage defects scores were summed to create a total knee tibiofemoral cartilage defects score. Intra-observer reliability (expressed as intra-class correlation coefficient, ICC) was 0.90 and 0.89 for the medial and lateral tibiofemoral compartments, respectively. A cartilage defect was considered absent if the total score was <2, and present if the score was ≥3. ICC was 0.90 and 0.85 for the medial and lateral tibiofemoral compartments, respectively [26].

Bone area

Plateau area was determined using the independent workstation Osiris, by creating an isotropic volume from the input images, which were reformatted in the axial plane. One trained observer performed the measurements, with random cross-checks blindly performed by an independent trained observer. The CV was 2.3% [25].

BMLs

BMLs were defined as areas of increased signal intensity adjacent to subchondral bone present in the tibia assessed on coronal T2-weighted fat-saturated images. A BML was defined as present if it appeared on two or more adjacent slices [27]. One trained observer, blinded to patient characteristics, assessed the presence or absence of lesions. The reproducibility for determination was assessed using 50 randomly selected knees (κ = 0.81, P < 0.001).

Osteophytes

Osteophytes were measured from MR images, which have been shown to be more sensitive than X-rays [28]. Osteophytes were measured from coronal images by two independent trained observers. In the event of disagreement between observers, a third independent observer reviewed the MRI. Intra-observer and inter-observer reproducibility for agreement on osteophytes ranged between 0.85 and 0.93 (κ-statistic).

Statistical analysis

Cartilage volume was initially assessed for normality, and the distribution of this outcome variable was found to approximate the normal distribution. Linear regression was used to assess the relationship between body composition measurements and cartilage volume as a continuous variable. Adjustment was made for potential confounders including age, gender, BMI, the presence of osteophytes and participation in strenuous activity. The correlation between BMI and body composition measurements was determined using Pearson’s correlation coefficient. As BMI and body composition measurements were highly correlated, collinearity between these variables was assessed by computing tolerance for each model, where tolerance of < 0.2 warrants caution. If tolerance was not within acceptable limits (i.e., < 0.2), height was included in the model instead of BMI to reduce collinearity. The relationship between body composition and tibiofemoral cartilage defects score was examined using ordinal logistic regression. Logistic regression was used to examine the relationship between body composition and the presence/absence of BMLs. Multiple regression was used to adjust for potential confounders. The independent samples z-test was used to compare the relationship between body composition measures and cartilage volume in men and women. A P < 0.05 (two-tailed) was considered to be statistically significant. With a sample size of 153 subjects, this study has 80% power to show a correlation as low as 0.26 between body composition and knee cartilage volume (α-error of 0.05, two-sided significance), thus explaining up to 7% of the variance of knee cartilage volume. All analyses were performed using the SPSS statistical package (standard version 15.0; SPSS, Chicago, IL, USA).

Results

Descriptive characteristics of study population

The characteristics of the 153 subjects (81% women) who participated in the study are presented in Table 1. BMI was approximately normally distributed in this population, with a mean (±S.D.) of 32 (9). The relationship between body composition measures and tibial cartilage volume

Measures of fat mass including total body, android, gynoid and trunk, were not significantly associated with tibial cartilage volume in univariate or multivariate analysis adjusting for potential confounders (Table 2). As BMI was highly correlated with total body fat mass, diagnostic tests for collinearity were performed. Since tolerance of 0.13 was found for total body fat mass, which was not considered within acceptable limits, height was included in the multivariate model instead of BMI.

Weak correlations were obtained between BMI and skeletal muscle mass (r = 0.29), and the diagnostic tests for collinearity yielded a tolerance value that was not considered within acceptable limits (<0.20). Thus, height was included in this multivariate model. In univariate analyses, for every 1 kg increase in skeletal muscle mass (which does not include BMC), there was a 81 mm³ increase in tibial cartilage volume (95% CI: 69, 94; P < 0.001; Table 2).
These associations persisted after adjusting for potential confounders including age, gender, BMI, the presence of osteophytes, tibial plateau area and participation in strenuous activity ($P < 0.001$). Similar results were obtained when subjects with BMI $> 35$ were excluded from the analysis (regression coefficient $\beta = 43$; 95% CI: 7, 80; $P = 0.02$). The difference in the relationship between body composition measures and cartilage volume in men and women was not statistically significant ($P > 0.45$).

The relationship between body composition measures and tibiofemoral cartilage defects

The mean total body fat mass was significantly higher in subjects with tibiofemoral cartilage defects compared with those without defects ($P = 0.05$; Fig. 1A). In contrast, there was no significant difference in mean skeletal muscle mass between subjects with and without tibiofemoral cartilage defects ($P = 0.12$).

In univariate analysis, for every 5 kg increase in total body fat (OR $= 1.11$; 95% CI: 1.02, 1.20; $P = 0.02$), and

**Table 2** Relationship between body composition measures and knee joint structures

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Univariate regression coefficient (95% CI)</th>
<th>P-value</th>
<th>Multivariate regression coefficient (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cartilage volume$^a$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m$^2$</td>
<td>0.58 (−10.67, 11.83)</td>
<td>0.92</td>
<td>3.22 (−5.94, 12.39)</td>
<td>0.49</td>
</tr>
<tr>
<td>Measures of fat mass</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total body</td>
<td>−1.4 (−7.1, 4.3)</td>
<td>0.63</td>
<td>1.8 (−2.7, 6.4)</td>
<td>0.77</td>
</tr>
<tr>
<td>Android</td>
<td>−0.70 (−5.5, 53)</td>
<td>0.98</td>
<td>−62 (−162, 37)</td>
<td>0.22</td>
</tr>
<tr>
<td>Gynoid</td>
<td>−20 (−55, 16)</td>
<td>0.27</td>
<td>28 (−30, 86)</td>
<td>0.34</td>
</tr>
<tr>
<td>Trunk</td>
<td>0.12 (−11, 11)</td>
<td>0.98</td>
<td>−6.9 (−27, 13)</td>
<td>0.50</td>
</tr>
<tr>
<td>Measure of muscle mass</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skeletal muscle mass</td>
<td>81 (69, 94)</td>
<td>&lt;0.001</td>
<td>46 (23, 69)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Tibiofemoral cartilage defects score$^b$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m$^2$</td>
<td>1.02 (0.97, 1.07)</td>
<td>0.45</td>
<td>1.02 (0.98, 1.06)</td>
<td>0.29</td>
</tr>
<tr>
<td>Measure of fat mass</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Total body</td>
<td>1.11 (1.02, 1.20)</td>
<td>0.02</td>
<td>1.31 (1.04, 1.64)</td>
<td>0.02</td>
</tr>
<tr>
<td>Android</td>
<td>1.29 (1.02, 1.58)</td>
<td>0.03</td>
<td>1.47 (1.09, 1.97)</td>
<td>0.05</td>
</tr>
<tr>
<td>Gynoid</td>
<td>1.11 (1.01, 1.23)</td>
<td>0.04</td>
<td>1.21 (0.97, 1.52)</td>
<td>0.10</td>
</tr>
<tr>
<td>Trunk</td>
<td>1.04 (1.01, 1.07)</td>
<td>0.01</td>
<td>1.11 (1.02, 1.20)</td>
<td>0.01</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skeletal muscle mass</td>
<td>1.04 (0.99, 1.10)</td>
<td>0.12</td>
<td>1.08 (0.95, 1.22)</td>
<td>0.23</td>
</tr>
<tr>
<td>BMLs$^c$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m$^2$</td>
<td>1.02 (0.97, 1.07)</td>
<td>0.45</td>
<td>1.02 (0.96, 1.07)</td>
<td>0.60</td>
</tr>
<tr>
<td>Measures of fat mass</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total body</td>
<td>1.03 (1.01, 1.05)</td>
<td>0.03</td>
<td>1.09 (1.01, 1.18)</td>
<td>0.03</td>
</tr>
<tr>
<td>Android</td>
<td>1.18 (0.94, 1.47)</td>
<td>0.15</td>
<td>1.26 (0.69, 2.32)</td>
<td>0.45</td>
</tr>
<tr>
<td>Gynoid</td>
<td>1.16 (1.00, 1.34)</td>
<td>0.05</td>
<td>1.37 (0.95, 1.98)</td>
<td>0.09</td>
</tr>
<tr>
<td>Trunk</td>
<td>1.04 (0.99, 1.09)</td>
<td>0.06</td>
<td>1.10 (0.97, 1.24)</td>
<td>0.14</td>
</tr>
<tr>
<td>Measure of muscle mass</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skeletal muscle mass</td>
<td>0.98 (0.90, 1.06)</td>
<td>0.56</td>
<td>1.11 (0.92, 1.34)</td>
<td>0.26</td>
</tr>
</tbody>
</table>

$^a$Tibial cartilage volume (mm$^3$) per unit increase in the respective body composition measurement. Age, gender, BMI, presence of osteophytes, tibial bone area and participation in strenuous activity included in the multivariate model. Collinearity identified between total body fat mass, skeletal muscle mass and BMI, and thus height included in the multivariate model. $^b$OR per unit increase in the respective body composition measurement. Age, gender, BMI, tibial cartilage volume, presence of osteophytes and participation in strenuous activity included in the multivariate model. Per 5 kg increase in total body fat. $^c$OR per unit increase in the respective body composition measurement. Age, gender, BMI and presence of osteophytes included in the multivariate model.
for every 1 kg increase in android (OR = 1.31; 95% CI: 1.04, 1.64; P = 0.02), gynoid (OR = 1.11; 95% CI: 1.01, 1.23; P = 0.04) and trunk (OR = 1.04; 95% CI: 1.01, 1.07; P = 0.01) fat mass, there was an associated increase in tibiofemoral cartilage defects score. After adjusting for potential confounders, this association persisted for total body (OR = 1.09; 95% CI: 1.01, 1.18; P = 0.03), android (OR = 1.47; 95% CI: 0.99, 2.17; P = 0.05) and trunk fat (OR = 1.11; 95% CI: 1.02, 1.20; P = 0.01), with a similar trend for gynoid (OR = 1.21; 95% CI: 0.97, 1.52; P = 0.10) fat mass.

In univariate and multivariate analyses, no significant association was obtained between skeletal muscle mass and the tibiofemoral cartilage defects score. After adjusting for potential confounders, this association persisted for total body fat mass (OR = 1.03; 95% CI: 1.01, 1.05; P = 0.03), android (OR = 1.09; 95% CI: 1.01, 1.09; P = 0.01), and trunk fat (OR = 1.02; 95% CI: 1.00, 1.04; P = 0.01), with a similar trend for gynoid (OR = 1.05; 95% CI: 0.98, 1.13; P = 0.10) fat mass.

In univariate analysis, for every 1 kg increase in total body fat mass (OR = 1.03; 95% CI: 1.01, 1.05; P = 0.03), there was an associated increased presence of BMLs (Table 2). This result persisted after adjusting for age, gender, BMI and the presence of osteophytes (OR = 1.09; 95% CI: 1.01, 1.18; P = 0.03). No significant associations were seen between android, gynoid and trunk fat and BMLs after adjusting for potential confounders.

No significant association was found between skeletal muscle mass and the presence of BMLs in either univariate or multivariate analyses (Table 2). Similar results were obtained when subjects with BMI > 35 were excluded from the analysis.

The relationship between body composition measures and the presence/absence of BMLs

The mean total body fat mass was significantly higher in subjects with BMLs compared with those without BMLs (P = 0.03; Fig. 1B). In contrast, there was no significant difference in mean skeletal muscle mass between subjects with and without BMLs (P = 0.67).

In univariate analysis, for every 1 kg increase in total body fat mass (OR = 1.03; 95% CI: 1.01, 1.05; P = 0.03), there was an associated increased presence of BMLs (Table 2). This result persisted after adjusting for age, gender, BMI and the presence of osteophytes (OR = 1.09; 95% CI: 1.01, 1.18; P = 0.03). No significant associations were seen between android, gynoid and trunk fat and BMLs after adjusting for potential confounders.

No significant association was found between skeletal muscle mass and the presence of BMLs in either univariate or multivariate analyses (Table 2). Similar results were obtained when subjects with BMI > 35 were excluded from the analysis.

Discussion

In this study, measures of fat mass were positively associated with increased cartilage defects and BMLs, while
no significant association was found with skeletal muscle mass. Skeletal muscle mass was positively associated with tibial cartilage volume.

The mechanism by which excess fat mass mediates the relationship between obesity and early deleterious structural changes at the knee joint may be via either increased load on the joint, a metabolic effect or a combination of both. In contrast, skeletal muscle mass is associated with increased cartilage volume, which may be due to co-inheritance, a commonality of lifestyle or environmental factors between cartilage and muscle, or a protective effect of increased muscle on the joint. These complex relationships between body composition and knee structure warrant further investigation.

In this study of a relatively healthy population, we found that fat mass, but not skeletal muscle mass, was significantly higher in subjects with increased cartilage defects and BMLs, which are features of early knee pathology. Cartilage defects are an early feature of cartilage pathology and have been shown to be associated with knee pain [29], to predict cartilage loss in both healthy, asymptomatic populations [30] and in those with OA and to predict joint replacement independent of cartilage volume [31]. BMLs are associated with knee pain [29, 32, 33] and progression of knee OA [34–36]. Although previous studies have shown a relationship between BMI and cartilage defects and BMLs, this is the first study to show that the relationship is associated with fat mass.

The mechanism by which obesity affects these early structural changes may be via increased load on the joint due to excess fat, with skeletal muscle mass remaining relatively stable. Alternatively, the effect of fat mass may occur via systemic processes. This is supported by our findings that measures of fat mass throughout the body, including those of the trunk and android fat, were associated with cartilage defects. Indeed, there is growing evidence to suggest a metabolic link between obesity and joint integrity, since BMI has been associated with the development of OA in non-weight-bearing joints such as the hands [4, 5]. It is now well established that adipose tissue is metabolically active, releasing a multitude of pro-inflammatory cytokines including TNF, IL-1, as well as key mediators of metabolism termed the adipokines [37]. Given that BMLs have a mixed histopathology including granulation, oedema, diffuse necrosis, fibrinoid deposition, hyperplasia of blood vessel walls [38] and more recently evidence of ischaemia and reperfusion injury [39, 40], it is possible that inflammatory factors released from adipose tissue contribute to these vascular changes. Moreover, recent evidence suggests a mediating role of the adipokine leptin in the relationship between obesity and cartilage volume [15]. A similar relationship may exist for earlier signs of cartilage pathology, such as defects.

While fat mass was associated with early structural changes at the knee, we did not find a significant association between fat mass and cartilage volume. Indeed, we found a positive association between skeletal muscle mass and cartilage volume. This is consistent with previous cross-sectional studies that have shown that different measures of muscle mass were associated with reduced risk of radiographic knee OA [18, 20] and increased tibial cartilage volume [13, 14]. This work is further supported by longitudinal data in healthy, asymptomatic subjects, whereby increased muscle mass was associated with reduced cartilage loss over 2 years [14] and increased quadriceps strength protected against incident knee OA [11, 12].

The mechanism by which skeletal muscle, but not fat mass is associated with cartilage volume remains unclear. It may be that in this relatively healthy population, the effect of fat mass on cartilage volume loss takes time to develop, occurring downstream from the effect on early structural changes such as cartilage defects and BMLs. Thus, it may be that the positive association between cartilage volume and skeletal muscle mass may in part reflect co-inheritance. However, this finding cannot completely be explained by body size, as we included height in the model. Alternatively, this relationship may reflect common lifestyle or environmental factors such as physical activity, which similarly affect both cartilage and muscle. It is also possible that muscle contributes to greater joint stability and even load distribution [41], which may in turn produce an optimal biomechanical environment that confers beneficial effects on cartilage. Longitudinal research will be required to clarify this relationship.

A limitation of this study is that it is a cross-sectional study; future longitudinal studies will be needed to confirm these findings. Although we did not have knee radiographs, we determined the presence of osteophytes from MRI. This has previously been shown to be a more sensitive method for determining the presence of osteophytes than radiography [28]. In this population, only 16% of subjects had knee osteophytes, thus the population consisted of relatively healthy subjects. Our findings remained unchanged after adjusting for osteophytes. Another potential limitation is this study included 80% women. Strengths of this study include examining a population with a spectrum of weight extending from normal weight to obese, and also using very sensitive, validated measures of knee structure. Moreover, the relationships between body composition measurements and knee structures were independent of body size. BMI was used except where there was significant collinearity, in which case height was used in the model instead of BMI.

In this study, skeletal muscle mass was positively associated with tibial cartilage volume, while measures of fat mass were associated with features of early knee pathology including cartilage defects and BMLs, both of which may be seen in early OA. Thus, these findings warrant further investigation, since it may be that attention to muscle preservation during weight loss may be more effective in reducing the risk of knee OA than simple weight loss alone. The role of fat mass also warrants further attention as this may act via a metabolic effect which may, in turn, inform alternative therapeutic approaches.
Fat mass was associated with increased cartilage defects and BMLs, which are features of early knee OA.

Skeletal muscle mass was positively associated with cartilage volume.

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References


23 Hanna FS, Bell RJ, Davis SR et al. Familial, structural, and environmental correlates of MRI-defined bone marrow lesions, which are features of early knee OA. J Rheumatol 2001;28:1244–8.


38 Hunter DJ, Gerstenfeld L, Bishop G et al. Bone marrow lesions from osteoarthritis knees are characterized by sclerotic bone that is less well mineralized. Arthritis Res Ther 2009;11:R11.

