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BMI vs. body composition and radiographically-defined osteoarthritis of the knee in women: a 4-year follow-up study

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Abstract

Objective—To elucidate the role of body mass index (BMI) and knee osteoarthritis (OAK) by evaluating measures of body composition including fat mass and skeletal muscle mass (SMM).

Methods—Data is from 541 women enrolled in the Michigan Bone Health Study, a longitudinal, population-based study. At visits in 1998 and 2002, radiographs were taken of both knees and were evaluated for the presence of OAK (≥ 2 on the Kellgren and Lawrence (K-L) scale). Joint space width (JSW) was measured with electronic calipers. Fat mass and SMM were determined using bioelectrical impedance analysis.

Results—In 2002, the prevalence of OAK was 11% in this population of women, whose mean age was 47 years. Fat mass, lean mass, SMM, waist circumference and BMI was greater in women with OAK compared to those without OAK. In multiple variable analyses adjusted for age, fat mass and SMM explained OAK prevalence and increasing OAK severity better than models with BMI; further SMM explained more variation than did fat mass. SMM was positively associated with level of left and right medial JSW while there was no consistent association of JSW and BMI or fat mass.

Conclusion—Fat mass and SMM were associated with K-L OAK score and the amount of joint space, with more variation explained by SMM. SMM was highly associated with JSW. Therefore, though obesity, frequently characterized with BMI, is a frequently reported risk factor for OAK, this mis-attribution may mean that interventions that focus on weight loss as treatment for OA should be aware that this may negatively impact muscle mass.

Keywords

osteoarthritis; body mass index; body composition; fat mass; skeletal muscle mass

INTRODUCTION

Obesity, usually characterized by body mass index (BMI), is considered a major risk factor for prevalent osteoarthritis (OA)[1–7]. Data from the Chingford general population survey suggests that women in the highest tertile of BMI have 6-fold increased odds of knee osteoarthritis

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(OAK), and nearly 18 times increased odds of bilateral OAK, compared to women in the lowest tertile of BMI[3]. Longitudinal studies show that increased weight precedes the presentation of OAK. In a longitudinal study of men and women aged 40–64 years, Manninen et al.[5] found that every standard deviation increase in BMI (3.8 kg/m^2) was associated with a relative risk of 1.4 (95% CI, 1.2–1.5) for developing OAK.

However, not all obese persons develop OAK, nor are all individuals with OAK obese, suggesting that other factors aside from obesity, defined by BMI, are important. It has also been proposed that muscle mass or muscle strength is protective for the development of OA[8–10].

Because BMI is a measure of both fat and lean mass, the relative contribution of adipose tissue and muscle mass, and their contribution to muscle strength, cannot be disaggregated. Studies limited to the use of BMI as a measure of body composition may unduly impede our understanding of the mechanisms associated with the development and progression of OAK. Use of BMI does not adequately provide a means of understanding the physiological role that could be relevant for OA including joint loading or more systemic biochemical factors. For example, muscle strength, assessed as torque, reflects the capacity to do work and is strongly influenced by body mass, but is differentially expressed with respect to skeletal muscle mass vs. total body mass.

Based on an anticipation of the constraints of BMI, we related body composition measures to the development and progression of osteoarthritis of the knee. We addressed the following hypotheses: (1) we hypothesized that in middle-aged women, variation in the development and progression of radiographically-determined OAK is better explained by individual body composition measures such as fat mass, skeletal muscle mass and waist circumference as compared to BMI, a generalized measure of obesity; and (2) we hypothesized that body composition measures would explain more variation in two measures of knee osteoarthritis, including the osteophyte-based Kellgren-Lawrence OA ordinal classification or the amount of joint space width (JSW) and its change than would BMI.

MATERIALS AND METHODS

Study population

This sample is from women enrolled in the Michigan Bone Health Study (MBHS), a longitudinal, population-based study conducted among women living in and around Tecumseh, Michigan. The sampling frames for the study were the historical family records of the Tecumseh Community Health Study (TCHS) which was a population-based, prospective cohort study established in 1959 to study risk factors for common chronic and infectious diseases. Women eligible for MBHS were the daughters of TCHS participants who, in 1988, were between the ages of 20 and 40 years, not pregnant and premenopausal. These women were contacted using letters, telephone calls and in-person visits. There were 539 women successfully recruited into the MBHS, a participation rate in excess of 80 percent. In 1992, a second sampling frame based on a community census of Tecumseh was developed to include women whose parents had not participated in the TCHS. As a result, an additional 121 women in the desired age range of 24 to 44 years (of a possible 135 eligible) were recruited (90 percent participation rate), for a total of 660 participants.

Knee x-rays were taken as a part of the 1992, 1995/6, 1998/9 and 2002/3 annual data collections, with the 1998 and 2002 collections taken using a semi-flexed positioning[11]. To avoid drawing conclusions that may be based on positioning alone, only the 1998 and 2002 time points are used for these analyses and included a total of 541 women. For the purposes of this study, women could be either lost or gained to follow-up between the 1998 and 2002 visits. However,

the cohort was stable; of those women in the 1998 data set with OA and body composition measures, 88% of them had data in the 2002 group.

Further, women lost to follow-up or recovered were similar to each other in terms of OA prevalence, overweight prevalence and age. For example, of the women lost to follow-up (i.e., had data in 1998 but not in 2002), 13% of them had OA compared to 16% of those recovered in 2002. The weights were also similar: 62.1% of those lost to follow-up were overweight in 1998 (as defined by BMI $\geq$ 25) while of those recovered in 2002, 66% were overweight.

Women were ineligible for an annual data collection if they were pregnant; participants who were excluded for pregnancy became eligible again for subsequent data collection as long as they were not pregnant. The University of Michigan Institutional Review Board approved the study protocol, and written informed consent was obtained from each participant.

Osteoarthritis measures

Weight-bearing anterio-posterior radiographs in a semi-flexed position[11] were taken of both knees using General Electric radiographic equipment (model X-GE MPX-80; General Electric Medical Systems, Milwaukee, WI) and Kodak film (X-DA with Kodak rare earth intensifying screens, Eastman Kodak, Rochester, NY). The source film distance was 40 inches and standard radiographic techniques were used. Radiographs were evaluated by at least two readers with a third consensus reader for the presence of OA defined by the Kellgren and Lawrence (K-L) scale depicted in the Atlas of Standard Radiographs of Arthritis (0=normal, 1=doubtful OA, 2=minimal OA, 3=moderate OA, and 4=severe OA)[12]. This scale is based on the degree of osteophyte formation, joint space narrowing, sclerosis, and joint deformity. OA was defined as the presence of at least 1 knee with a grade of 2 or higher. Apart from the K-L criteria, joints could also be classified as showing changes consistent with rheumatoid arthritis, missing, or unable to evaluate.

To promote reproducibility over the period of observation, readers reviewed the K-L grading criteria and evaluation films that were representative of each K-L level of OA. There were 25 knee radiographs that were evaluated independently by each reader and their results were compared for consistency. After standardization procedures were completed, two readers (JJ, DJ), both board-certified musculoskeletal radiologists, independently evaluated x-rays and classified both knees. Scores from 2 readers were compared and any score that was not congruent was reread and, if necessary, subjected to consensus evaluation. Further, a sample of 110 knee radiographs that had been used in previous evaluations was again read to assess the potential for drift in scoring over time. Films were not read side-by-side to minimize the likelihood of having correlated errors.

Joint space width (JSW) was measured on both the medial and lateral aspect of each knee radiograph with electronic calipers. Measurement locations were ascertained by identifying the centerline of each joint using the medial and lateral tibial condyle edge and then establishing points that were 50% and 75% between the centerline and the condyle edge. Ten percent of radiographs were remeasured for quality control. The 4-year difference in JSW was established by subtracting the JSW values in 1998 from those ascertained in 2002.

Body composition measures

Fat mass and skeletal muscle mass were determined from the impedance and conductance measures of the bioelectrical impedance analysis (BIA). BIA is based on measurement of the transmission speed of a ¼ volt electrical pulse between electrodes attached at the feet and across the knuckles of the hand. Because fat-free mass is comprised of water, proteins and electrolytes, conductivity is greater in fat-free mass than in fat mass[13]. Resistance and reactance are used

to estimate total body water, and by extension, fat mass and lean mass, with the latter including bone[14]. The CV% of the resistance and reactance measures are less than 2% each in a reproducibility study of 20 women similar to the population being characterized. Skeletal muscle mass (SMM) was calculated by the method of Janssen[15] who subsequently indexed SMM to height for a skeletal muscle index (SMI) and developed cut points relating to the risk of disability associated with SMI[16]. These variables were available from annual assessments and were treated as time-varying covariates.

Weight and height, measured annually with a calibrated balance beam scale and stadiometer, were used to calculate BMI [weight (kg)/height $(m)^2$]. Waist circumference (cm) was measured annually with a non-stretching tape at the narrowest point of the mid-torso at maximum inhalation. Elbow breadth (cm) was assessed as an index of skeletal frame size using a Martin calipers.

Data analysis

Univariate distributions of the eight continuous measures of body composition were examined for normality. To meet the assumptions of normality and to reduce skewness, natural log transformations were applied to the body composition measures of fat mass, skeletal muscle mass, and waist circumference. The frequencies of the K-L score for OAK and categorical covariates were examined overall and by year of visit.

Repeated measures mixed-effects logistic model (SAS, Proc Nlmixed) was used to evaluate across time (i.e., between 1998 and 2002) the relationship between the presence of OAK (a dichotomous variable, K-L ≥ 2 vs. K-L < 2) and continuous measures of BMI and body composition (fat mass, skeletal muscle mass, and waist circumference). Non-proportional odds models were used to evaluate the relationship *across time* between the (ordinal) K-L OA severity measure (scale) and measures of BMI and body composition (fat mass, skeletal muscle mass, and waist circumference). A random slope model was tested and found to be non-significant, so random intercept models were analyzed.

Analyses of covariance (SAS, Proc GLM) was used to evaluate the relationship between measures of joint space width for 2002 as well as changes from 1998 and measures of BMI and body composition (fat mass, skeletal muscle mass, and waist circumference) as well as age.

Covariates were retained in models if their inclusion changed the beta coefficients by 10% or more. The appropriateness of model fitting was assessed both graphically and using model r^2 (for ANCOVA) and Akaike Information Criterion (AIC) for mixed-effects models.

RESULTS

In 1998, the frequency of knee OA, defined as a K-L score > 2 was 11.6% among women who had x-ray data at this visit. In 2002, the frequency of knee OA was 11% in the population of women whose mean age was 47 years (Table 1). These frequencies differ slightly due to the fact that women could be lost to follow-up or recovered between the 1998 and 2002 visits. In the 4-year observation period, mean fat mass and mean skeletal muscle mass both increased by 2.4%. The mean BMI in 2002 was 29 kg/m^2 as compared to a mean BMI of 28.3 kg/m^2 in 1998. There was no increase in skeletal size as characterized with elbow breadth.

As shown in Table 2, fat mass was 41% greater in women with knee OA as compared to those without OAK. Lean mass and skeletal muscle mass were 11.5% and 10% greater, respectively, in women with OAK as compared to those without OAK. Notably, while women with OAK had higher absolute average muscle mass than women without OAK, the ratio of the skeletal

muscle mass to fat mass indicated that women with OAK had less skeletal muscle mass relative to the amount of fat mass. Mean BMI was 24% greater in women with K-L-defined OAK compared to women without knee OA. Waist circumference was 17% greater in women with OAK as compared to those without knee OA. Elbow breadth, a measure of skeletal size, was only 3% greater in women with knee OA.

There was no evidence that 4-year changes (from 1998 to 2002) in body composition measures were statistically significantly different in women with or without OAK (see Table 2).

There was moderate change with time in joint space width (JSW) of the medial aspect at both the 50% and 75% sites, shown in Table 3. The median 75% right medial JSW changed from 4.95 mm in 1998 to 4.28 mm in 2002, representing a −0.68 mm loss over the 4-year period. The median 75% left medial JSW changed from 5.11 mm in 1998 to 4.5 mm in 2002, representing a −0.57 mm median loss over the 4-year period.

In this early OAK, narrowing of the medial JSW was frequently offset in the lateral aspect. As a result, there was little JSW change in the lateral aspect over the 4-year period. The median 75% left lateral site JSW changed from 5.4 mm in 1998 to 5.36 mm in 2002, while the median 75% right lateral site JSW changed from 5.71 mm in 1998 to 5.6 mm in 2002. This represents a difference of −0.02 mm in the 75% left lateral JSW and −0.15 mm loss in the 75% right lateral sites (Table 3).

Associations of body composition with OAK

Mixed-effects logistic models that included the body composition measures fat mass and skeletal muscle mass had a better (smaller) AIC goodness of fit statistic than models based on BMI (seen in Table 4). Using multiple variable mixed-effects logistic analyses, it was determined that the odds of having OAK vs. no knee OA increased 8-fold for each 1 unit higher $_{\log}$fat mass and 477-fold for each 1 unit higher $_{\log}$skeletal muscle mass, following adjustment for skeletal muscle mass and fat mass, respectively, as well as age. Waist circumference and elbow breadth did not explain statistically significant variation in having knee OA and were excluded from reported models (Table 4).

After adjustment for age, increases in both $_{\log}$fat mass and $_{\log}$skeletal muscle mass were associated with greater odds of more severe K-L scores from 0 to 4 (Table 5) until the most severe levels were reached (likely because there were few women with the most severe level of OAK). Notably, the association of K-L scores was much stronger with skeletal muscle mass as compared to fat mass, as demonstrated by the larger beta coefficients (representing log odds) in Table 5. Further, the body composition model explained more variation than did the BMI model as indicated by the lower goodness of fit criterion. Although the BMI model did not fit as well as the body composition models, increasing severity was also associated with greater $_{\log}$BMI with the exception of the comparison to only the most severe group (K-L = 4).

Associations of body composition with joint space width (JSW)

Skeletal muscle mass was positively and significantly associated with level of both left and right medial JSW ($P < 0.01$ and $P < 0.0001$, respectively), that is, women with greater skeletal muscle mass had greater JSW, shown with the positive beta coefficient in Table 6. Greater age was negatively associated with JSW. There was no association of BMI with JSW in 2002 or with 4-year JSW change (Table 6).

Four-year changes in both BMI and fat mass were statistically significantly associated with 4-year JSW change, after adjusting for baseline age and baseline BMI or fat mass, respectively (Table 7). Four-year changes in skeletal muscle mass were not associated with 4-year changes

in JSW. However, participants in this population are not at an age when great loss of muscle mass would be expected, thus inhibiting our ability to observe changes in SMM.

DISCUSSION

The amount of skeletal muscle mass is a key body composition measure related to knee OA in mid-aged women and skeletal muscle mass explained more variation in the K&L scores and joint space than a measure of fat mass. This relationship would not have been elucidated with a measure of BMI which is described as a measure of obesity and is more highly correlated with fat mass than lean mass. In head-to-head comparisons, statistical models that included age and body composition (fat mass and skeletal muscle mass) to explain both the odds of having OAK as well as the severity of OAK had a better statistical fit, as assessed by a lower AIC, than models with age and BMI. This better fit occurred in measures of joint space width as well as the Kellgren-Lawrence osteophyte-based classification of OA. Women with OAK had less skeletal muscle mass per unit of fat mass than women without OAK.

Previous work of body composition and OA of the knee has centered largely on the role of obesity[1–7] as assessed by BMI, but BMI is a problematic measure. While it incorporates both lean and fat mass, measures of BMI are more highly correlated with fat mass than with lean mass. We identified that greater skeletal muscle mass was a more consistent predictor of OAK status (as assessed by K-L score or joint space width) than was fat mass.

Understanding whether OA is a function of fat or muscle mass (or both) has important clinical implications. These data would suggest that interventions that target weight loss as a treatment modality for osteoarthritis need to be cognizant of the impact of the weight loss intervention on muscle mass. Further, these findings suggest that approaches that optimize muscle contraction and muscle strength should be more seriously considered in treatment regimens and may be effective interventions.

The associations of body composition with joint space and K-L scores may not always be parallel. This is likely due to the fact that the K-L scores are based on osteophytes, and that the cut point of 2 is designated as osteoarthritis. However, joint space is unlikely to be contributing to K-L scores until values of 3 and 4 are assigned. In contrast, joint space is more frequently defined based on either comparison to a previous image (as in a longitudinal study) or less commonly, relative to joint space observed across a sample cross-sectionally. The observations in this cohort with respect to body composition and both K-L scores and joint space are particularly relevant because they characterize those features of early osteoarthritis initiation where the roles of muscle mass may be different than in older populations where muscle mass is widely known to be diminished.

This study helps to focus on the role and possible contribution of muscle mass in addition to fat mass. Clinical and animal studies of joint loading have provided evidence that abnormal loads can lead to changes in the composition, structure, and mechanical properties of articular cartilage[17–19]. These abnormal loads have been attributed to obesity, joint instability, or trauma. Biomechanically, muscle forces are a major determinant of how loads are distributed across a joint surface. Decreasing the muscle forces acting about a joint will ultimately alter loading conditions. Failure by the quadriceps to adequately absorb forces about the knee can cause greater dynamic loads being placed on the articular cartilage, resulting in progressive degeneration.

Recently, a population-based study of African-American and Caucasian women concluded that “precise” measures of body composition conveyed no advantage over the measurement of BMI in the assessment of risk for radiographically-defined osteoarthritis of the knee[20]. Unfortunately, the study evaluated lean mass (comprised of muscle and bone as well as

intracellular and extracellular fluid) rather than muscle mass, so there is no opportunity for doing a direct comparison. Potentially, there is also an age-related effect. The women reported, were on average, 15 years older than the population we report, and were likely to have experienced a decline in muscle mass with age which would be compensated for with an increase in adipose tissue. Notably, we found the role of skeletal muscle mass was highly contributory in explaining joint space width, and less informative about the osteophyte-based K-L classification. The Johnston County Osteoarthritis Project did not report the body composition measures in relation to joint space width[20]. A second study evaluated body composition and knee OA based on MRI-defined tibial cartilage volume, and reported that muscle mass was an independent predictor of cartilage volume[21]. However, the study population included 43 men and 43 women—and since muscle mass and prevalence of knee OA have different presentation and risk profiles in men and women, the findings are probably not comparable to our study.

It is worth identifying that women studied had somewhat greater fat mass and greater lean mass than values reported from the National Health and Nutrition Examination Survey (NHANES) III, values which were also estimated from bioelectrical impedance[22]. In the NHANES III data, the average fat mass for Non-Hispanic white women aged 40–49.9 years was 25.9 kg compared to our population where the average fat mass was 28.9 kg in 1998. Likewise, in the NHANES III data, the average lean mass for Non-Hispanic white women aged 40–49 years was 44.8 kg compared to our population where the average lean mass was 45.8 kg in 1998. There are no published data on the amount of skeletal muscle mass.

In summary, the use of body composition measures, rather than a summary obesity measure like BMI, provides information that is more consistent with the underlying pathophysiology of knee osteoarthritis. Remarkably, body composition measures were consistently associated with two different measures of knee OA, including the osteophyte based-K-L score and the amount of joint space width. By extension, these findings suggest that interventions that focus on weight loss need to be equally cognizant of the impact of the intervention on muscle mass. Further, these findings suggest that approaches that optimize muscle contraction and muscle strength may be effective interventions.

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Table 1

Age and body size characteristics of the Michigan Bone Health Study (MBHS) population coincident with x-rays for knee osteoarthritis

	1998 (n=485)		2002 (n=483)	
	Mean	**SD**	**Mean**	**SD**
Age (years)	43.1	4.85	46.9	4.85
Body composition measures				
Fat mass (kg)	28.9	12.4	29.6	13.8
$_{log}$(Fat mass)	3.3	0.39	3.3	0.43
Lean Mass	45.8	6.93	47.6	7.53
Skeletal muscle mass (kg)	20.5	2.73	21.0	2.98
$_{log}$(Skeletal muscle mass)	3.0	0.13	3.0	0.14
Waist circumference (cm)	85.9	14.4	89.3	15.4
$_{log}$(Waist circumference)	4.44	0.16	4.48	0.17
Elbow breadth (cm)	6.14	0.32	6.17	0.3
SMM-Fat mass ratio	0.81	0.29	0.83	0.33
Body mass index (kg/m^2)	28.3	6.23	29.0	6.85
$_{log}$(BMI)	3.32	0.21	3.34	0.23

Table 2

Body composition measures and their four year antecedent change in those with and without x-ray defined knee osteoarthritis (K-L score ≥ 2) in 2002

	No OAK in 2002		OAK in 2002		*P*-value
	n	$\bar{x}\pm SD$	n	$\bar{x}\pm SD$	
Age	429	46.5 ± 4.9	53	49.7 ± 3.5	<0.0001
Body composition measures					
Fat mass (kg)	429	28.3 ± 12.6	52	40.0 ± 18.4	<0.0001
Lean mass (kg)	429	47.0 ± 7.2	52	52.4 ± 8.4	<0.0001
Skeletal muscle mass (kg)	429	20.8 ± 2.9	52	22.8 ± 3.3	<0.0001
Waist circumference (cm)	429	87.7 ± 14.4	53	102.3 ± 16.7	<0.0001
Elbow breadth (cm)	429	6.1 ± 0.3	53	6.3 ± 0.3	<0.0001
SMM-Fat mass ratio	429	0.85 ± 0.3	52	0.68 ± 0.3	<0.0005
BMI (kg/m^2)	429	28.3 ± 6.3	53	35.0 ± 8.2	<0.0001
Body composition change (4-year)*					
Fat mass (kg)	382	0.37 ± 6.2	43	0.01 ± 8.2	0.73
Lean mass (kg)	382	1.61 ± 3.1	43	1.28 ± 3.6	0.52
Skeletal muscle mass (kg)	382	0.41 ± 1.2	43	0.34 ± 1.4	0.73
Waist circumference (cm)	382	2.99 ± 6.5	44	2.91 ± 8.6	0.95
Elbow breadth (cm)	382	0.02 ± 0.2	44	0.03 ± 0.2	0.87
BMI (kg/m^2)	382	0.56 ± 2.6	44	0.39 ± 3.8	0.77

* Change in body composition measures from 1998 to 2002.

Table 3

Medial and lateral joint space width (JSW, mm) of right and left knees at two sites (50% and 75%) medially or laterally from the joint center, in 1998 and 2002

	1998	2002	Difference	
	Median, IQR*	Median, IQR	Median, IQR	*P*-value
50% site				
Left lateral (mm)	5.72, 1.88	6.42, 1.51	0.63, 1.61	<0.0001
Right lateral (mm)	6.03, 2.02	6.47, 1.51	0.25, 1.56	<0.0001
Left medial (mm)	5.68, 1.36	5.51, 1.39	−0.13, 1.19	0.0004
Right medial (mm)	5.94, 1.54	5.51, 1.16	−0.42, 1.07	<0.0001
75% site				
Left lateral (mm)	5.4, 1.44	5.36, 1.16	−0.02, 1	0.62
Right lateral (mm)	5.71, 1.35	5.6, 1.23	−0.15, 1.01	0.0017
Left medial (mm)	5.11, 1.32	4.5, 1.04	−0.57, 1.09	<0.0001
Right medial (mm)	4.95, 1.22	4.28, 1.06	−0.68, 1.04	<0.0001

* Inter-quartile range

Table 4

Comparison of two logistic models of the odds of having knee osteoarthritis in 483 women according to body composition measures (Model 1) and BMI (Model 2) using the AIC goodness of fit criterion

	Model 1		Model 2	
	β coefficient (SE)	**Odds Ratio (95% CI)**	**β coefficient (SE)**	**Odds Ratio (95% CI)**
Age (years)*	**0.16 (0.043)**	**1.18 (1.1, 1.3)**	**0.15 (0.044)**	**1.16 (1.1, 1.3)**
Body composition measures				
$_{log}$(Fat mass) (kg)	**2.08 (0.64)**	**8.02 (2.3, 28.1)**		
$_{log}$(Skeletal muscle mass) (kg)	**6.17 (1.84)**	**476.99 (12.8, 17735.1)**		
$_{log}$(BMI) (kg/m^2)			**6.76 (1.28)**	**864.97 (70.5, 10617.9)**
AIC goodness of fit measure (smaller is better)	545.7		548.7	

*Age is grand-mean centered at 41.5 years.

†Statistically significant individual predictors at $P < 0.05$ are shown in bold.

Table 5

Comparing two logistic non-proportional odds models of the log odds of increasing OA severity defined by K-L score from 0 to 4, according to body composition (Model 1) or BMI (Model 2) using the AIC goodness of fit criterion

	Model 1 for body composition	Model 2 for BMI
	β-coefficient (*P*-value)	β-coefficient (*P*-value)
Age* (years) for		
K-L 0 vs. 1–4	0.23 (<.0001)	0.21 (<.0001)
K-L 0,1 vs. 2–4	0.19 (<.0001)	0.17 (<.0001)
K-L 0–2 vs. 3,4	0.26 (0.0002)	0.24 (0.0003)
K-L 0–3 vs. 4	0.33 (0.0115)	0.31 (0.0072)
Body composition		
$_{log}$(Fat mass, kg) for		
K-L 0 vs. 1–4	0.79 (0.0111)	
K-L 0,1 vs. 2–4	1.35 (<0.002)	
K-L 0–2 vs. 3,4	−0.30 (0.67)	
K-L 0—3 vs. 4	0.004 (0.997)	
$_{log}$(Skeletal muscle mass, kg) for		
K-L 0 vs. 1–4	5.98 (<.0001)	
K-L 0,1 vs. 2–4	5.58 (<.0001)	
K-L 0–2 vs. 3,4	5.85 (0.007)	
K-L 0–3 vs. 4	5.01 (0.17)	
$_{log}$(BMI, kg/m^2) for		
K-L 0 vs. 1–4		4.11 (<.0001)
K-L 0,1 vs. 2-4		4.86 (<.0001)
K-L 0–2 vs. 3,4		2.19 (<0.05)
K-L 0–3 vs. 4		1.96 (0.25)
AIC criterion (smaller is better)	1733.2	1754.3

Table 6

Comparing the association of BMI vs. body composition measures as predictors of medial joint space width at two locations (75% site and 50% site), measured in 2002, in left and right knees

Left knee, medial	**At 75%**			**At 50%**		
Model #1	**B**	***P*-value**	**Model r^2**	**β**	***P*-value**	**Model r^2**
Age	−0.02	0.01	r^2=6.9%	−0.02	0.02	r^2=4.5%
BMI	0.01	0.43		−0.00	0.97	
Skeletal muscle mass	0.07	<0.0001		0.06	0.003	
Waist circumference	−0.02	<0.02		−0.01	< 0.14	
Model #2	**β**	***P*-value**	**Model r^2**	**β**	***P*-value**	**Model r^2**
Age	−0.02	<0.01	r^2=6.9%	−0.02	0.0224	r^2=4.6%
Fat mass	0.01	0.37		0.01	0.49	
Skeletal muscle mass	0.08	<0.0001		0.06	<0.003	
Waist circumference	−0.02	<0.009		−0.02	<0.03	
Right knee, medial						
Model #3	**β**	***P*-value**	**Model r^2**	**β**	***P*-value**	**Model r^2**
Age	−0.03	0.002	r^2=10%	−0.04	0.0003	r^2=9.1%
BMI	−0.01	0.45		−0.03	0.14	
Skeletal muscle mass	0.1	<0.0001		0.09	<0.0001	
Waist circumference	−0.01	0.31		−0.00	<0.61	
Model #4	**β**	***P*-value**	**Model r^2**	**β**	***P*-value**	**Model r^2**
Age	−0.03	0.002	r^2=10%	−0.04	0.0004	r^2=8.7%
Fat mass	−0.00	0.81		0.00	0.59	
Skeletal muscle mass	0.1	<0.0001		0.09	<0.0001	
Waist circumference	−0.01	0.11		−0.02	<0.02	

Table 7

Comparing the association of BMI or body composition *changes* (after adjusting for baseline age and baseline BMI or body composition) as predictors of *changes* medial joint space width at two locations (75% site and 50% site), *between 1998 and* 2002, in left and right knees

Left knee, medial	**75% site**			**50% site**		
Model #1	**β**	***P*-value**	**Model r^2**	**β**	***P*-value**	**Model r^2**
Baseline age (1998)	−0.02	0.104	r^2=2.8%	−0.02	0.046	r^2=3.2%
Baseline BMI (1998)	−0.01	0.48		0.01	0.49	
4-year change in BMI	−0.05	0.005		−0.05	0.005	
Model #2	β	*P*-value	Model r^2	β	*P*-value	Model r^2
Baseline age (1998)	−0.01	0.12	r^2=3.1%	−0.02	0.046	r^2=4.2%
Baseline fat mass (1998)	0.001	0.76		0.003	0.35	
4-year change in fat mass	−0.02	0.002		−0.02	0.001	
Model #3	β	*P*-value	Model r^2	β	*P*-value	Model r^2
Baseline age (1998)	−0.01	0.16	r^2=0.6%	−0.02	0.09	r^2=1.2%
Baseline skeletal muscle mass (1998)	0.0003	0.985		0.01	0.75	
4-year change in skeletal muscle mass	0.02	0.53		0.05	0.24	
Right knee, medial						
Model #4	β	*P*-value	Model r^2	β	*P*-value	Model r^2
Baseline age (1998)	−0.01	0.32	r^2=1.5%	−0.01	0.16	r^2=1.1%
Baseline BMI (1998)	−0.003	0.62		0.01	0.17	
4-year change in BMI	−0.04	0.027		−0.01	0.45	
Model #5	β	*P*-value	Model r^2	β	*P*-value	Model r^2
Baseline age (1998)	−0.01	0.32	r^2=1.9%	−0.02	0.14	r^2=1.9%
Baseline fat mass (1998)	−0.001	0.72		0.01	0.047	
4-year change in fat mass	−0.02	0.011		−0.01	0.34	
Model #6	β	*P*-value	Model r^2	β	*P*-value	Model r^2
Baseline age (1998)	−0.01	0.42	r^2=0.6%	−0.01	0.36	r^2=1.8%
Baseline skeletal muscle mass (1998)	0.02	0.28		0.04	0.045	
4-year change in skeletal muscle mass	0.03	0.48		0.06	0.15	