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Weight, Rather Than Obesity Distribution, Explains Peak External Knee Adduction Moment During Level Gait

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Abstract

Objective—To determine whether a lower-body obesity pattern increases estimated forces on the medial compartment of the knee joint.

Design—Cross-sectional clinical biomechanical study.

Results—Nineteen normal weight (body mass index, $22.8 \pm 1.8 \text{ kg/m}^2$), 20 centrally obese (body mass index, $35.0 \pm 4.0 \text{ kg/m}^2$ and waist-hip ratio ≥ 0.85 for women; ≥ 0.95 for men), and 20 lower-body obese (body mass index, $36.4 \pm 5.4 \text{ kg/m}^2$) adults aged 37–55 yrs and without knee pain were recruited. There were no intergroup differences for age. Weight did not differ between obese groups, but thigh girth differed between groups ($P < 0.0001$). In univariate analysis, both obesity group and thigh girth were significantly related to peak external knee adduction moment in mid-stance phase. However, in multivariate analysis after adjusting for weight, no statistically significant differences persisted using either obesity distribution or thigh girth as predictors. Weight was a significant predictor of external knee adduction moment, explaining 33% ($P < 0.0001$) of variance in external knee adduction moment for level gait.

Conclusions—These data do not support a significant difference in knee medial compartment loading based on obesity distribution, but do support greater torque with higher weight. This suggests that the mechanism of obesity increasing risk for knee osteoarthritis may not be related to obesity distribution.

Keywords

Obesity; Knee Osteoarthritis; Adduction Moment; Gait Analysis

Thirty-four percent of adults in the United States are overweight (body mass index [BMI] of $25.0\text{--}29.9 \text{ kg/m}^2$) and an additional 32.2% are obese ($\text{BMI} \geq 30.0$).¹ Obesity-attributable sick leave and health insurance expenditures are substantial.^{2–4} Disability accounts for an important portion of the human and economic cost of obesity, and knee osteoarthritis (OA) is one of the principal causes of disability.⁵

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The National Health and Nutrition Examination Survey (NHANES)-I demonstrated an almost 4-fold increased risk of OA in American women with a BMI ≥ 30 kg/m² compared with <25 kg/m².⁶ This was further confirmed in the NHANES-III,⁷ as well as in Saudi women (odds ratio, 3.28).⁸ The effect of increased BMI has also been associated with a significantly increased risk of progressive cartilage loss,⁹ osteophyte progression,¹⁰ joint space narrowing on 1-yr follow-up,¹¹ and development of contralateral knee OA.¹² Evidence supporting a causative relationship was strengthened by numerous epidemiologic studies,^{13–23} and prospective studies,^{11,24,25} which have corroborated a proportional relationship between BMI and incident knee OA, and for middle-aged women with a BMI ≥ 30 kg/m², the odds of knee replacement are 6.4–10.5 times that of women with a BMI <25 kg/m².²⁶

The medial compartment of the knee is affected by OA 10 times as frequently as the lateral compartment,²⁷ possibly because it normally bears 2.5 times as much load as the lateral compartment.^{28–30} The external knee adduction moment (EKAM) is the primary determinant of load distribution on the medial compartments of the knee,³¹ and it is a predictor of knee OA severity³² and progression.³³ In addition, higher body weight has been associated with higher knee joint load and EKAM,³⁴ but this finding has not been consistent.³⁵

It has been hypothesized that obesity may contribute to knee OA through increased thigh circumference inducing genu varum.³⁶ An analysis of adolescent tibia vara revealed that dynamic knee varus in the absence of static varus malalignment in subjects with increased thigh fat results in pathologic compressive forces.³⁷ However, the effects of thigh circumference on knee joint moments have not been reported in adults. Davids et al. modeled the influence of simulated increased thigh girth on knee joint compressive forces. The compressive forces increased by 16% due solely to changes in gait dynamics associated with increased thigh volume, effectively increasing the moment arm of the weight force, which was attributed to increased knee varus.³⁷ The importance of the bias toward larger lever arms is underscored by Hunt et al.,³⁸ who showed that larger moment arms were the predominant feature associated with knee loading in patients with OA. In obese individuals, the effect of the increased moment arm on the EKAM would be expected to be compounded by the increase in the magnitude of the weight force (because of obesity), further adding to the compressive forces acting across the knee. The potential influence of mass distribution (thigh girth) on the EKAM is therefore substantial, but not documented, in obese adults.

Considering that women generally have larger thigh girth than men at equivalent BMI levels, and the relationship between obesity and knee OA is stronger in women,^{14,16,39} if greater thigh girth were associated with a higher EKAM, then thigh girth might explain a potential mechanism for the higher risk for knee OA in obese women. Therefore, this study was initiated to assess whether increased thigh girth is associated with more extreme medial compartment loading (EKAM) than that of either centrally obese or normal weight subjects.

METHODS

Subjects

Men and women, aged 37–55 yrs, were recruited through public advertisements. Volunteers were excluded if they had a history of a knee injury that required use of a brace or gait aid; neuromuscular disease; acute illness; wasting illness; intraarticular corticosteroid injection into either knee within the past 3 mos; knee surgery within the previous 6 mos; total knee arthroplasty; rheumatologic, metabolic, or hematologic abnormality with potential to affect lower-limb function (a comprehensive list was used for screening). Our institutional review board approved the protocol, including processes for recruitment and informed consent.

Cutoff values for thigh girth have not been reported, but waist-hip ratio (WHR) is an accepted method of characterizing mild-moderate obesity pattern into lower-body and central patterns. Therefore, subjects with a BMI of $>30 \text{ kg/m}^2$ and $\leq 37 \text{ kg/m}^2$ were stratified by WHR measurement into a predominantly central obesity group ($\text{WHR} \geq 0.85$ for women and $\text{WHR} \geq 0.95$ for men; $n = 20$) and a lower-body obesity group ($\text{WHR} < 0.85$ for women and $\text{WHR} < 0.95$ for men; $n = 20$). Control subjects with a BMI $< 24.9 \text{ kg/m}^2$, who were age-, sex-, and height-matched to lower-body obese subjects were studied for comparison ($n = 20$).

Subject Characterization

Demographic variables were assessed by questionnaire. Physical activity level was assessed using the Physical Activity Scale for the Elderly questionnaire.^{40–42}

Anthropometric Measurements

With subjects barefoot, height was measured to the nearest 1 mm with a stadiometer, and weight was measured to the nearest 0.1 kg with a balanced scale. Waist circumference at the level of the right iliac crest, hip circumference at the widest gluteal protuberance,⁴³ and thigh circumference at the level halfway between the inguinal ligament and upper border of the patella^{44,45} were measured to the nearest 1 mm with a Gulick II plus (Gulick II measuring tape; Country Technology Inc., Gays Mills, WI) tape measure. All measurements were confirmed by a second examiner and completed in duplicate. If there was a difference of $>1 \text{ cm}$ or 1 kg between measurements, four measurements were taken and averaged.

To objectively validate the classification of lower and central obesity, the center of mass in the vertical axis of each subject was estimated using the reaction board method. This involved having each subject lie supine on a board supported at one end by a weighing scale and at the other end by a supporting point that was aligned with the feet. Measuring the weight by this method, while knowing the full body weight from measurement on the balance scale, enabled the calculation of the vertical center of mass. The position of the center of mass as a percentage of the height from the feet was then calculated.

Motion Analysis

Joint motion and loading were estimated through collection of three-dimensional kinematics and kinetics data with a computerized motion analysis system (Optotrak Motion Analysis System [Northern Digital Inc., Waterloo, Ontario] and force plate data [Model 9286, Kistler, Amherst, NY]). Kinematic data were acquired at 60 Hz and filtered at 6 Hz (accuracy $<1 \text{ mm}$). Markers (three per segment) were affixed to the lateral aspect of the foot, crest of the tibia, over the lateral condyle of the femur using a femoral tracking device,⁴⁶ a sacral extension (markers mounted on a board fixed to the skin over the sacrum, tracking the pelvis), and upper thoracic extension (markers mounted on a board fixed to the skin over the sternum, tracking the trunk).⁴⁷ All markers were orientated laterally/anterior so as to be seen by the imaging system positioned on the right/front side. A digitizing wand was used to define localized skeletal landmarks that determined the link-segment model. Ground reaction data were collected during level walking at a sampling rate of 240 Hz and were time synchronized with the kinematic data.

Visual 3D software (C-Motion Inc., Germantown, MD) was used to perform kinematic and kinetic calculations. To address the purpose of this research, the first peak EKAM was determined during the first 50% of stance (loading response). Because this moment does not always have a second peak, the magnitude of the frontal plane moment during the last 50% of stance (propulsion) was measured at the point where the vertical ground reaction force peaked for a second time. Kinetic data were normalized to body mass.⁴⁸ To assess the

overall loading of the knee caused by the EKAM, the angular impulse (time integral of the frontal plane moment) was calculated for the entire stance phase. In addition, the amount of foot external rotation during stance was assessed based on the position of the long axis of the foot relative to the direction of progression.

Subjects walked along a level walkway at a self-selected cadence. The ensemble average of five trials during stance was used to represent the biomechanics of each subject. To confirm the absence of significant error as a result of collecting motion analysis data on obese individuals, the root mean square linear knee translations during walking were assessed.

Statistical Analysis

Sample size was determined through power analyses using published data for differences in varus moment between adults without knee osteoarthritis and adults with knee OA.⁴⁹ On the basis of summary statistics and the effect size observed in that study, 20 subjects in each of 3 groups, normal weight, predominance of abdominal obesity, and predominance of thigh obesity were found to be sufficient. This sample size was estimated for a three-sample balanced analysis of variance, providing >75% power detect a mean difference of 0.9 in peak EKAM (% body weight $\times$ height), assuming a standard deviation of 0.92 and $\alpha = 0.05$.

The primary predictor variable was body shape (lower-body obesity, central obesity, and normal weight) for categorical analyses and thigh girth for continuous analyses. The primary outcome measure was the first peak EKAM, an estimate of loading of the medial compartment of the knee. For univariate analyses, categorical variables were compared using the χ^2 test, and continuous variables were compared with one-way analysis of variance with Tukey's method for pairwise comparisons. Multivariable linear regression of the first and second peak EKAM on predictor variables, body shape and thigh girth, controlling for weight, height, sex, age, BMI, WHR, and interaction terms, was performed using the PROC GLM procedure in SAS Version 9.1 (SAS Institute Inc., Cary, NC). Each EKAM was analyzed before normalization as well as using the standard % mass normalization to body mass. An α level of 0.05 was used to determine significance for all tests.

RESULTS

Subject Characteristics

Group characteristics are presented in Table 1. There were no statistically significant differences between groups in age or sex. Group mean weight ($P < 0.0001$), BMI ($P < 0.0001$), and self-reported physical activity level (Physical Activity Scale for the Elderly) ($P < 0.0045$) differed between lower-body obese and normal weight subjects as well as between central obese and normal weight subjects in a statistically significant manner, but did not differ between obese groups (lower body and central). The lower-body obese group mean WHR differed from those for the central obese group ($P < 0.0001$), by definition and thigh girth differed between all groups ($P < 0.0001$).

The center of mass as a function of percent height, measured by the reaction board, significantly differed between central (55.3%) and lower-body (53.9%) obesity groups ($P < 0.0002$), but neither significantly differed from normal weight subjects (54.6%), confirming the skew in weight distribution between the obese groups.

An effort was made to judge if marker tracking during motion analysis was different for the obese and control groups, thereby influencing our primary measure (EKAM). Measures of knee linear translations, which require independent estimates of the knee joint center based on the thigh and shank models, were considered to be sensitive to errors that might occur as a result of skin motion artifact (assuming all groups had similar knee joint translations).

Group averages for these translations are represented in Table 2. Relative to the control group, the potential additional error in estimates of the knee joint center, in the medial-lateral (z) direction, for the central and lower-body obese groups was 3.0 and 1.5 mm, respectively. These deviations would cause errors within the central and lower-body obese groups of 6.6% (2.86 ± 0.03 N m) and 4.5% (1.36 ± 0.01 N m), respectively. Given the magnitude of difference between groups, we did not view these errors to be significant.

External Knee Adduction Moments

In univariate analysis, both the first and second peak EKAM in mid-stance phase significantly differed between each of the obese groups and the normal weight group during level gait ($P < 0.0001$; $R^2 = 0.29$) (Fig. 1). Simple linear regression also revealed that thigh girth was a significant predictor of first peak EKAM during level gait ($P = 0.0007$; $R^2 = 0.18$). However, both of these differences disappeared after normalizing EKAM to body mass (Table 3).

Foot external rotation was not significantly associated with body shape group, but was associated with thigh girth ($P = 0.0036$). Neither body shape group nor thigh girth was independent predictors of peak EKAM after controlling for weight. Weight was the strongest predictor of EKAM, explaining 33% ($P < 0.0001$) of variance in the first peak EKAM during level gait.

Outlier Analysis

Bivariate graphical analyses with EKAM as the dependent variable demonstrated four outlier values within the central obesity group. In regression analyses, these four subjects from the central obesity group had studentized and jackknife residuals near -2 with high influence. Therefore, analyses were repeated with these four outliers removed to reduce the risk of making a type I error. Analysis of these four outliers revealed that although weight, height, BMI, WHR, and thigh girth did not differ between these subjects and the other obese subjects, their EKAM and impulse were significantly more extreme. The mean $\pm$ SD EKAM for nonoutliers and outliers were -34.8 ± 12.3 and -79.5 ± 7.2 N m, respectively, and the EKAM normalized to body mass were -0.4 ± 0.1 and -0.7 ± 0.1 N m/kg, respectively.

Removal of these four subjects resulted in a less extreme mean EKAM in the central obesity group. Specifically, after removal of the four outlier subjects, significant differences existed in the first peak EKAM only between each obese group (central: -36.9 ± 13.4 N m; lower: -33.0 ± 11.3 N m) and the normal weight group (-21.6 ± 8.9 N m) ($P = 0.0004$). Similar to the results for the whole sample, there were no significant intergroup differences in the first peak EKAM after normalizing to body mass. However, in contrast to results for the whole sample, there were no differences in the second peak EKAM or in the gait impulse either before or after normalizing to body mass.

DISCUSSION

The lack of a statistically significant association between either body shape or thigh girth and EKAM, after controlling for the effect of weight suggests that excess weight is the main factor responsible for the more extreme medial compartment loading during obese gait. Thus, rather than thigh or abdominal fat distribution, it seems to be the presence of obesity that increases medial compartment loading. The increased medial loading with obesity found in this study may help to explain why obese subjects have been noted to have enlarged medial tibial bone area.⁵⁰

The lack of a difference between lower-body and central obesity on estimates of medial knee joint loading is consistent with the findings of epidemiologic studies of obesity and

knee OA.^{19,20,51,52} After adjusting for age, race, and BMI, the NHANES-I did not demonstrate an association between body fat distribution (central or peripheral fat distribution) and knee OA.⁵¹ Data from the Baltimore Longitudinal Study of Aging, also after adjusting for BMI, did not reveal a significant association between percent body fat (measured by skin folds) or WHR and knee OA.²⁰ The NHANES-III also demonstrated that continuous waist circumference did not contribute to predicting risk for knee OA once BMI was in the model.⁵³ Our study was designed to address a limitation of these studies, the lack of assessment of thigh circumference in obese adults. The combined results of these studies, therefore, suggest that neither increased central nor thigh fat distribution seems to be the mechanism by which obesity increases risk for knee OA.

It is interesting that when treated as categorical variables, the higher tertiles of waist circumference within each tertile of BMI increased risk for knee OA in NHANES-III although not when BMI and waist circumference were treated as continuous variables.⁵³ Our finding that centrally obese subjects had a more extreme EKAM than lower-body obese subjects, although not significantly so, may be consistent with this increased risk for knee OA in subjects categorized as the highest tertile of central obesity in NHANES-III. However, the consistency among the results of NHANES-I, Baltimore Longitudinal Study of Aging, NHANES-III continuous analyses, and the results of the current study suggest that obesity is a much stronger risk factor for knee OA than the pattern of obesity.

Although not independent of weight, the presence of a more extreme absolute first peak EKAM and impulse in those with a central obesity pattern is indicative of the moment that is actually experienced by the knee joint. Considering that an increased first peak EKAM has been associated with risk for knee OA and angular impulse is related to severity of knee OA,^{32,54,55} this increased absolute load may have consequences. In addition, considering that 4 of the 20 subjects with a central obesity pattern had first peak EKAM values considerably more extreme than others (outliers) and not all obese people develop knee OA, it may be this subset that is at increased risk for knee OA. This supposition is supported by the fact that normalizing the first peak EKAM to body mass for the whole central group did not bring first peak EKAM values to comparable levels with the control group (as was seen when the outliers were removed).

There was a proportional decrease in second peak EKAM for both obese groups that is reflected in significantly lower, normalized values for the lower-body obesity group. This relative decrease in the second peak points to a somewhat modified walking strategy seen in the obese groups (and evident in Fig. 1). Although all three groups showed a relative decrease in the second peak EKAM, the lower-body obesity group demonstrated the greatest reduction in magnitude, such that the bimodal characteristics were minimized and the amplitude was not different from the control group. Previous research has associated changes in the second peak EKAM with the amount of toe-out, gait speed, or sex.^{55,56} Such associations were not demonstrated in the current study. Given that a symmetrical bimodal ground reaction force was seen in all groups, the changes observed for the lower-body obesity group point toward dynamic alignment changes that may be positively influenced by body mass distribution. These changes are also represented in the impulse data where trends point to greater loading in the central obesity group.

Although our results regarding estimated knee joint loading in subjects without OA were consistent with results reported in epidemiologic studies including subjects with knee OA, there were several potential limitations. First, the EKAM is an estimate of knee joint forces rather than a direct measure. However, this estimate is highly correlated with risk for progressive knee OA and therefore merited assessment.^{32,33} Second, there was a relative lack of male subjects in this study despite efforts to recruit both men and women. Although

this limited ability to compare results by sex, the availability of female subjects was informative, considering both the prevalence of knee OA and the association between obesity and knee OA is higher in women. Importantly, results for the overall study and for women only did not differ. When restricted to the 50 female subjects, nonnormalized EKAM significantly differed between control (-21.2 N m) and lower (-33.6 N m) by 12.4 N m (95% confidence interval, 1.3 – 23.5 ; $P < 0.0001$), and between control and central (-44.0 N m) by 22.8 N m (95% confidence interval, 10.8 – 34.9 ; $P < 0.0001$). Even after controlling for weight, body shape was a significant predictor of EKAM ($P = 0.0001$). In addition, toe rotation did not differ between central and lower-body obese groups in women.

This study found that estimates of medial knee joint loading are more dependent on weight than on fat distribution or thigh girth in obese subjects. This study did not assess the effects on knee joint loading in people with high BMI or high thigh girth because of excess muscle. Although this could be studied separately in subjects with excess muscle, we are not aware of an increased risk for knee degeneration in such individuals, and excess fat tissue is by far, the most common reason for a high BMI.

This study was designed to assess the effect of fat distribution on medial knee joint loading in an effort to address a portion of the larger question, “why does obesity increase risk for knee OA?” Although the mechanism is unclear, it does seem that excess weight plays an important role. With obesity, that excess weight is mostly fat, a noncontractile tissue that may load the knee without providing joint protection. In other words, whereas lean mass may aid in controlling knee joint movement or providing active stabilization of the joint, excess fat would not provide such control. These circumstances may lead to exceeding a threshold of EKAM that either damages cartilage or prevents it from healing and therefore contributes to medial knee OA.

There are likely numerous factors, outside the focus of this study, that may better characterize why obesity predisposes to knee OA and which adults are at increased risk. Longitudinal epidemiologic studies have suggested that factors in the local joint environment, such as strength, laxity, malalignment,^{57,58} and history of an injury⁵⁹ may interact with obesity to contribute to risk for knee OA.^{57,58,60–62}

Because this study was intended to assess for differences in the dynamic frontal plane moment at the knee in people without knee OA, who had lower-body and central obesity, other risk factors associated with knee OA were not assessed. For example, radiographic lower-limb alignment was not measured because the focus of this study was on the EKAM. Although lower-limb mechanical axis is recognized as a predictor of knee peak adduction moments,⁶³ the correlation between radiographic lower-limb alignment and the EKAM has not been consistently reported to be high. Although lower-limb mechanical axis is recognized as a predictor of knee peak adduction moments,⁶³ it explains $<50\%$ of the variability in the dynamic frontal plane moment.⁶⁴ Harrington⁶⁵ reported that the inclination of the tibia in the coronal plane rather than angulation at the knee predicts the adduction moment because of the ground reaction force. From this, he concluded that there is no correlation between knee angular deformities on radiographs and joint forces. Others have also reported that static alignment does not predict the location or magnitude of knee joint forces.^{29,30}

In contrast, studies of patients after unicompartmental arthroplasty have revealed a relationship between knee coronal radiographic alignment and the knee adduction moment.^{66,67} Although reasons for the differences in correlations have not been fully elucidated, preliminary evidence suggests that the relationship may be stronger in subjects after mechanical realignment of the knee.⁶⁸ The discrepancy between static alignment and

dynamic moments also may be as a result of antalgic or proprioceptive compensations, such as increased toe-out, widened base, or alteration of gait velocity.^{65,68} Therefore, for studies of the development of knee OA or surgical realignment, radiographic mechanical alignment may be of value.

CONCLUSIONS

Obesity is a significant predictor of medial knee joint loading. Thigh girth or lower-body obesity do not seem to explain this effect. Increased weight, rather than distribution of fat, better explains the increased medial knee joint loading with obesity.

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FIGURE 1.

External knee adduction moment (EKAM) by group: plot of the mean frontal plane moments at the knee during stance phase demonstrates more extreme moments in the obese groups than in the control group.

TABLE 1Characteristics of subjects (mean $\pm$ SD)

	Lower body obese	Central obese	Normal weight
Sex, F:M	19:1	14:6	17:2
Age, yrs	49.3 $\pm$ 5.7	49.1 $\pm$ 4.6	48.7 $\pm$ 3.8
Weight, kg	99.7 $\pm$ 14.9	102.2 $\pm$ 11.9	60.6 $\pm$ 7.5
BMI, kg/m ²	36.4 $\pm$ 5.4	35.1 $\pm$ 4.0	22.8 $\pm$ 1.8
Thigh girth, cm	65.9 $\pm$ 5.5	60.1 $\pm$ 3.4	48.0 $\pm$ 2.8
Waist-hip ratio	0.81 $\pm$ 0.05	0.93 $\pm$ 0.06	0.85 $\pm$ 0.07
PASE score	150.0 $\pm$ 11.1	148.2 $\pm$ 16.9	159.2 $\pm$ 11.1

TABLE 2

Mean RMS translations (m) for the stance phase of walking

	X	Y	Z
Control	0.008216	0.004506	0.002221
Central	0.012947	0.007405	0.005231
Lower	0.010366	0.006203	0.003717

TABLE 3Mean peak EKAM (N m, mean $\pm$ SD)

	Central obese	Lower obese	Normal weight	<i>P</i>
Gait speed	1.20 $\pm$ 0.13	1.06 $\pm$ 0.15	1.28 $\pm$ 0.18	0.0705
First EKAM	-43.28 $\pm$ 20.03	-33.01 $\pm$ 11.31	-21.61 $\pm$ 8.88	<.0001
First EKAM normalized to mass	-0.42 $\pm$ 0.18	-0.34 $\pm$ 0.12	-0.36 $\pm$ 0.13	0.1771
Second EKAM	29.72	15.77	15.75	0.0117
Second EKAM normalized to mass	-0.29	-0.16	-0.26	0.0513
Impulse	-17.11	-10.25	-7.32	0.0022
Impulse normalized to mass	0.16	0.11	0.12	0.1030