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ORIGINAL ARTICLE

Knee Power Is an Important Parameter in Understanding Medial Knee Joint Load in Knee Osteoarthritis

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Objective. **To determine the extent to which knee extensor strength and power explain variance in knee adduction moment (KAM) peak and impulse in clinical knee osteoarthritis (OA).**

Methods. **Fifty-three adults (mean ± SD age 61.6 ± 6.3 years, 11 men) with clinical knee OA participated. The KAM waveform was calculated from motion and force data and ensemble averaged from 5 walking trials. The KAM peak was normalized to body mass (Nm/kg). The mean KAM impulse reflected the mean total medial knee load during stride (Nm × seconds). For strength, the maximum knee extensor moment attained from maximal voluntary isometric contractions (MVIC) was normalized to body mass (Nm/kg). For power, the maximum knee extensor power during isotonic contractions, with the resistance set at 25% of MVIC, was normalized to body mass (W/kg). Covariates included age, sex, knee pain on the Knee Injury and Osteoarthritis Outcome Score, gait speed, and body mass index (BMI). Relationships of the KAM peak and impulse with strength and power were examined using sequential stepwise forward linear regressions.**

Results. **Covariates did not explain variance in the KAM peak. While extensor strength did not, peak knee extensor power explained 8% of the variance in the KAM peak ($P = 0.02$). Sex and BMI explained 24% of the variance in the KAM impulse ($P < 0.05$). Sex, BMI, and knee extensor power explained 31% of the variance in the KAM impulse ($P = 0.02$), with power contributing 7% ($P < 0.05$).**

Conclusion. **Knee extensor power was more important than isometric knee strength in understanding medial knee loads during gait.**

INTRODUCTION

Given that knee musculature has the capacity to modify the loading environment at the knee (1), there has been much interest in a potential role of muscle in the development and progression of knee osteoarthritis (OA). Deficits in knee extensor strength, i.e., reductions in the net maximum extensor moment during voluntary contractions, are related to pain and mobility impairments in people with knee OA (2). However, changes in knee extensor muscle strength are not necessarily related to progression of knee OA. In a prospective, 30-month study

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Significance & Innovations

- Much research has investigated the role of muscle strength, or the ability of a muscle to produce a moment, in the incidence and progression of knee osteoarthritis (OA). Knee extensor power reflects the ability to generate an extensor muscle moment quickly and has the potential to add to our understanding of the role of muscle function in knee OA.
- Knee extensor power significantly explained the variance in the knee adduction moment (KAM) peak and impulse during gait, whereas knee extensor strength did not.
- Knee extensor power may have better potential than strength in explaining the KAM peak and impulse during gait because power reflects the speed with which joint moments can be developed to move or support the knee.

with 265 subjects with symptomatic knee OA, no associations were found between quadriceps strength and cartilage loss at the tibiofemoral joint, regardless of limb alignment (3). Similarly, in elderly women with established knee OA (n = 79), stronger quadriceps strength did not protect against radiographic progression over 2.5 years (4).

The role of knee extensor strength on the incidence of radiographic knee OA is controversial. Slemenda et al showed that women who developed incident knee OA (n = 13) had 15–18% lower knee extensor strength at baseline when compared to controls (n = 107) after adjusting for body mass ($P = 0.053$) and lean muscle mass ($P = 0.085$) (5). However, in the Multicenter Osteoarthritis Study, which included 2,519 knees with no radiographic signs of OA at baseline, knee extensor strength did not predict incident radiographic evidence of knee OA 30 months later (6). Palmieri-Smith et al compared isometric quadriceps strength in 348 women with mild, moderate, and severe radiographic knee OA and found no difference in strength between the moderate and severe knee OA groups (7). Also, quadriceps strength was not different between women with (n = 22) and without (n = 13) mild knee OA, suggesting that knee extensor strength may not be affected in women with mild disease (8).

While primarily responsible for sagittal plane motion, the knee extensors may play an important role in the knee adduction moment (KAM). The quadriceps are active during activities that produce a frontal plane moment (9). Using a biomechanical model, Shelburne et al demonstrated that the quadriceps were capable of developing forces that counteract the external KAM during gait, despite a small moment arm to do so (10). It is likely that quadriceps and other muscles primarily controlling sagittal plane movement can modify frontal plane loads by enhancing joint stability through co-activation, which increases the contact forces between the tibia and femur during activity (11). However, cross-sectional and longitudinal studies suggest that a measure of strength may not represent this ability for the knee to modify frontal plane knee loads. Correlations between knee extensor strength and medial knee joint loads during gait were low, at best, in early or established knee OA (12,13). The KAM peak is a surrogate for medial loading which is often, but not always, elevated in people with knee OA compared to healthy counterparts (14). Lim et al found a trend toward a weak relationship between quadriceps strength and KAM peak in 184 people with knee OA ($r = 0.14$, $P = 0.059$) (13). Similarly, a bivariate relationship between quadriceps strength and loading rate in 204 participants with knee OA ($r = 0.27$, $P = 0.008$) was explained by walking speed (15). After 6 months of high intensity resistance training in 26 women with knee OA, the KAM peak did not change (16). These studies could point to the possibility that knee extensor strength may not be the best variable to represent muscle function in knee OA.

Knee power may better reflect the capacity of muscle to manage knee loads than strength. Knee power accounts for the rate of moment development, and, in the case of knee extensor power, it is the product of the extensor moment of force and the velocity of movement (17). Therefore, compared to strength, power could demonstrate better relationships with the KAM because it reflects speed of movement, and faster accelerations of the body's center of mass during gait may result in higher ground reaction forces, therefore larger knee joint moments (18). Furthermore, power is a more robust predictor of functional outcomes than strength in the aging literature (19) and shows promise in understanding knee OA pathology (20). To date, the role of knee extensor power in understanding medial knee loads has not been explored. Therefore, the purpose of this study was to determine the extent to which knee extensor strength and power explain variance in KAM peak and impulse among participants with clinical knee OA. We hypothesized that knee extensor power would explain a larger proportion of the variance in the KAM peak and KAM impulse than knee extensor strength in participants with knee OA.

PATIENTS AND METHODS

This cross-sectional study of associations was conducted as a secondary analysis from an ongoing longitudinal study.

Study sample. Community dwelling adults between ages 40–70 years who met the American College of Rheumatology (ACR) clinical criteria for knee OA were recruited from a local rheumatologist's and orthopedic surgeon's office. The clinical guidelines set forth by the ACR include having knee pain on most days of the month and at least 3 of the following 6 criteria: age $\geq$50 years, stiffness lasting $<$30 minutes, crepitus, bony tenderness, bony enlargement, and no palpable warmth (21). Exclusion criteria included having a diagnosis of other forms of arthritis (e.g., rheumatoid arthritis), active nonarthritic disease (e.g., gout), conditions that might be exacerbated by the protocol (e.g., unstable angina), current/past use of intraarticular therapies (e.g., cortisone injections), or previous knee surgeries (e.g., high tibial osteotomies, joint replacements). In

addition, potential participants were excluded if they required an adaptive walking aid such as a cane or a walker on a regular basis, sustained lower extremity trauma within the past 3 months, had ipsilateral hip or ankle conditions, or had contraindications to magnetic resonance imaging, an element of a larger study. If a patient had bilateral knee OA, the extremity that the patient reported as having more severe symptoms was used as the study leg. The study was approved by the Human Research Ethics Board at McMaster University and all participants provided written informed consent.

Dependent variables. The KAM waveform was calculated from barefoot gait trials. Kinematic data were collected using 3 Optotrak Certus camera banks (totaling 9 cameras; Northern Digital) sampling at 100 Hz. Kinetic data were collected using a synchronized floor-mounted force plate (Advanced Mechanical Technology) sampling at 1,000 Hz. Rigid clusters of infrared-emitting diode markers were secured to the sacrum, thigh, shank, and foot of the study leg (3 markers per cluster). The thigh and shank clusters were positioned on the lateral aspect of the study extremity adjacent to the estimated center of mass of these segments. The foot cluster was affixed to the lateral aspect of the foot. A digitizing probe was used to define localized skeletal landmarks. These landmarks were used to create the link-segment model during a static reference trial.

Participants ambulated barefoot at self-selected speeds until 5 trials were captured in which the participant struck the force plate cleanly with the study leg. Using commercial software (Visual 3D, C-Motion), the marker data were lowpass filtered with a fourth-order Butterworth filter with a 6-Hz cutoff frequency (22), and the KAM waveform was generated using inverse dynamics with a 3-dimensional floating axis coordinate system (23). The mean peak KAM from 5 trials represented the maximum medial knee load experienced during level walking. The peak KAM has been shown to be a reliable and valid measure (24,25) that is sensitive to radiographic progression (26). The peak KAM is presented normalized to body mass (Nm/kg) to allow comparisons between people of different body sizes. The KAM impulse for each of the 5 gait trials was calculated using the trapezoidal rule to integrate the stance phase portion of the KAM waveform (Matlab 7.0.1, Mathworks). The mean KAM impulse over the 5 trials was used as the representation of total knee loading magnitude because it incorporates the magnitude and duration of the entire stance phase. The KAM impulse has been shown to be a reliable and stable measure (27), associated with increased pain in knee OA (28), and sensitive to radiographic disease progression (29). The KAM impulse values in non-normalized units of Nm $\times$ seconds were used in the analysis, as normalizing the KAM impulse may divert attention from the absolute mechanical loading at the knee (30).

Independent variables. An isokinetic dynamometer (Biodex System 3) was used to measure knee extensor strength and power of the test extremity. Participants were seated upright with the hips in 80° of flexion. The center of rotation of the knee was aligned with the axis of rotation of the dynamometer. The lever was secured using a Velcro strap around the lower leg of the test extremity, approximately 3 cm proximal to the medial malleolus. To avoid unwanted movement, straps were secured over the thigh of the test extremity, across the waist, and over both shoulders. To measure knee strength, 5 maximal effort isometric knee extensor contractions (MVIC) were performed with the knee positioned at 60° of flexion. Participants were verbally encouraged to give a maximal effort and hold the contraction for 5 seconds with a 5-second rest period between each contraction. Knee extensor strength was the maximum moment measured during 1 of the 5 measurement trials. As recommended by Bennell et al, strength was normalized to body mass (Nm/kg) to account for differences in body mass (31). Isometric knee extensor efforts have demonstrated excellent reliability (32).

Dynamic contractions where the speed of movement can vary are necessary to measure knee extensor power. Ten isotonic contractions with the resistance set at 25% of MVIC were performed as quickly as possible. Participants started the trial with the knee at 90° flexion and were verbally encouraged to use their full extension and flexion range at maximum velocity. Data were recorded at 10 Hz. Moment values were converted from foot-pounds to Nm, and velocity values were converted from degree/second to radian/second (1 degree/second $\times$ 0.0174532925). Power, measured in watts (W), was calculated as the product of moment in Nm and velocity in radian/second. The maximum value from any of the 10 isotonic trials was normalized to body mass and used to represent knee extensor power (W/kg). This maximum power value may have occurred at any point in range of motion, with all occurring near midrange.

Covariates. We examined whether the following variables were potential covariates in the analyses because these have been previously identified as being related to KAM: age (33), sex (34), knee pain (35), gait speed (36), and obesity (37). Knee pain was measured using the pain subscale of the Knee Injury and Osteoarthritis Outcome Score (KOOS-pain) (38). The KOOS was developed to assess self-reported knee pain, physical function, quality of life, and sport and recreation. For the purpose of this study, only the pain subscale was used. Participants were asked to consider pain over the last week and scores from this 5-point Likert subscale were then normalized, with 100 indicating no symptoms and 0 indicating extreme symptoms. Gait speed was measured during gait trials between first heel strike and the second heel strike (meters/second). Obesity was characterized by body mass index (BMI), which was calculated from measured height and body mass (kg/m^2) while participants were barefoot, wearing a t-shirt and shorts.

Statistical analysis. Statistical analyses were performed using SPSS software. Descriptive statistics (means and SDs) were calculated for the demographic, dependent, independent, and covariate variables. Pearson's correlation coefficients examined bivariate relationships between the

Table 1. Descriptors of the knee OA participants (n = 53)*

	Mean ± SD	Minimum, maximum
Age, years	61.6 ± 6.3	41, 70
Body mass, kg	75.0 ± 16.2	51.0, 117.0
Height, meters	1.625 ± 0.119	1.070, 1.941
BMI, kg/m^2	27.9 ± 5.6	19.7, 41.8
KOOS pain, scale 0–100	74.7 ± 17.3	42.0, 100.0
Gait speed, meters/second	1.16 ± 0.22	0.73, 1.61
Knee extensor strength, Nm	110.4 ± 47.5	26.9, 251.8
Knee extensor strength normalized, Nm/kg	1.5 ± 0.6	0.3, 3.5
Knee extensor power, W	279.8 ± 163.7	43.4, 991.4
Knee extensor power normalized, W/kg	3.77 ± 1.89	0.55, 10.04
KAM peak, Nm/kg	0.37 ± 0.17	0.04, 0.77
KAM impulse, Nm × seconds	9.8 ± 6.7	0.9, 26.9

* OA = osteoarthritis; BMI = body mass index; KOOS = Knee Injury and Osteoarthritis Outcome Score; W = watts; KAM = knee adduction moment.

KAM impulse and KAM peak with the independent variables and each of the potential covariates. For the correlations, Bonferroni adjustments were made for multiple comparisons, resulting in an alpha set at 0.002. Two stepwise, forward linear regression analyses were performed for each of the 2 dependent variables: KAM peak and KAM impulse. These analyses aimed to evaluate the extent to which variance in the dependent variable could be explained by 1) knee extensor strength and 2) knee extensor power. In both regressions, all potential covariates were entered before the independent variable, either knee extensor strength or knee extensor power. In and out *P* values were set at 0.05 and 0.10, respectively.

RESULTS

Fifty-three participants (42 women, 11 men) completed the study (Table 1). Correlations between the covariates and the dependent measures (KAM peak and impulse) were significant at the corrected alpha level of 0.002 (Table 2). Figure 1 shows the relationships of KAM impulse and KAM peak with each of knee extensor strength and knee extensor power. While KAM impulse and KAM peak were related to knee extensor power, they were not related to knee extensor strength.

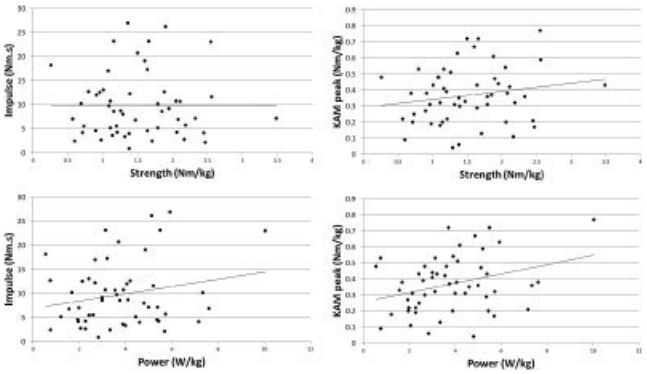

Figure 1. Relationships between dependent variables (knee adduction moment [KAM] peak and KAM impulse) with knee extensor strength and power. W = watts.

Table 3 shows the stepwise linear regression analyses for KAM peak and mean KAM impulse as dependent variables. When the KAM peak was the dependent variable, model 1 showed that no variables met the entry criteria. However, model 2 showed that peak knee extensor power explained 8.2% of variance in the KAM peak ($P = 0.022$). When the mean KAM impulse was the dependent variable, 24.2% of the variance in KAM impulse was explained by the covariates sex and BMI ($P < 0.001$); knee extensor strength did not add significantly to model 1 ($P > 0.05$). On the other hand, 31.0% of the variance in KAM impulse was explained by model 2 ($P < 0.001$), with knee extensor power contributing 6.8% ($P = 0.018$).

DISCUSSION

Knee extensor power shared a positive relationship with the medial compartment knee load during level walking in this sample of participants with clinical knee OA. Therefore, those capable of producing large knee powers also

Table 2. Correlation coefficients between covariates, independent variables, and dependent variables*

	Age	Sex	Pain	Gait speed	BMI	Knee strength	Knee power	KAM impulse
Age, years	1							
Sex†	0.20	1						
Pain	−0.14	−0.05	1					
Gait speed, meters/second	−0.11	−0.05	0.34	1				
BMI, kg/m^2	−0.05	0.09	−0.40	−0.08	1			
Knee strength, Nm/kg	−0.27	−0.23	0.38	0.15	−0.40	1		
Knee power, W/kg	−0.19	−0.35	0.35	0.11	−0.35	0.70‡	1	
KAM impulse, Nm × seconds	0.02	−0.29	−0.10	−0.02	0.40	−0.01	0.21	1
Peak KAM, Nm/kg	0.08	−0.08	0.06	0.13	0.05	0.18	0.32	0.81‡

* BMI = body mass index; KAM = knee adduction moment; W = watts.
† Men = 0, women = 1.
‡ Significance based on Bonferroni-corrected *P* value ($P = 0.002$).

Table 3. Sequential forward linear regression models of the knee adduction moment peak and impulse*

Independent variables	Cumulative adjusted R^2	Change in adjusted R^2	Standardized β coefficient	Unstandardized β coefficient (95% CI)	*P*
Dependent variable: KAM peak, Nm/kg					
Model 1					
No variables met entry criteria					
Model 2					
1. Power	0.082		0.315	0.029 (0.004, 0.053)	0.022
Excluded variables					
Age					0.291
BMI					0.203
Sex					0.811
Gait speed					0.652
KOOS pain score					0.689
Dependent variable: KAM impulse, Nm × seconds					
Model 1					
1. BMI	0.145		0.402	0.477 (0.171, 0.783)	0.033
2. BMI + sex	0.242	0.174			< 0.001
BMI			0.431	0.512 (0.223, 0.801)	0.001
Sex			−0.332	−5.411 (−9.380, −1.442)	0.009
Excluded variables					
Age					0.359
Gait speed					0.935
KOOS pain score					0.615
Strength					0.403
Model 2					
1. BMI	0.145		0.402	0.477 (0.171, 0.783)	0.003
2. BMI + sex	0.242	0.097			< 0.001
BMI			0.431	0.512 (0.223, 0.801)	0.001
Sex			−0.332	−5.411 (−9.380, −1.442)	0.009
3. Sex + BMI + power	0.310	0.068			< 0.001
BMI			0.533	0.633 (0.340, 0.927)	< 0.001
Sex			−0.229	−3.739 (−7.769, 0.290)	0.068
Power			0.319	1.127 (0.200, 2.055)	0.018
Excluded variables					
Age					0.164
Gait speed					0.382
KOOS pain score					0.928

* Model 1 incorporates knee extensor strength as a potential predictor. Model 2 incorporates knee extensor power as a potential predictor. 95% CI = 95% confidence interval; KAM = knee adduction moment; BMI = body mass index; KOOS = Knee Injury and Osteoarthritis Outcome Score.

demonstrated large KAM peaks and impulses during a stride. Knee extensor strength was not related to either the KAM peak or impulse. Therefore, the rate of moment development by the knee extensors may be more important than the extensor moment itself to the KAM peak and impulse in participants with knee OA. These data suggest that the ability to generate moments quickly may be important to consider in studying the initiation and progression of knee OA.

Power may have outperformed strength in the current study because the speed with which joint moments can be developed to move the knee, or respond to joint loads that result from weight bearing, may be important to the mechanical pathology of knee OA. While no studies were identified that related power with the KAM, some work examined the impact of muscle function on loading rates. Compared to 19 women who engaged in lower extremity strength training at least 3 times a week for a year, 18 sedentary women demonstrated greater maximum slopes of vertical ground reaction force after heel strike (39). Eighteen adults with knee pain demonstrated greater speeds of the foot and ankle at heel strike and reduced duration of quadriceps activation measured using electromyography (EMG), compared to 14 asymptomatic controls (40). Strength or power cannot be inferred from EMG, but these data suggest a potential relationship between muscle function and control of extremity speeds. An inability to control extremity speed and loading rates during the weight-acceptance period of gait could result in greater impact loads on articular cartilage and subchondral bone, therefore facilitating OA changes within the joint. However, the findings of the current study suggest that capacity for greater power was related to larger KAM impulses, which may not be desirable in knee OA. Although knee extensor power only explained 6.8% of the variance in KAM impulse, this significant contribution warrants further investigation into the role of the knee musculature's ability to develop force quickly in response to load and, importantly, to determine the effect of power training on medial knee loads for those with symptomatic knee OA.

Nonetheless, in the knee OA literature, research that has examined the relationship between muscle power with self-reported function and performance tasks suggests that power training may have benefits in knee OA (20,41–44). Compared to knee extensor strength, greater leg power measured on a leg press was more strongly related with better performance of fast walking and repeated chair-stand tasks, as well as self-reported function on the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) in 39 adults after total knee arthroplasty (41). Muscle power at lower resistances and higher velocities was more predictive of self-reported physical function on the WOMAC than muscle strength in 40 participants with knee OA (43). Berger and colleagues used a dynamometer to measure knee extensor MVICs and isotonic power at 10%, 20%, 30%, 40%, and 50% of isometric MVIC. Power recorded when resistance was relatively light predicted a small portion of the variance in self-reported function (20% MVIC; $r^2 = 0.13$, $P < 0.05$, and 30% MVIC; $r^2 = 0.12$, $P < 0.05$) (43). Therefore, it appears that although higher knee extensor power could relate to greater exposure to medial knee loading in a stride, it could concurrently relate to improved mobility and reduced symptoms for people with knee OA. While this discrepancy may seem counterintuitive, use of pain medications has also demonstrated this pattern. Pain medications concurrently reduce symptoms and increase the knee adduction moment peak (45).

Furthermore, there are numerous studies in the healthy-aging literature that show muscle power to be a stronger predictor of functional performance than strength. With aging, the knee extensors have shown greater losses of power than strength (19). In more than 1,400 elderly participants, variation in leg power accounted for more of the variance in the performance of important mobility tasks (gait speed and stair climb time) than strength, where poor muscle power was associated with a 2–3-fold greater risk in mobility problems than poor muscle strength (46). More specifically, the velocity component of muscle power was found to be more compromised than the force production component in older adults with mobility limitations (47). When force and velocity contributions to muscle power were compared between middle-aged adults (ages 40–55 years, n = 25), older healthy adults (ages 70–85 years, n = 28), and older adults with mobility limitations (ages 70–85 years, n = 26), velocity was found to be highly associated with power production capability in the older adults with mobility limitations for a multiple chair-rise task ($r^2 = 0.59$, $P = 0.0007$) and stair-climb time task ($r^2 = 0.29$, $P = 0.034$) (47). However, these studies included only healthy aging samples. The positive relationship between power and KAM impulse in this study should be studied further to determine whether power and impulse increase with advancing disease severity over time. Longitudinal study of OA populations would be required in order to determine the validity of this hypothesis.

The impact of power training on disease progression in knee OA remains unclear. Power training, both aquatic and land-based, appears to be well tolerated by people with clinical knee OA (44,48). These pilot studies demonstrate benefits of power training to muscle outcomes as well as symptoms; however, power training programs aimed to improve lower extremity muscle power in knee OA must identify the ideal resistance, positioning, and velocities that relate to activities of daily living, and, for those without severe disease, recreation and sport. Given the positive relationship found between power and both KAM peak and impulse, it will be important to investigate power training regimens and, specifically, to quantify the potential increases in the KAM in people with knee OA. If power training results in an increase in the KAM in people with knee OA, such a finding must be weighed against the other potential benefits to symptoms, mobility, and quality of life. It is very likely that such clinical improvements are more important.

Muscle strength has been postulated as a determinant of the KAM between different levels of disease severity in knee OA (7). In the current study, knee strength did not explain variance in the KAM peak or impulse, nor was it related to these dependent variables. Similar findings have been previously reported in lean and obese knee OA patients, where maximal knee extensor strength was not related to external KAM peak (12,13). These findings support the notion that maximal isometric quadriceps strength may not be the best variable to relate with dynamic knee loading during gait.

A limitation of the study is the cross-sectional study design. Acquiring longitudinal data will aid in our understanding of the impact of changes in muscle power and strength impairments in explaining variability in changes in the KAM. Because of the numerous age-related physiologic and neurologic mechanisms that could lead to declines in muscle power, such as dropout of type 2 fibers, alterations in agonist/antagonist coactivation, and decline in motor unit firing rate (49), other measurements of muscle function, such as using EMG or nerve conductions studies, would help explain mechanisms of underlying declines or increases in muscle power and strength, which may impact mobility. Finally, knee power and strength were measured in this study using a concentric protocol, despite the fact that the knee extensor activity during gait is largely eccentric. The concentric protocol was chosen for these study participants to avoid the potential for muscle injury.

Further investigation is warranted to explore the relationship between isotonic muscle power with gait mechanics and the impact of this relationship on the incidence and progression of knee OA. In this study, the KAM peak and impulse were used to represent medial compartment knee joint loading during walking and a positive relationship was found between KAM and power. Several of the training programs currently in use for those with knee OA are aimed at increasing muscle power. In light of the findings of the current study, these power-focused programs should be studied to weigh the effect of increased medial compartment load with the benefits in terms of mobility and reduced symptoms in knee OA.

In conclusion, unlike strength, knee extensor power shared a positive relationship with the magnitude of the KAM peak and impulse during gait in individuals with clinical knee OA. Cross-sectionally, those capable of pro-

ducing large knee extensor powers also showed large KAM peaks and impulses. Further work is necessary to examine the impact of power training on KAM in a prospective design and relate these findings with improvements in clinical signs and symptoms.

AUTHOR CONTRIBUTIONS

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be submitted for publication. Dr. Maly had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study conception and design. Calder, Beattie, Callaghan, Adachi, Maly.

Acquisition of data. Calder, Acker, Arora, Beattie, Adachi, Maly.

Analysis and interpretation of data. Calder, Acker, Arora, Beattie, Adachi, Maly.

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