associations of peak vGRF and vGRF rate of loading with biomarker concentrations of both serum C2C and CPII. Next, we determined associations for peak vGRF and vGRF rate of loading with a breakdown to synthesis turnover ratio (C2C:CPII), where a higher ratio indicates more collagen break down compared to synthesis. Pearson Product (r) Moment and Spearman (ρ) rank order correlations were used to associate normal and non-normally distributed data, respectively. An alpha level of 0.05 was used for all analyses.

Results: Peak vGRF (1.13 ± 0.07 %BW; ρ= - 0.53, P=0.02) and vGRF loading rate (7.52 ± 2.29 BW/s; ρ= - 0.47, P=0.04) were negatively associated with the serum C2C:CPII concentration ratio (0.33 ± 0.13). Peak vGRF was not significantly associated with C2C (145.22±19.29 ng/ml; r= - 0.25, P=0.33) or CPII (510.53 ± 207.1 ng/ml; ρ= 0.32, P=0.18) individually. Similarly, vGRF loading rate was not significantly associated with C2C (r= - 0.18, P=0.45) or CPII (ρ= 0.27, P=0.27) individually. Post hoc analyses, found no associations between time since ACLr or contralateral vGRF and serum biomarkers.

Conclusions: There were significant moderate associations linking higher peak vGRFs and vGRF loading rates with lower collagen type II breakdown to synthesis ratios, an average of 3.5-4 years following ACLr. These findings are contrary to our a priori hypotheses that higher peak and rate of loading would associate with more collagen breakdown relative to synthesis. Individually C2C and CPII concentrations did not significantly associate with peak vGRF or vGRF loading rate, yet more loading (peak and higher rate) weakly associated with less breakdown and more synthesis. The associations between greater loading and lower serum C2C:CPII concentration ratios may be driven by higher overall concentrations of collagen synthesis, in the involved limb of ACLr individuals with more joint loading. An increase in collagen synthesis may be a response to higher loads exerted in the ACLr limb. More collagen synthesis may be necessary to continue to tolerate higher loads at an injured joint. Future research should examine if patients that develop PTOA synthesize collagen differently in response to higher loading during gait.

148
KINETIC GAIT ADAPTATIONS FOLLOWING ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION: IMPLICATIONS FOR POST-TRAUMATIC KNEE OSTEOARTHRITIS

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Purpose: Anterior cruciate ligament injury and reconstruction surgery (ACLR) incur a 3-5x increase in the risk of developing post-traumatic knee osteoarthritis (PTOA). This OA phenotype progresses rapidly, with as many as 25% of patients developing radiographic joint changes within 5 years. Furthermore, these injuries typically occur in individuals 15-25 years of age, thus PTOA resulting from ACLR represents a long-term health concern. While development of PTOA is multifactorial, loading characteristics of the articular cartilage have received substantial attention in the literature, with greater loading hypothesized to result in greater cartilage degradation. Given the repetitive nature of walking and its integral role in human locomotion, gait biomechanics are commonly evaluated in efforts to identify loading characteristics that influence cartilage degradation. While cartilage loading cannot be directly quantified in vivo, peak impact forces and joint moments are commonly evaluated as biomechanical surrogates. Additionally, animal models suggest that the rate of loading may play a more important role in cartilage degradation than the magnitude of loading. While loading rates during gait have been evaluated in individuals diagnosed with knee OA, we are unaware of any literature evaluating the potential role of loading rates in the development of PTOA following ACLR. Therefore, the purpose of this investigation was to compare gait kinetics between the healthy and surgically reconstructed limbs following ACLR. We hypothesized that greater loading and loading rates would be observed in the ACLR limb compared to the healthy limb.

Methods: Three-dimensional gait biomechanics were obtained from twenty-three individuals with a history of unilateral ACLR (15 females, 8 males; age = 22±4 years, mass = 74±20 kg, height = 1.70±0.12 m, time since ACLR = 39±29 months) via an optoelectric motion capture system integrated with force plates. Subjects walked along a 6m walkway at a self-selected speed. The force plates were staggered such that data for both limbs could be obtained in a single trial. Peak vertical ground reaction force (vGRF; N) and instantaneous loading rate (vGRF-LR; N/s), internal knee extension moment (KEM; Nm), and sagittal plane knee stiffness (KS; Nm/°) were identified during the first 50% of the stance phase. vGRF-LR was calculated as the 1st time-derivative of the vGRF; KEM was calculated via standard inverse dynamics; and KS was calculated as the ratio of the change in KEM to the change in knee flexion angle. Forces were normalized to body weight (xBW) and moments were normalized to the product of body weight and height (%BW*Ht). All dependent variables were compared between healthy and ACLR limbs via paired-samples t-tests ($p \leq 0.05$).

Results: No differences were observed between limbs for peak vGRF (1.13±0.07 xBW vs. 1.14±0.09 xBW, p=0.318) or peak vGRF-LR (59.97±17.51 BW/s vs. 55.44±17.59 BW/s, p=0.216). However, peak KEM (4.4±1.2 %BW*Ht vs. 5.0±1.5 %BW*Ht, p=0.003) and KS (0.35±0.11 %BW*Ht/° vs. 0.40±0.12 %BW*Ht/°, p=0.003) were significantly greater in the healthy limb compared to the ACLR limb.

Conclusions: While ground reaction force characteristics did not differ between the ACLR and healthy limbs, knee extension moment and stiffness were greater in the healthy limb than in the ACLR limb. The vGRF reflects the summative loading of all lower extremity joints. In contrast, KEM and KS are joint-specific. Reduced knee joint-specific loading in the ACLR limb in the presence of similar vGRF characteristics between-limbs suggests that contributions from the ankle and hip extensors to the vGRF are increased in the ACLR limb to compensate for reduced contributions from the knee extensors. These data may also suggest that insufficient, rather than excessive, loading may contribute to PTOA following ACLR. Insufficient loading may alter nutrient and waste exchange characteristics, thus resulting in a cellular energy imbalance due to the avascular nature of articular cartilage. Furthermore, altered joint-specific loading may disrupt the normal catabolic and anabolic processes that are necessary to regulate articular cartilage remodeling. Quadriceps dysfunction is a common, lingering complication following ACLR. As the quadriceps is the primary contributor KEM and KS, future research is necessary to determine if interventions targeting quadriceps dysfunction improve gait biomechanics in manners that would reduce the risk of PTOA following ACLR.

149
GAIT ADAPTATIONS TO EXERCISE-INDUCED FLARES OF OSTEOARTHRITIS RELATED KNEE PAIN

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Purpose: To evaluate the effect of a 20 minute walking stimulus (20MWS) that is intended to simulate participation in daily activity on self-reported pain, ambulatory function (walking speed, vertical ground reaction force (GRF), and stance time) and knee joint kinetics in older adults with chronic knee pain. Acute exercise is known to exacerbate pain in osteoarthritis (OA) on a short term basis, however very little is known about patient compensations to acute flares of OA pain. Understanding the motor system adaptations to pain may provide insight into short and long term efficacy of pain therapies and their impact on joint health. We hypothesized that there would be significant increases in pain and concomitant decreases in ambulatory function and joint kinetics in response to the 20MWS in adults with OA related knee pain.

Methods: Seven older adults (62.3 +/- 6.2 years; 24.8 +/-2.8 kg/m2) were enrolled in the study after completing the informed consent process and a Physical Activity Readiness Questionnaire (PAR-Q+). Inclusion criteria were ages 50-75 years, BMI <35 kg/m2, good general health, met the American College of Rheumatology clinical classification criteria for OA in at least 1 knee, ability to walk unaided, no history of cardiovascular or neurological disorders. Knee symptoms were evaluated using the Knee Osteoarthritis Outcome Score (Table 1). For the 20MWS, subjects walked on a treadmill at a pace similar to their preferred overground speed. Pain was evaluated on an 11 point verbal numeric pain rating scale in the first and final 2 minutes.

Gait analysis was performed pre and post 20MWs while subjects walked overground on an 11m walkway at preferred and faster than preferred speeds. 3 trials per speed were collected at each timepoint. External inter-segmental moments were calculated for the lower limb using an inverse dynamics procedure. The primary gait outcomes were: walking speed, maximum vertical GRF, time in stance on more affected leg, peak external knee flexion, adduction, and internal and external rotation moments on the more affected leg. A 2 x 2 factor Analysis of