601

DRY NEEDLING AND TREADMILL RUNNING ON INDUCING PATHOLOGICAL CHANGES IN RAT ACHILLES TENDON THE EFFECT OF DRY NEEDLING AND TREADMILL RUNNING ON INDUCING PATHOLOGICAL CHANGES IN RAT ACHILLES TENDON TREADMILL RUNNING ON INDUCING PATHOLOGICAL CHANGES IN RAT ACHILLES TENDON

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Purpose: Achilles tendinopathy is a common degenerative condition without a definitive treatment. An adequate chronic animal model of Achilles tendinopathy has not yet been developed. The purpose of this study was to evaluate the individual and combined effects of dry needling and treadmill running on the Achilles tendon of rats.
Methods: Percutaneous dry needling, designed to physically replicate microrupture of collagen fibers in overloaded tendons, was performed on the right Achilles tendon of 80 Sprague-Dawley rats. The rats were randomly divided into two groups: a treadmill group, which included rats that underwent daily uphill treadmill running (n = 40), and a cage group, which included rats that could move freely within their cages (n = 40). At the end of weeks 1 and 4, 20 rats from each group were sacrificed, and bilateral Achilles tendons were collected. The harvested tendons were subjected to mechanical testing and histological analysis.
Results: Dry needling induced histological and mechanical changes in the Achilles tendons at week 1, and the changes persisted at week 4. The needled Achilles tendons of the treadmill group tended to show more severe histological and mechanical changes than those of the cage group, although these differences were not statistically significant. Dry needling combined with free cage activity or treadmill running produced tendinopathy-like changes in rat Achilles tendons up to 4 weeks after injury.
Conclusions: Dry needling is an easy procedure with a short induction period and a high success rate, suggesting it may have relevance in the design of an Achilles tendinopathy model.

Pain and Disability — Clinical

602

PAIN SUSCEPTIBILITY PHENOTYPES IN PEOPLE WITH OR AT RISK OF KNEE OA WITH INCONSISTENT PAIN: THE MULTICENTER OSTEOARTHRITIS STUDY (MOST)

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Purpose: It is well recognized that a number of different factors beyond just structural features contribute to the pain experience in people with knee OA. It is possible that, independent of structural pathology, characteristics such as psychological factors, sleep, and sensitization may increase the risk of an individual developing symptoms and functional limitations. Therefore, we examined pain susceptibility phenotypes in people with or at risk of knee OA using indicators of psychological and neurophysiological aspects of pain and assessed their relation to incident pain and change in function 2 years later.
Methods: We used data from the 60-month visit of the Multicenter Osteoarthritis (MOST) Study, a NIH-funded longitudinal prospective cohort of 3026 older adults with or at risk of knee OA. We identified individuals who were *free* of consistent frequent knee pain (CFKP), defined as pain on most days during the past month at both the clinic visit and a telephone screen ~1 month before the clinic visit. We excluded participants who had a total knee replacement or possible peripheral neuropathy. We used latent class analysis to determine groupings of baseline psychological and neurophysiological characteristics that may identify individuals at higher risk for developing pain or worsening function. We focused on widespread pain (pain above and below the waist, on both sides of the body and axially, excluding knee pain), sleep (4 point scale from very good to very bad), pain catastrophizing (single item from the Coping Strategies Questionnaire), positive affect and depressive symptoms (Center for Epidemiological Studies Depression (CES-D) scale), and quantitative sensory testing (punctate temporal summation and pressure pain thresholds). We examined the relation of these phenotypes to incident CFKP and symptomatic OA (SxOA) defined as having both radiographic OA (ROA: KL grade ≥2 in the tibiofemoral joint) and CFKP in the same knee, 2 years later using the Lanza method for binary outcomes, which does not permit the inclusion of covariates in the model. We therefore used a separate model to examine predictors of class membership, which included age, sex, education, race, BMI and presence of ROA. Lastly, we assessed the relation of the identified phenotypes to change in patient-reported WOMAC function 2 years later using the BCH method, which allows continuous outcomes (WOMAC function) and for inclusion of the phenotypes (classes) and covariates in the same model.
Results: 1202 participants met inclusion criteria (mean, SD age; 67.4, 7.7; BMI 29.8, 5.5 kg/m^2, 57% women). A 3-class model was identified. We labeled the classes as low, moderate and high risk phenotypes due to an increasing prevalence of identified known psychological and neurophysiological risk factors for pain in each of the groups. A similar trend in demographic variables was seen as the largest proportions of women, ROA, and non-Caucasians, having higher age, comorbidities and BMI were found in the high risk group in comparison with the moderate and low risk groups. Women were more likely to be in the moderate and high risk groups, Odds Ratio (OR) 3.36 (2.41, 4.68) and OR 6.89, 95% CI (1.57, 9.84) compared with the low risk group. Those in the moderate and low risk groups had a significantly lower incidence of CFKP over 2 years [unadjusted OR (uOR) 0.52, 95% CI (0.30, 0.75) and uOR 0.65 (0.40, 0.91)] respectively compared with those in the high risk group. Conversely, there were no significant findings for incident SxOA. Change in WOMAC function over 2 years was minimal in the high and moderate groups (means of 0.42 and 0.48 respectively), whereas the low risk group increased the most 1.74. Significant risk factors for decline in function, found only in the low risk group were BMI OR 0.157 (0.01, 0.30) $p < 0.04$, age 0.121 (0.01, 0.23) $p < 0.03$ and non-Caucasian race OR 3.938 (0.65, 7.23) $p < 0.02$.
Conclusions: In a sample of people with or at risk of knee OA free of CFKP, 3 pain susceptibility phenotypes were identified comprising psychological and neurophysiological indicators. Women were more likely to demonstrate the high and moderate pain susceptibility phenotypes suggesting that sex specific pain phenotypes may be an avenue for future study. In contrast to previous trajectory studies, our results indicate that those in the low pain-risk phenotype experience the greatest decline in function. This may be due to existing poor function at baseline in the high and moderate risk phenotypes (i.e., floor effects). Whether longer follow-up time may discern different patterns and risks for developing pain and functional limitations merits further study.

603

ADULTS AT RISK FOR ACCELERATED KNEE OSTEOARTHRITIS REPORT DIFFERENT PATTERNS OF KNEE SYMPTOMS THAN THOSE AT RISK FOR COMMON KNEE OSTEOARTHRITIS: DATA FROM THE OSTEOARTHRITIS INITIATIVE

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Purpose: Accelerated knee osteoarthritis (KOA) may be a unique subset of KOA where a knee progresses from normal appearance to advance-stage disease within 4 years. Individuals with accelerated KOA report greater symptoms (i.e., pain, dysfunction, and other symptoms) than those with common KOA, especially the year before they develop radiographic evidence of accelerated or common KOA. It is unclear if adults who develop accelerated KOA experience symptoms differently than those who develop common KOA. Hence, we examined if adults who develop accelerated KOA had greater knee symptoms with certain activities than those with or without common KOA and if the pattern of symptoms differed between those with accelerated or common KOA.
Methods: We conducted a case-control study using data from baseline and the first 4 annual visits of the Osteoarthritis Initiative. Participants had no radiographic KOA at baseline (Kellgren-Lawrence [KL] < 2). We classified 3 groups: 1) accelerated KOA: ≥1 knee developed advance-stage KOA (KL = 3 or 4) within 48 months, 2) common KOA: ≥1 knee increased in radiographic scoring (excluding those with accelerated KOA), and 3) No KOA: no change in KL grade by 48-months. Blinded readers recorded KL grades based on weight-bearing, fixed-flexion posterior-