Osteoarthritis and Cartilage 27 (2019) 392–400

Osteoarthritis and Cartilage

Review

Osteoarthritis year in review 2018: mechanics

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ARTICLE INFO

Article history:
Received 26 November 2018
Accepted 20 December 2018

Keywords:
Osteoarthritis
Mechanics
Cartilage

SUMMARY

Objective: To review recent biomechanics literature focused on the interactions between biomechanics and articular cartilage health, particularly focused on macro-scale and human studies.

Design: A literature search was conducted in PubMed using the search terms (biomechanics AND osteoarthritis) OR (biomechanics AND cartilage) OR (mechanics AND osteoarthritis) OR (mechanics AND cartilage) for publications from April 2017 to April 2018.

Results: Abstracts from the 559 articles generated from the literature search were reviewed. Due to the wide range of topics, 62 full texts with a focus on *in vivo* biomechanical studies were included for further discussion. Several overarching themes in the recent literature were identified and are summarized, including 1) new methods to detect early osteoarthritis (OA) development, 2) studies describing healthy and OA cartilage and biomechanics, 3) ACL injury and OA development, 4) meniscus injury and OA development, and 5) OA prevention, treatment, and management.

Conclusions: Mechanical loading is a critical factor in the maintenance of joint health. Abnormal mechanical loading can lead to the onset and progression of OA. Thus, recent studies have utilized various biomechanical models to better describe the etiology of OA development and the subsequent effects of OA on the mechanics of joint tissues and whole body biomechanics.

Introduction

Mechanical loading is critical to joint health. However, abnormal mechanical loading can lead to the onset and progression of osteoarthritis (OA)[1]. Specifically, OA results from an imbalance of anabolism and catabolism in the joint, which may be influenced by the biological and mechanical environment[2]. Thus, biomechanical studies are critical to understanding OA development, prevention, and treatment. Various biomechanical models have been utilized in an attempt to better describe the etiology of OA development and the effects of OA on the biomechanics of joint tissues. Furthermore, large-scale biomechanical studies have been used to describe how injury alters the mechanical environment of the joint, whether surgery or other interventions can restore normal joint function, and whether biomechanical measures can be used as indicators of OA risk.

While OA is a disease of joint degeneration involving multiple tissues[3], in this review we focused on studies aimed at understanding the interactions between biomechanics and articular cartilage health. Specifically, this review summarizes the recent literature (between April 2017 and April 2018) regarding these topics. Due to the large number of publications, we have focused on macro-scale and human studies. Specifically, we highlight recent biomechanics studies that investigate 1) new methods to detect early OA development, 2) studies describing healthy and OA cartilage and biomechanics, 3) ACL injury and OA development, 4) meniscus injury and OA development, and 5) OA prevention, treatment, and management.

Methods

A PubMed literature search was performed, and recent publications were reviewed. PubMed was searched for publications between April 2017 and April 2018 with search terms including (biomechanics AND osteoarthritis) OR (biomechanics AND cartilage) OR (mechanics AND osteoarthritis) OR (mechanics AND cartilage). This search yielded 559 results. All of the abstracts from

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https://doi.org/10.1016/j.joca.2018.12.011
1063-4584/

the articles in the search were reviewed. Due to the wide range of topics, 62 full texts with a focus on *in vivo* biomechanical studies were included for further discussion.

Results

Identification of early changes in articular cartilage composition and mechanics leading to OA

Osteoarthritic changes in articular cartilage, including changes in cartilage composition and its response to mechanical loading, occur over time. However, most often OA is not identified until its advanced stages, where radiographic changes have occurred and symptoms are present[4,5]. Thus, recent efforts have focused on identifying earlier markers of the disease, which may aid in developing preventative measures. To this end, new methodologies have been applied to the study of cartilage properties in healthy and early OA tissues. For example, Pedoia *et al.*[6] used machine learning techniques to analyze a data set that included demographics, clinical information, gait kinematics and kinetics, and quantitative magnetic resonance imaging (MRI) relaxometry metrics to identify subpopulations of patients and data that may be associated with OA progression. This study identified that a combination of quantitative MRI metrics, specifically 1/T2 − 1/T1rho, was associated with cartilage lesion progression over 2 years using whole-organ MRI score (WORMS) within a subpopulation of individuals with OA. Previously, T1rho and T2 relaxation times have been shown to be sensitive to alterations in cartilage proteoglycan content and collagen organization, respectively[7−11]. Another study by the same group examined relationships between 3D proximal femur shape, cartilage morphology, joint biomechanics, and cartilage biochemical composition as measured by T1rho and T2 in subjects with hip OA[12]. Their findings revealed that there is a relationship between femur shape variations and morphological and compositional markers of hip joint degeneration, suggesting that 3D MRI-based bone shape variations may be a potential biomarker of early hip joint degeneration. magnetic resonance (MR) diffusion tensor imaging (DTI) at 3T has also recently been investigated as a means to assess collagen and proteoglycan architecture. Specifically, in a study by Ferizi *et al.*[13], osteochondral samples from patients undergoing knee replacement were harvested and mechanically overloaded. DTI, mechanical properties, and histology were performed prior to mechanical injury and at one and 2 weeks post injury. This study found that in the most severe injury group, DTI was sensitive to early changes in cartilage following mechanical injury, and these changes were correlated with alterations in mechanical properties and histology. Furthermore, a recent study explored the use of ultrasound imaging and an acoustic parameter referred to as "average magnitude ratio" (AMR) to reflect cartilage degeneration in enzymatically degraded porcine cartilage *in vitro*[14]. This study suggests that with more development AMR could be a parameter to assist ultrasound diagnosis of OA in the future. These studies reveal the potential utility of new imaging methodologies that may be utilized together with MR imaging techniques[7−9,15] to detect earlier cartilage changes associated with primary OA or joint injury.

In addition to novel imaging techniques, other studies have assessed biochemical biomarkers that may be associated with degenerative changes in cartilage. Osteochondral and synovial samples were harvested from varus-aligned medial OA patients who underwent total knee replacement and biomechanical, histological, and immunohistochemical analyses were performed[16]. In this study, higher numbers of neutrophils, which were detected by CD15 immunostaining, were associated with increased synovitis, increased cartilage degeneration by Osteoarthritis Research Society International (OARSI) grading, and decreased cartilage aggregate and dynamic moduli. This study suggests that large numbers of neutrophils in the synovium may be an indicator of cartilage degeneration and impaired cartilage mechanical properties. Another study tested the hypothesis that changes in serum biomarkers in response to a mechanical stimulus in patients with medial knee OA are related to cartilage thickness changes 5 years later[17]. In this study, the serum biomarkers C1,2C (a type I and type II collagen degradation marker) and CS846 (an aggrecan synthesis marker) were measured following a 30 min walk. MRI was used to measure cartilage thickness at the time of study entry and at a 5 year follow-up. Cartilage thickness changes over 5 years were correlated with changes in biomarkers induced by walking. Interestingly, this study identified medial tibial cartilage thinning in patients with increased C1,2C. Together, these recent studies have improved our understanding of biochemical and mechanical biomarkers with the potential to serve as novel indicators and predictors of cartilage degradation. Together with prior work[18−20], these studies point to an interplay between mechanical and biological factors that are important to consider when assessing OA development and progression.

Healthy and OA cartilage and biomechanics

Studies investigating the relationships between biomechanics, cartilage loading, and cartilage properties in healthy tissue provide important baseline data to compare with diseased populations. In recent publications, healthy cartilage properties and mechanics have been explored using a variety of techniques, including kinematic analysis, *in vivo* imaging techniques, and finite element models (FEM).

For example, a recent kinematic study investigated the effect of knee joint loading, using active and passive orthoses during running, on serum levels of cartilage oligomeric matrix protein (COMP), a biomarker of cartilage metabolism[21]. This study found that COMP levels increased immediately after running with both types of orthoses, and that changes in COMP concentration after physical activity were highly influenced by the COMP baseline level. Another kinematic analysis compared lower extremity kinematics and kinetics between young healthy women with a greater or lesser degree of valgus knee alignment during gait[22]. This study found that the group with greater valgus alignment demonstrated decreased knee abduction moments, knee adduction angles, knee abduction angular impulse, and knee adduction range of motion at peak vertical ground reaction force. These findings may suggest that women with greater valgus knee alignment have biomechanics that promote lateral tibiofemoral joint loading, and thus have increased propensity for lateral OA development.

Various imaging methodologies have recently been implemented to study the relationships between cartilage function and cartilage loading *in vivo* in healthy individuals. For example, Harkey *et al.*[23] investigated the association between habitual walking speed and resting femoral cartilage thickness and deformation using ultrasound after 30 min of walking. These ultrasound images were acquired by having subjects flex their dominant legs to 140° and imaging the femoral condyles above the superior region of the patella (Fig. 1). This study found that while habitual walking speed was not associated with resting cartilage thickness, it was significantly associated with greater medial femoral cartilage deformation in the region superior to the patella. Van Rossom *et al.*[24,25] investigated the relationships between knee cartilage thickness, loading during gait, and cartilage composition measured using T1rho and T2 mapping. Specifically, these studies used gait data to create 3D musculoskeletal models, resulting in estimates of the cartilage contact forces and pressure maps. Local cartilage

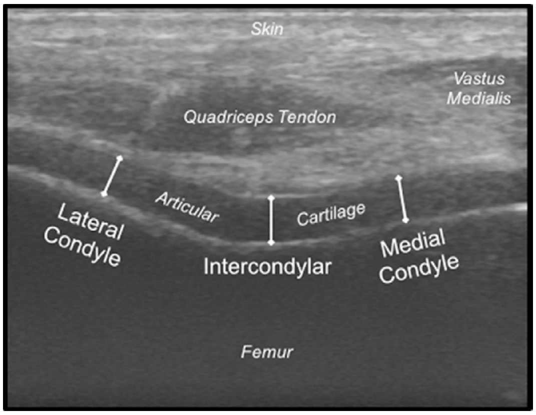

Fig. 1. Ultrasound has been utilized as a tool to measure cartilage thickness *in vivo*. In this study, cartilage thickness was measured in three locations within a single image (lateral condyle, intercondylar space, and medial condyle) and was defined as the distance between the cartilage-bone and synovium-cartilage borders. Image from Harkey *et al.*[23].

thickness was found to be related to local cartilage pressures on both the medial and lateral femoral condyles. Further, these studies found that thicker cartilage was associated with higher condylar loading during walking. Decreased T1rho and T2 relaxation times correlated with increased compressive forces and pressures[24]. Based on these studies, the authors suggest that medial femoral condyle cartilage is "adapted" to localized loading during gait[25]. Also in healthy individuals, Yin *et al.*[26] analyzed *in vivo* tibiofemoral articular cartilage contact biomechanics during a dynamic step-up motion, using MRI and dual fluoroscopic imaging, and found that both the medial and lateral compartments of the knee experienced convex (femur) to convex (tibia) contact in the sagittal plane upon fitting a circle to the curvature of the femoral condyles and tibial plateaus. Together, these studies provide important baseline information regarding cartilage composition and morphology during various activities, which can serve as a point of comparison for identifying the development and progression of disease.

FEM studies have been performed to understand the relationship between joint alignment and cartilage stresses. It was found that during quasi-static loading, femoral internal rotation and adduction led to increased patellar cartilage stress, whereas tibial rotations had minimal influence[27]. A similar methodology was utilized to investigate whether recreational runners with patellofemoral pain (PFP) exhibited greater peak patellar cartilage stress as compared to pain-free runners, and to determine the kinematic and kinetic predictors of peak patellar cartilage stress during running. Knee external rotation was found to be the best predictor of peak hydrostatic pressure and peak maximum shear stress, which were elevated in runners with PFP[28]. Thus, given appropriate boundary conditions, FEM provides additional insight into kinematic movements leading to increased cartilage stress, providing another avenue into understanding the relationship between mechanical loading and OA development.

Several recent kinematic studies have been used to identify the effects of knee OA on activities of daily living, particularly gait[29–35]. For example, Tanimoto *et al.*[30] studied shank angular velocity using an inertial sensor during the swing phase of gait in subjects with knee OA. This study found that worse knee function scores were correlated with decreased angular velocity and increased variability in angular velocity, suggesting that control of the swing limb is affected by degree of disability. Another study[31] examined differences in gait mechanics and muscle activation between asymptomatic and symptomatic individuals with radiographic evidence of knee OA. The authors of this study suggested that symptomatic individuals have stiffer frontal and sagittal plane dynamics during gait, with more muscle activity and more torsional loading in the transverse plane than the asymptomatic group. Ogaya *et al.*[32] applied a musculoskeletal model of the lower limbs and trunk with 92 muscle-tendon units to subject-specific gait data obtained from individuals with knee OA and healthy elderly control subjects. Individual muscle contributions to knee extension mechanics during the early stance phase of gait were analyzed. This study found that OA patients had decreased dependency on vasti muscles to control knee movement during early stance as compared to control subjects. Furthermore, this study found that hip adductor muscles compensate in part for this weaker knee extension through control of mediolateral motion. A study by Roberts *et al.*[36] compared measures of *in vivo* dynamic knee joint loads obtained from gait analysis in advanced stage OA, and microarchitecture of subchondral trabecular bone samples collected during total knee arthroplasty. This study suggests that anteromedial tibial plateau bone volume correlates with peak external rotation moment, and that medial to lateral bone volume ratios correlate with measures of medial to lateral joint loading, such as knee adduction moment.

A number of recent kinematic studies focused on changes in biomechanics associated with hip OA[29,37]. For example, one study investigated differences in gait mechanics between men and women with hip OA[29]. This study found sex-based differences in gait mechanics that are normally observed in asymptomatic individuals were not observed in those with OA. Asymptomatic women had 12% increased peak adduction moments and 23% increased external rotation moments than asymptomatic men, while women and men with hip OA had no significant differences. This finding suggests the need to account for sex when designing interventions. Another study identified that high daily cumulative hip moments in the frontal plane predicted radiographic hip OA progression over 12 months in female patients[37]. Changes in biomechanics in stair ascent and descent in patients with hip OA were also studied[38]. It was found that patients with hip OA had limited range of hip joint motion during stair ascent and employed compensatory strategies that suggest impaired hip abductor function. These studies have revealed new insights into the physiologic function of cartilage and the relationships between kinematics and joint loads, as well as the associations between *in vivo* biomechanics and cartilage properties.

ACL injury and OA development

ACL injury increases the risk of OA development. Several recent studies have investigated alterations in kinematics, cartilage function, and cartilage morphology following ACL injury that may be related to OA development. One study characterized the proteoglycan and collagen content following ACL transection (ACLT) in a rabbit model and related these measures to altered chondrocyte mechanics that were observed upon mechanical loading of cartilage (Fig. 2)[39]. This study identified that at 9 weeks following ACLT, proteoglycan content was reduced and collagen orientation was altered in the cartilage of the superficial zone. These changes were associated with changes in chondrocyte morphology. The authors hypothesized that the chondrocyte deformation response to mechanical compression in OA changes because of alterations in the matrix structure. Another study assessed whether different approaches of ACL rupture, specifically surgical transection or nonsurgical rupture induced by joint loading, had different effects on joint biomechanics in rodent models of post-traumatic OA[40]. An increase in anteroposterior knee laxity was found in the surgical transection model, while no differences were detected in the

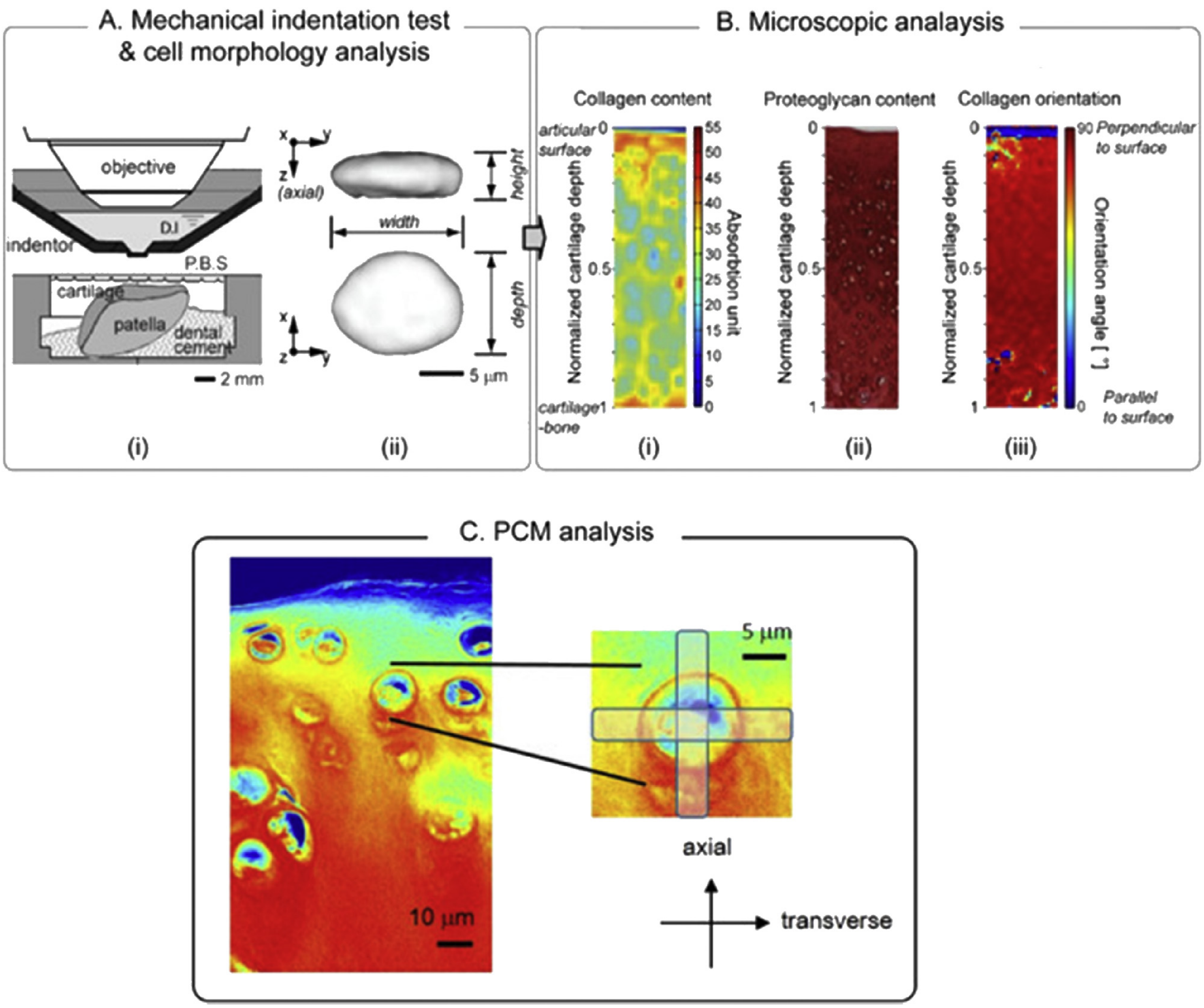

Fig. 2. Mechanical indentation and analysis schematic (A), microscopic analysis via Fourier transform infrared imaging, digital densitometry, and polarized light microscopy (B), and pericellular matrix analysis based on optical density profiles for proteoglycan content (C) from Han *et al.*[39].

overloading model. These findings are important when considering the use of animal models in studying OA following ACL injury.

Recent studies have focused on the lasting effects of ACL injury, even after ACL reconstruction (ACLR). For example, Pietrosimone *et al.*[41] identified changes in gait mechanics that were associated with alterations in biochemical markers of cartilage metabolism at 6 months following ACL injury. This study found decreased biomechanical loading in the ACLR limb, which was associated with markers of deleterious joint tissue metabolism, such as matrix metalloproteinase-3, interleukin-6, and a ratio of collagen breakdown to collagen synthesis (C2C/CPII). Another study identified that individuals with ACLR who had poor knee function (determined by self-reporting and/or hop test performance) had changes in knee moments during running that may be associated with lower knee joint contact forces[42]. Wellstandt *et al.*[43] determined that hip joint biomechanics early after ACL injury and reconstruction differ between those with and without OA. Specifically, this study determined that patients with knee OA 5 years after reconstruction walked with smaller sagittal plane hip angles and lower sagittal and frontal plane external hip moments both before and after reconstruction than those who did not develop OA within the 5 year time frame. Kim *et al.*[44] identified regions of increased T2 relaxation times in the patellofemoral cartilage, particularly in the medial trochlear region, 3 years post-ACLR even when reconstructions were clinically successful. The authors suggest that the abnormal patellofemoral cartilage loading after reconstruction likely leads to these changes in cartilage composition, which are detected by increased T2 relaxation times. Together, these studies suggest that early changes in gait biomechanics following ACL injury and ACLR may be associated with changes in joint tissue metabolism and cartilage composition, potentially leading to OA development.

Several recent studies in the literature have attempted to quantify the effects of ACL injury on cartilage function and health. One *in vivo* study[45] compared cartilage mechanics and thickness between ACL deficient and intact knees using MRI based 3D surface models of the knee joint. ACL deficiency was associated with decreased patellar cartilage thickness and increased exercise-induced patellar cartilage strain as compared to ACL intact knees. The authors suggest that this is representative of altered patellofemoral joint loading and possible degeneration of the patellar cartilage. Another study[46] found that knees with single-bundle hamstring ACLR without meniscal injury had thinner cartilage as compared to healthy knees 2–3 years post reconstruction.

Furthermore, subjects with both ACLR and meniscal injury experienced greater contact forces, which were associated with increased lateral cartilage volume and thickness. Finally, a study by Chen *et al.*[47] examined changes in dynamic contact stress profiles in the tibial plateau after ACL transection using cadaveric models. This study identified differences in the locations of the stress profiles on the tibial plateaus between the intact and ACL transected knees, although the magnitude and shape of the stress profiles remained the same. Together, these findings provide evidence that altered loading associated with ACL injury results in changes in the loading experienced by the cartilage, which may ultimately lead to cartilage degradation.

Furthermore, the relationships between altered biomechanics and cartilage health following ACL injury have been investigated using quantitative MRI in recent studies. Teng *et al.*[48] assessed gait and tibiofemoral joint cartilage properties via T1rho and T2 relaxometry in subjects before ACLR, 6 months after surgery, 1 year after surgery, and 2 years after surgery. This study found that higher knee flexion moments, knee flexion angles and ground reaction forces were associated with increases in T1rho and T2 relaxation times at later time points post-surgery. Thus, this study demonstrates a link between altered gait mechanics after ACL injury and changes in cartilage composition within a relatively short time frame from reconstruction[48]. Similarly, the use of quantitative ultrashort echo time enhanced T2* mapping (UTE-T2*) for early detection of OA was investigated[49,50]. Increased UTE-T2* values of the medial knee cartilage correlated with increased varus alignment and knee adduction moment. Together, these studies suggest that altered gait mechanics are related to degradative changes in the cartilage after ACL injury.

Given the high likelihood of OA development following ACL injury, several studies have investigated factors related to improving post-ACL injury outcomes and reducing the risk of further injury. Interestingly, Filbay *et al.*[51] found that delaying ACLR and managing ACL rupture with exercise therapy alone may shift prognostic factors for 5 year clinical outcomes in a positive direction. A FEM was utilized to optimize graft placement in ACLR surgery[52]. This study found that ACL graft placement had a significant influence on cartilage contact pressures and meniscal stresses. This information may guide surgeons to optimize ACLR surgery to minimize deleterious alterations in biomechanics that may be related to OA development. In a randomized control trial of the efficacy of 10 post-operative gait training sessions in individuals with gait asymmetries following ACLR[53], it was determined that the gait training programs tested were not able to alter gait mechanics in male athletes in the short term. However, meaningful gait asymmetries were mostly resolved 2 years after ACLR. Finally, a study by Levins *et al.*[54] concluded that the geometry of the femoral notch and tibial spine of the contralateral knee at the time of ACL injury are associated with the risk of suffering a contralateral ACL injury in female athletes. This information has the potential to identify individuals at increased risk for ACL injury, who might benefit from targeted risk-reduction interventions. Together, these recent studies have investigated means to avoid the negative effects of ACL injury on long-term joint health.

Meniscus injury and OA development

The meniscus is known to be a critical stabilizer of the knee joint. Often, those with meniscal injury or degeneration subsequently develop OA. In order to understand the development of OA following meniscus injury, it is imperative to understand its mechanical and structural properties. A recent study by Luczkiewicz *et al.*[55] used a FEM of the knee based on MRI to model varying heights, and thus cross-sectional shapes, of the meniscus with a compressive load of 1,000 N. They found that the changes in meniscal cross-section affected the meniscal external shift, mediolateral translation, and congruency of the knee. In addition to studying the shapes and heights of the meniscus, Shriram *et al.*[56] used FEM to study the effects of stiffness variations in polycarbonate urethane artificial meniscal implants. The model showed that the artificial menisci exhibited lower peak cartilage contact pressure compared to the meniscectomized knee and that the implant with a stiffness of 11 MPa restores the intact knee contact mechanics[56]. These studies highlight important parameters for consideration in predicting the susceptibility of different shaped menisci to lead to cartilage degeneration and considerations to allow optimization of meniscal implants for knees that have already suffered from catastrophic meniscus degeneration.

To assess meniscal lesion effects on cartilage *in vivo*, Russell *et al.*[57] MR imaged healthy and posterior horn meniscus-injured human subjects both cross-sectionally and longitudinally over 3 years. At baseline, there were elevated T1rho relaxation times in the lateral tibia adjacent to the meniscus lesions, despite the fact that the subjects were not diagnosed with OA by radiograph. After 2 years, T2 relaxation times were elevated in both the medial and lateral tibia in the meniscus-injured group and overall these subjects had lower quality of life, as measured by the Knee Injury and Osteoarthritis Outcome Score (KOOS). Despite these findings, at baseline there was no difference in gait biomechanics between the healthy and injured group. This study points to the use of T1rho and T2 as noninvasive tools, which are sensitive to early changes in cartilage composition following meniscus injury. Jacobs *et al.*[58] assessed differences in gait profiles of rats with medial meniscus transection (MMT) and monoiodoacetate (MIA) injection to simulate OA. The combined analysis of dynamic gait data with spatiotemporal data increased the sensitivity of detecting differences between the two OA models (Fig. 3). Specifically, a shuffling gait was observed in the MMT model and an antalgic gait in the MIA model. Thus, this methodology may provide valuable data on gait characteristics in response to different joint injuries in rodent models.

Due to the lack of clear guidance on when to perform a partial meniscectomy in patients with an MRI verified meniscus tear, Hare *et al.*[59] investigated whether symptoms related to medial meniscal injuries in middle-aged patients are distinct or similar to those with early radiographic knee OA. Over 80% of the patients had moderate severity for self-reported knee pain, pain during stair walking and when twisting the knee, and lack of confidence in their knee. These symptoms are indistinguishable from symptoms reported in patients with early radiographic knee OA[59], leading the authors to infer that these degenerative meniscal tears are early signs of knee OA.

Toward OA prevention, treatment, and management

Slowing the progression of OA is of utmost importance. Thus, several studies have focused on treatment strategies for improving gait biomechanics in individuals with OA. For example, a study by Brenneman *et al.*[60] investigated a lower limb strengthening protocol for its effect on improving gait biomechanics in women, and found only subtle differences in gait biomechanics after the intervention, despite improved self-reported strength. Another notable study[61] used biofeedback assisted gait training to reduce medial tibiofemoral contact force (MTFF) during gait. This study demonstrated that subjects were able to successfully increase their MTFF by modifying their gait, although they had more difficulty decreasing their MTFF using the biofeedback protocol. Finally, another study[62] examined the effect of a gait treatment program using a biomechanical device (APOS System) in hip OA patients.

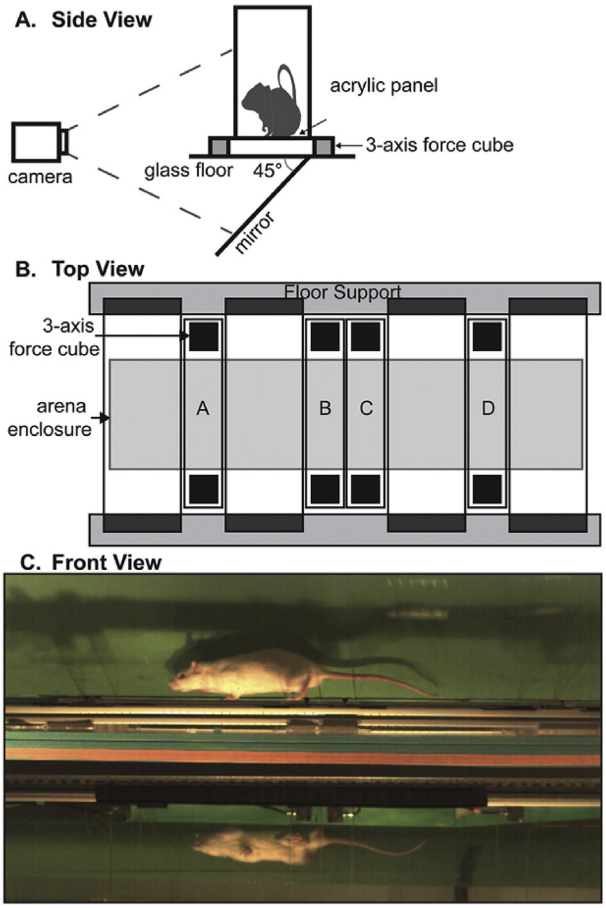

Fig. 3. The Experimental Dynamic Gait Arena for Rodents (EDGAR) was used to obtain spatiotemporal and dynamic gait data. This schematic shows a side view of EDGAR (A), floor design with instrumentation panels (B), and a single frame from a representative trial video (C). Image from Jacobs *et al.*[58].

Sagittal-plane hip joint kinematics and kinetics improved throughout the study, suggesting improvement of gait with the intervention. These studies highlight the recent challenges, as well as successes, in working toward the development of training protocols aimed at reducing the effects of OA.

Furthermore, recent studies have focused on methods for unloading the cartilage in an effort to prevent disease progression. Trad *et al.*[63] used a FEM to investigate the effect of varying the high tibial osteotomy correction angle on the stress distribution in both compartments of the human knee joint, and to determine the optimal correction angle necessary to achieve a balanced loading between the medial and lateral compartments. This study found that shifting the correction angle to a valgus alignment of 0–10° shifted the mechanical load from the medial to the lateral compartment, and that a balanced stress distribution was achieved by 4.5° of valgus correction. This balanced stress distribution unloads the medial compartment, which may slow the progression of OA. Furthermore, Birmingham *et al.*[64] found that, in patients with knee OA and varus alignment, a medial opening wedge high tibial osteotomy improved their KOOS scores by an average of 14.2 points at 5 years after surgery. Thus, this surgery may influence symptoms of OA. Goodwin *et al.*[65] investigated the effects of orthotic devices that are meant to limit cartilage loading after trauma in young healthy individuals. Subjects completed above ground walking using a medial unloader brace and a lateral heel wedge. These devices did not significantly affect gait mechanics in young healthy individuals. Kluge *et al.*[66] investigated the interaction between gait speed and lateral wedge insole on gait biomechanics in an attempt to identify whether walking speed can influence the effectiveness of lateral wedges in reducing knee adduction moment. This study determined that different walking speeds did not confound the effect of lateral wedge insoles in healthy individuals, and that the insoles were successful in reducing the external knee adduction moment and angular impulse. Finally, another study[67] investigated the effect of a hip brace on unloading the cartilage in patients with hip OA. Using gait analysis, this study determined that peak hip abduction moment was decreased on the OA side with use of the brace, which was associated with decreased pain. These recent studies have assessed a variety of treatment options, ranging from surgical techniques to braces, and have shown the beneficial effects of these treatments on biomechanics and cartilage loading.

Conclusion

Over the past year, biomechanical studies have provided new and exciting advances in elucidating the relationships between biomechanics, cartilage properties, and OA development. Notably, studies have indicated that biomechanical measurements may potentially serve as both predictors and early indicators of joint degeneration. Earlier detection of OA development may in the future allow for earlier intervention and disease prevention. Notably, several non-invasive imaging methods of probing joint health have been examined[6–8,14,23–25,44,48–50,57,68]. Additionally, correlating biomechanical measurements with serum or synovial fluid biomarkers has also been on the rise in orthopedics, with the hopes of linking the biological and biomechanical changes in OA. Importantly, recent biomechanical studies have further confirmed that cartilage is adapted to physiologic *in vivo* joint loading and disruption of this balance may contribute to joint degeneration. To this point, several studies have assessed the effects of knee injuries on both biomechanics and cartilage health. Furthermore, a significant effort has also been made towards researching ways to stall the progression of OA. Overall, significant strides have been made in investigating biomechanics in various OA populations, including primary OA and ACL and meniscus-injured subjects at the molecular and whole-body scale to better understand the mechanical and biological relationship affecting the pathogenesis and progression of OA. While much progress has been made in the field, there is still much work to be done in order to prevent the development and progression of OA.

Author contributions

All of the authors searched the literature, selected the articles, summarized the results, and wrote the manuscript.

Conflict of interest

The authors have no conflict of interest.

Acknowledgments

The authors would like to thank the National Institutes of Health (AR065527, AG028716), the Veteran's Affairs Rehabilitation Research Service Merit Review Award, and the Orthopaedic Research and Education Foundation (with funding provided by the Musculoskeletal Transplant Foundation).

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