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Masterclass

Knee osteoarthritis: Clinical connections to articular cartilage structure and function

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ABSTRACT

Articular cartilage is a unique biphasic material that supports a lifetime of compressive and shear forces across joints. When articular cartilage deteriorates, whether due to injury, wear and tear or normal aging, osteoarthritis and resultant pain can ensue. Understanding the basic science of the structure and biomechanics of articular cartilage can help clinicians guide their patients to appropriate activity and loading choices. The purpose of this article is to examine how articular cartilage structure and mechanics, may interact with risk factors to contribute to OA and how this interaction provides guidelines for intervention choices This paper will review the microstructure of articular cartilage, its mechanical properties and link this information to clinical decision making.

1. Introduction

Osteoarthritis (OA) is one of the top causes of disability in the world. According to the Centers for Disease Control, osteoarthritis affects an estimated 26.9 million adults in the United States (CDC, 2011). Osteoarthritis accounted for 55% of all arthritis-related hospitalizations with 409,000 hospitalizations for OA as the principle diagnosis in 1997 (Lethbridge-Cejku, Helmick, & Popovic, 2003). Costs associated with OA are typically underreported due to reporting methodology, and the fact that many people with OA do not enter the medical system until symptoms progress. The costs associated with OA in the most severe cases are typically confined to the elderly and these are the statistics most frequently reported. However, the direct medical costs of OA are only one part of the real costs of OA, particularly among the younger working population with OA. Indirect costs impact the productivity of workers, resulting in significant economic losses. These indirect costs include absenteeism, needed help with household or yard work, and declines in overall health due to inactivity (Dibonaventura, Gupta, McDonald, Sadosky, Pettitt, & Silverman, 2012; Gupta, Hawker, Laporte, Croxford, & Coyte, 2005). A cross-sectional analysis of nearly 5000 workers aged 20 years and older found that as the severity of OA increased, workers reported more frequent pain, poorer quality of life, greater use of healthcare resources and reduced productivity (Dibonaventura et al., 2012). Estimated total annual costs per worker ranged from $9801 for those with mild OA to $22,111 for those with severe OA, compared with $7901 for workers without OA. Indirect costs, based on loss of productivity, accounted for 70–74% of the total costs.

A number of factors are known to contribute to the development of OA (Alkan, Fidan, Tosun, & Ardicoglu, 2013; Farrokhi, Piva, Gil, Oddis, Brooks, & Fitzgerald, 2013; Holla et al., 2012; Holsgaard-Larsen & Roos, 2012; Wesseling et al., 2013). Most notably is aging, with a higher prevalence of OA in the elderly population (Buckwalter & Martin, 2004; Buckwalter, Martin, & Mankin, 2000; Ghosh & Smith, 2002; Zhai et al., 2006). Body mass index (BMI) and sex appear to be contributors, and other factors such as dietary intake have been implicated (Amin et al., 2008; Beavers, Serra, Beavers, Cooke, & Willoughby, 2010; Berenbaum, Eymard, & Houard, 2013; Griffin, Huebner, Kraus, Yan, & Guilak, 2012; Hansen, English, & Willick, 2012; Lohmander, Ostenberg, Englund, & Roos, 2004; de Luis, Izaola, Garcia Alonso, Aller, Cabezas, & de la Fuente, 2012; Muraki et al., 2013; Nguyen, Zhang, Zhu, Niu, Zhang, & Felson, 2011; O'Conor, Griffin, Liedtke, & Guilak, 2013; Shen et al., 2013). Hereditary factors also contribute to the problem. Multivariate regression found an odds ratio of 3.0 for the development of knee OA following meniscectomy in patients who have hand OA suggesting a genetic component due to the heritable nature of hand OA (Englund, Paradowski,

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& Lohmander, 2004). Research has shown a genetic contribution of 39–65% in knee OA, 60% at the hip and a 70% contribution to spine OA for an overall averaged genetic contribution of nearly 50% (Spector & MacGregor, 2004).

Additionally, trauma or joint injury increases the likelihood of OA development over the course of a lifetime (Kramer, Hendricks, & Wang, 2011; Lohmander & Felson, 2004). For those involved in sports and other athletic endeavors, the relationship between injuries such as meniscus and ligament tears and the long term health of the joint must be explored. The risk of posttraumatic OA, even in the presence of anatomic fracture fixation and ligament reconstruction, ranges from 20% to 50% (Kramer et al., 2011). For those with anterior cruciate ligament injuries (ACL), the risk of OA increases over the course of a lifetime (van der Hart, van den Bekerom, & Patt, 2008; Hunter, 2012; Stein et al., 2012). In a study of female soccer players 12 years after their index injury, Lohmander et al. (2004) found that 51% had radiographic changes that met the criterion for radiographic knee OA, compared with 7% on the uninjured side. Nearly 75% reported having symptoms that affected their knee-related quality of life. Additionally, injury to the meniscus, with or without concomitant ACL injury, leads to progressive deterioration of the knee (Englund, Guermazi, & Lohmander, 2009; Englund, Roemer, Hayashi, Crema, & Guermazi, 2012). It has been suggested that the meniscal damage is a better predictor of the eventual development of OA than the ACL tear itself (Amin et al., 2008). Those who have combined ACL and meniscus injuries appear to be at an even higher risk of OA than those who sustain an isolated injury (Brophy, Rai, Zhang, Torgomyan, & Sandell, 2012). In a review and meta-analysis, Richmond, Fukuchi, Ezzat, Schneider, Schneider, and Emery (2013) found that a history of joint injury increased the odds ratio of developing knee OA to 3.8, and hip OA to 5.0. A history of meniscectomy with or without ACL injury increased the odds of developing OA to 7.4. Anderson et al. (2011) summarized the trauma-associated incidence by stating that knee joint ligamentous or capsular injury results in a nearly 10-fold increase in the risk of OA.

Despite these odds ratios and the apparent increase in OA prevalence in those who have sustained injuries, there remains a portion of the population who do not develop OA (Baumgarten, 2007; Bedson & Croft, 2008). We only know about the patients who present for treatment; those who are successful in return to activity with minimal or no symptoms are rarely found in our research studies. Researchers continue to study the causes and relationships among the many factors (i.e. joint trauma, high body mass index, mechanical overload, age, female gender, genetics, occupation, diet and oxidative stress) that contribute to the development and progression of OA (Neogi, 2013; Regan, Bowler, & Crapo, 2008; Sutipornpalangkul, Morales, Charoencholvanich, & Harnroongroj, 2009; Zhang & Jordan, 2010). Despite the vast body of knowledge on OA, the mechanisms for the development and progression of OA are not clearly understood (Aigner, Rose, Martin, & Buckwalter, 2004). Not all patients who sustain joint trauma will go on to develop symptomatic OA, nor will all individuals develop OA as they age. Those who do develop symptomatic OA as they age may have very different rates of development and eventual outcomes (Holla et al., 2013).

Given the high prevalence of OA, interventions to prevent and treat the impairments, activity limitations and participation restrictions are imperative. The purpose of this article is to examine how articular cartilage structure and mechanics may interact with risk factors to contribute to OA and how this interaction provides guidelines for intervention choices. Rather than providing specific protocols, this paper will provide a theoretical link between articular cartilage microstructure, macrostructure and the guidelines rehabilitation professionals use when determining prognosis and designing a rehabilitation program. For the reader who is looking for specific protocols following articular cartilage injury or surgery, there are a number of excellent papers with this information (Irrgang & Pezzullo, 1998; Mithoefer, Hambly, Della Villa, Silvers, & Mandelbaum, 2009; Mithoefer, Hambly, Logerstedt, Ricci, Silvers, & Della Villa, 2012; Stone & Schaal, 2012; Vogt et al., 2013; Wilk, Briem, Reinold, Devine, Dugas, & Andrews, 2006).

2. Articular cartilage macrostructure

Articular cartilage is a unique, specialized tissue that, in a healthy state, allows nearly frictionless movement across its surface. Its structure and mechanical properties allow decades of repetitive loading forces, despite a limited capacity for repair. No artificial material has been able to replicate these properties.

Most articular cartilage is approximately 3–4 mm thick, while the patellar articular cartilage is between 6 and 8 mm thick (Bhatia, Ghodadra, & Verma, 2009; Schiller, 1995). Grossly, articular cartilage is comprised of cells (chondrocytes), matrix (fibers) and extracellular matrix. Articular cartilage's four layers or zones (superficial, transitional, deep and calcified) are distinguished by the shape and orientation of the chondrocytes, and the distribution of type II collagen (Bhatia et al., 2009; Schiller, 1995) (Fig. 1: layers of articular cartilage). The superficial or tangential zone is the outermost surface that is designed to resist forces from arthrokinematic roll, spin and glide. This layer contains elongated chondrocytes with fibers resting parallel to the surface. This orientation, along with its covering called the lamina splendens, optimizes resistance to shear forces (Walker, 1996). While this zone is the most cell and fiber-rich of all layers, the chondrocytes are relatively metabolically inactive making repairs to this outermost layer difficult (Bhatia et al., 2009; Walker, 1996). Therefore any injury or degenerative process that damages this protective surface will expose the underlying layers to shear forces. The adult who sustains a chondral injury may have difficulty healing due to loss of this protective layer.

The transitional, or middle zone contains fibers of a larger diameter that are more randomly dispersed along with chondrocytes which contain more metabolically active intracellular components (i.e. endoplasmic reticulum, mitochondria, Golgi membranes) suggesting a stronger repair capability (Bhatia et al., 2009). Below the transitional zone is the deep, or radial zone containing fibers that are larger than the previous two layers. Chondrocytes are oriented vertically relative to the underlying bone and articular surface, and similarly, fibers are perpendicular to the articular surface. Proteoglycan content is highest and water content the lowest in this zone (Bhatia et al., 2009). Unlike the

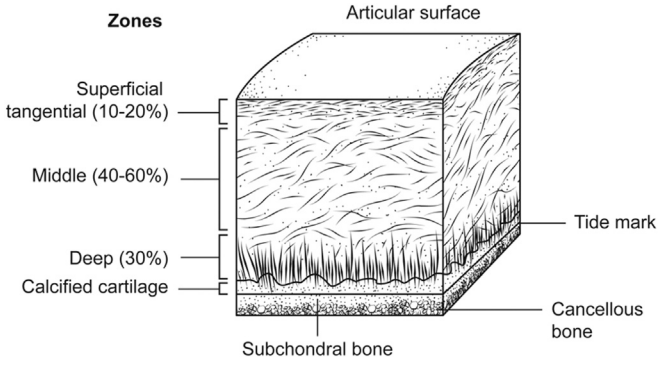

Fig. 1. Layers of articular cartilage. Note the horizontal orientation of cells in the superficial layer, contrasted with the vertical orientation in the deep layer.