WONDER WHY?

The Ligament Injury Connection to Osteoarthritis

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ABSTRACT

Osteoarthritis (OA) or degenerative joint disease (DJD) is more common than all the other types of arthritis combined. It is well-established that injury to a joint increases the chances that the joint will develop osteoarthritis over time. Precipitating causes include sudden impact or trauma, overuse or repetitive motion injuries, biomechanical abnormalities (congenital or acquired), ligamentous injury, joint hypermobility, obesity, intra-articular or systemic corticosteroids, avascular necrosis, and hereditary factors. Osteoarthritis, though the accepted term used to describe degenerative joint disease, is misleading because it primarily relates to cartilage, not bone, and involves degeneration, not inflammation. A lack of understanding about the development of osteoarthritis has resulted in a broad array of symptom-based treatment options such as rest, ice, heat, analgesics, anti-inflammatories, narcotics, braces and wraps, physical therapy and exercise, chiropractic, viscosupplementation, corticosteroid injections, and surgery. While advances have been made in joint replacement, cartilage repair, cartilage replacement, and spinal procedures, treatments to limit or even reverse articular cartilage breakdown have been lacking. Being that ligament injury, excess laxity, joint hypermobility, and clinical instability are known to be major causes of osteoarthritis, any treatment which can address restoration of ligament function would help reduce the incidence, pain, and dysfunction of osteoarthritis.

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KEYWORDS: cartilage, degeneration, hypermobility, instability, ligaments, osteoarthritis.

INTRODUCTION

Osteoarthritis (OA) is the most common form of arthritis and is typically found in the older population. With the aging of the active "baby-boomer" generation, the number of people who suffer from OA is expected to skyrocket. Also, there has been a rise in the number of reported cases in the younger adult populations and it is frequently associated with joint injuries. There are intrinsic causes for OA (defined as primary OA) which have a genetic and/or biomechanical etiology and extrinsic causes (defined as secondary OA) which are caused by external factors. Secondary OA is caused by sudden impact, direct trauma, overuse or repetitive motion injuries, avascular necrosis, corticosteroids, obesity, and ligamentous injury with resultant joint hypermobility and instability.

The ligamentous causes of OA will be the primary focus of this article. OA can appear in synovial joints, which are composed of cartilage, bone, and joint fluid contained within the joint capsule.[1, 2] Examples of synovial joints are the knees, hips, shoulders and fingers. (*See Figure 1.*) Osteoarthritis can also be found in the non-synovial joints of the cervical, thoracic, and lumbar spine regions. There are no standard treatment options which have been able to decrease or eliminate pain due to osteoarthritis, much less arrest the development of the disease. Progression of degeneration often eventually leads to joint replacement or spinal fusion. As a last resort, surgery is agreed upon by surgeon and patient when the pain, disability, and imaging studies are determined to be of sufficient degree to warrant it. While many joint and spine surgeries have

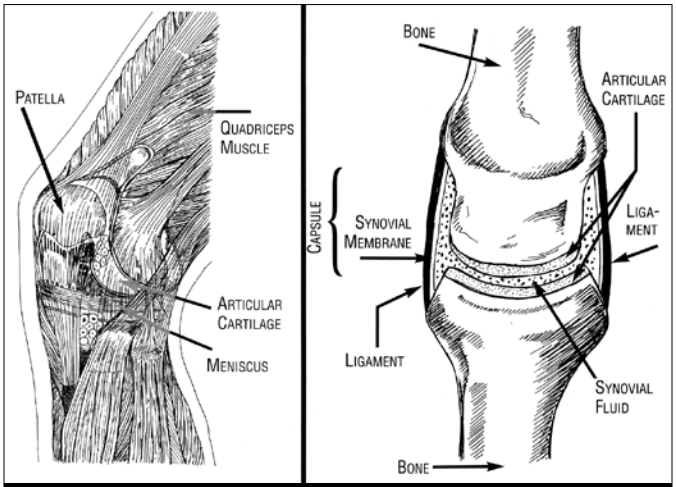

Figure 1. A synovial joint. The knee is an example of a synovial joint.

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a successful outcome, there are an alarming number of surgeries that aren't successful, usually not due to poor surgical technique, but rather due to an improper determination that degenerative joint cartilage and spinal discs are the only sources of a patient's pain. Much of this can be attributed to the surgeon exclusively relying on imaging studies, such as X-rays and magnetic resonance imaging (MRI), which do not reveal the significant pain generators of ligaments, joint capsules, muscles, and tendons. Therefore, because these soft tissues (connective tissues) are not considered in the diagnosis and alternative interventions are not presented in the discussion, many unnecessary surgeries are performed.

PREVALENCE

The number of reported cases of osteoarthritis have been on the rise in the past quarter century. In 1995 it was projected that approximately 21 million Americans suffered from osteoarthritis. (*See Figure 2.*) As of 2005, based on data collected from The National Health and Nutrition Examination Survey I (NHANES I), osteoarthritis affected 27 million of the 46 million people in the United States that suffer from arthritis. Also, recent data shows that one out of two Americans are at risk for knee osteoarthritis over their lifetime.[4] Hip osteoarthritis occurs in 0.7 to 4.4% of adults and knee osteoarthritis occurs in approximately 5% of the American population between the ages of 35 to 54.[3, 5-7]

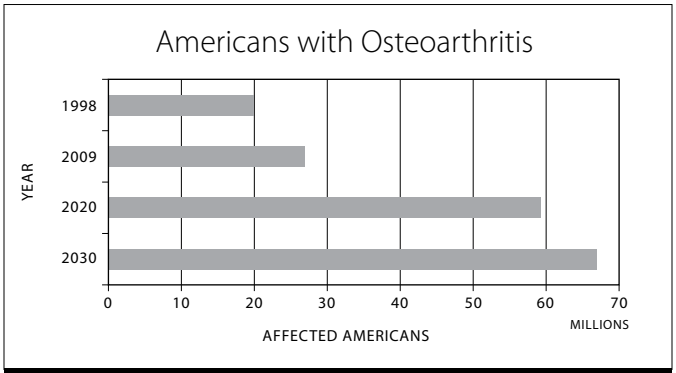

Figure 2. Projected amount of Americans with osteoarthritis.

While many joint and spine surgeries have a successful outcome, there are an alarming number of surgeries that aren't successful, usually not due to poor surgical technique, but rather due to an improper determination that degenerative joint cartilage and spinal discs are the only sources of a patient's pain.

It is estimated that 15% of the world's population also experiences pain and joint degeneration due to the presence of osteoarthritis.[8] The number of hospitalizations as a result of osteoarthritis has doubled in the last 15 years. In 1993, there were 322,000 hospitalizations, and in 2006 the number rose to 735,000.[9]

MEDICAL COSTS

The cost of treatment for osteoarthritis can put a large burden on both the patient and the health care system alike. Medications, even if effective in reducing pain, exact a great cost over the long-term, both in the costs of the medications themselves but also relative to the side effects, complications, and secondary medical problems (morbidity and mortality). The many treatment options that are regularly used to treat OA will be discussed later in this article but some perspective should be given here as to the financial burden associated with OA considering both medical/surgical (direct) costs and work-loss (indirect) costs.

One report estimated the total cost of bilateral knee joint replacements at over $85,000. This included the hospital stay, surgeon fees, anesthesiologist fees, a 5-day stay in an inpatient rehabilitation center, and a pathologist visit. However, this did not include outpatient physical therapy because the length of treatment is unknown. Luckily for this patient, much of the expenses were covered by insurance.[10] The cost of hip and knee replacements have risen from about $7,000 in 1997 to an average of $32,000 for the knee and $37,000 for the hip in 2003.[11] Another option for joint replacement is to travel overseas. Vibrant Medicare reported hip joint replacement costs in India to be between $5900 and $7300 (US currency), while in the UK the costs were between $13,700 and $19,800 (US currency). An estimated $7.9 billion were spent on hip and knee replacements in the United States in 1997.[12]

The average out-of-pocket expense as a direct result of osteoarthritis was approximately $2,600 per person per year with a total annual disease cost of $5,700.[13, 14] Job-related osteoarthritis costs were estimated to be between

$3.4 and $13.2 billion per year. Other studies reported average annual direct medical, drug, and indirect work loss costs were $8,601, $2,941, and $4,603, respectively.[15] Logically, the primary goal going forward for the health care field regarding osteoarthritis would be to utilize the most effective treatments available that are also the most cost-effective.

ETIOLOGY

There are many causes of joint injury reported in the literature as well as associated risk factors which increase the likelihood of joint degeneration. It may be caused by a systemic (genetic) predisposition or by local (mechanical) factors. For some the cause is known (secondary), but for others the cause is unknown (primary). For example, a person may have an inherited predisposition to develop the disease, but it may only materialize when a biomechanical insult (such as a knee injury) has occurred.[16] It should be emphasized at the outset that osteoarthritis is primarily a degenerative process, not an inflammatory one as the name implies. A more appropriate term would be osteoarthrosis or degenerative joint disease.

Ligament damage or weakness is one cause of joint degeneration. Joint subluxations, dysplasia, and incongruity prevent the normal distribution of weight and stresses on the articular surfaces of the joint leading to cartilage injury and joint degeneration. The disruption of ligaments and joint capsules, causing increased joint laxity, increases the risk of articular cartilage injury because the joint motion is no longer stabilized by the ligament structure.[10] These mechanical abnormalities cause changes in the areas of contact on opposing surfaces and increase the intensity of impact loading and shear and compression forces on some regions of cartilage. (*See Figure 3.*) The mechanical properties of articular cartilage depend on the macromolecular framework consisting of collagens and aggregating proteoglycans and the water within the macromolecular framework. The collagens give the tissue its strength, while the interaction of the proteoglycans with water gives the tissue its stiffness (resistance) to compression, resilience, and durability.[18, 19] The cartilage is the thickest in areas where contact pressure is greatest. After a ligament injury, joint motion becomes greater and may offset the contact surface to regions where the cartilage may be thinner and less able to support the applied stresses.[17] The loss of sensory innervations of the joint and surrounding muscles also increases the susceptibility of joint degeneration because of an increase in the instability of the joint.[18] When the load is applied slowly, the muscles are able to contract and absorb much of the energy and stabilize the joint. However, if the load is sudden, the muscles do not have time to respond to stabilize the joint and decrease the forces applied to the cartilage surfaces. Even normal levels of joint use may cause articular surface injury and degeneration in unstable, subluxed, or malaligned joints and in joints that do not have normal innervation.[20] Genetic hypermobility such as Ehlers-Danlos Syndrome and non-genetic hypermobility (Benign Hypermobility Syndrome) where trauma or injury is absent increase the likelihood of OA development. Further prospective studies are needed to study the effects of non-traumatic hypermobility as it relates to OA.

Direct trauma is a second cause of joint degeneration and is typically associated with athletic participation. The articular surface can be damaged by single or repetitive impact from a direct blow to the joint or bones that form

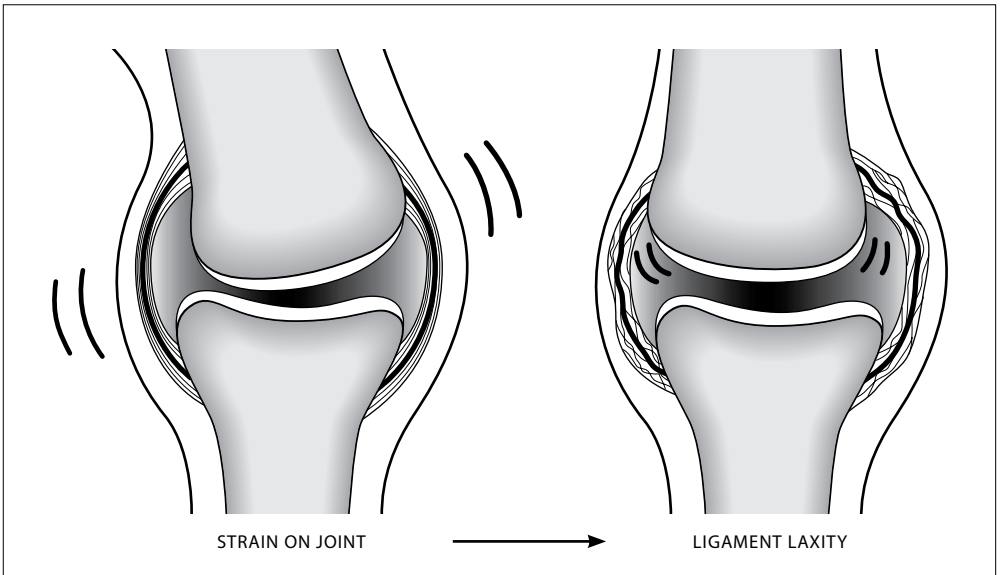

Figure 3. Ligament laxity can cause instability of the joint. The result is stretched ligaments and misaligned joints.

the joint. It can also be damaged by torsional loading resulting from twisting or turning of joint surfaces that are relative to each other. The rate of loading also affects the type of damage that may be caused by sudden impact axial compression or torsional strain. During slow impact loading, the movement of fluid within the cartilage allows it to deform and decrease the forces applied to the matrix macromolecular framework. In sudden or high impact loading, the matrix macromolecular framework suffers a greater level of stress because the loading occurs too fast to allow for adequate fluid movement and tissue deformation.[20] One study performed a 36 year follow-up of 141 participants that had sustained a hip or knee injury after 22 years of age and found that, due to the deleterious effects of trauma that had compromised the structural integrity of the joint, 96 (68%) of the participants had developed osteoarthritis in the injured joint.[21] Another study showed that 80% of American football players with a history of knee injury showed signs of osteoarthritis 10 to 30 years after retiring.[22] Soccer players also have an increased incidence rate of osteoarthritis in the lower extremity joints, mainly the knee, when compared to a control group of the same age. The most common types of injuries are sprains and strains, which are usually caused by excessive forces applied to a joint in an abnormal direction. This leads to a high number of meniscal and ligamentous injuries that ultimately translate to an increased instability within the joint.[23, 24] While direct trauma or compression to the cartilage surfaces can alone can cause OA over time, it is unquestionably the concomitant ligament injury in the majority of these cases which sets the joint up for OA development. When cartilage wear and degradation outpace cartilage repair, the wheels are set in motion for joint degeneration.

A third cause of joint degeneration is overuse. This can be seen in jobs involving manual labor with repetitive motions such as farming, construction work, and lifting heavy loads. Heavy manual labor and stresses in the work environment were major predictors in development of hip osteoarthritis.[25] Hip osteoarthritis was diagnosed in 41 subjects (4.9%) after a 22-year follow-up study of 840 participants. Baseball players also have an increased risk of developing osteoarthritis in their shoulders and elbows due to the repetitive motion of pitching and throwing.[26, 27] The average Major League Baseball pitcher throws over 3,000 pitches per season with little rest between games. Excess joint loading forces at the extremes of motion repeated many times over contribute to joint and connective tissue wear and degeneration. (*See Figure 4.*) A biomechanically sound shoulder and elbow joint, strong and well-conditioned muscles, excellent pitching technique and mechanics, and adequate rest afford the athlete the best case scenario for avoiding overuse injuries leading to degeneration. When all of these things are in place and injury still occurs, could it be that subtle, unrecognized ligament deficiency is responsible for overuse injuries?

Other risk factors for joint degeneration are above-average body weight, supported by the fact that for every 1 pound increase in weight, the overall force across the knee in a single-leg stance increases 2-3 pounds.[16, 18] Failure to accurately realign fractures, leaving room for abnormal movement and deviation;[28] car accidents, which subject the body to sudden impacts may cause injury to ligaments and muscles and lead to pain and weakness in the spine and extremities; poor posture, age, abnormal joint anatomy or alignment,[18] associated diseases, and genetics are other considerations leading to OA. Genetic factors account for 50% of cases of osteoarthritis in the hand and hip and a smaller percentage in the knees.[16]

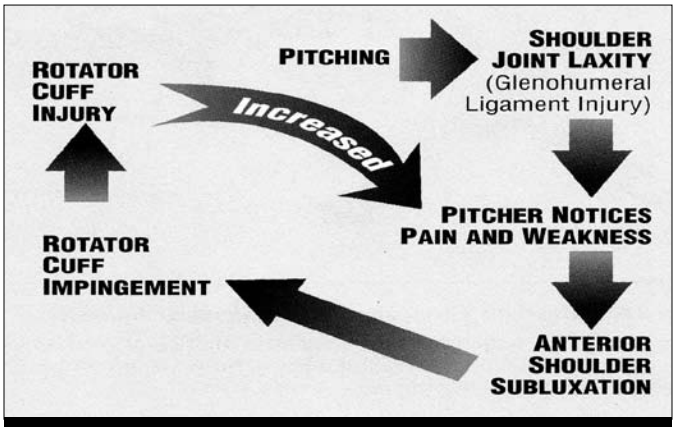

Figure 4. The pitcher's nightmare. Most pitchers experience this sequence of events to some degree. Shoulder joint laxity is the underlying etiology of the pitcher's shoulder pain.

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Common Locations for Osteoarthritis

THE KNEE

Knee joints are particularly susceptible to direct trauma and ligament injury because they are located between the two longest lever arms in the body, the tibia and

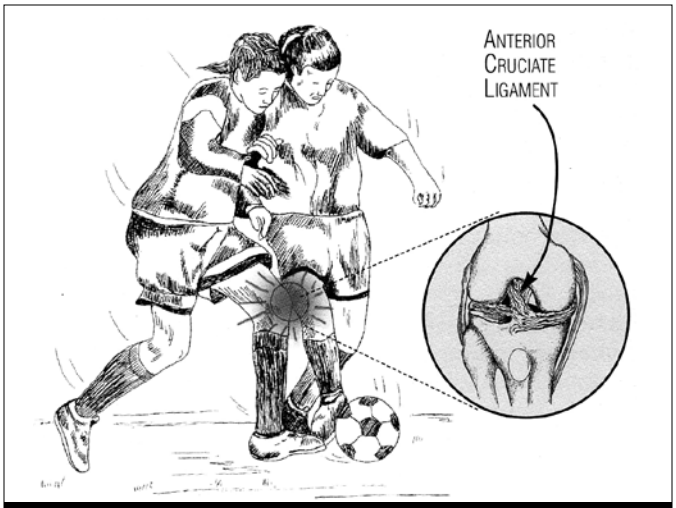

Figure 5. Mechanism of anterior cruciate ligament injury in agility sports. When trying to pivot around an opponent, an athlete decelerates and pivots on a planted foot, causing the ACL injury.

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femur, and they experience high repetitive impact loads.[29] (*See Figure 5.*) Because of their inherent vulnerability in different planes and joint angles, they are more likely to develop osteoarthritis after injury.

Meniscal tears, which are the result of traumatic impact or torsional loading, are a cause of osteoarthritis. Meniscal tears are believed to cause osteoarthritis because of decreased joint stability and the alteration of biomechanical forces. The primary function of the meniscus is to distribute the forces evenly across the knee joint. When significant tears of the meniscus occur or when meniscal tissue is removed with surgery, the contact forces increase over a smaller area of the cartilage leading to cartilage loss which is accelerated further by an acquired varus or valgus deformity. Research has shown that 13% to 43% of subjects that had meniscal damage and/or underwent a partial meniscectomy developed clinical symptoms associated with osteoarthritis.[30-32] An injury to the meniscus during middle-age, defined as a horizontal tear, is associated with degeneration and is likely a result of an already existent osteoarthritic process in the knee.[33, 34]

Osteoarthritis also has a high rate of incidence in both male and female soccer players who had a torn anterior cruciate ligament (ACL). One study found that 82% of female soccer players had radiographic changes in their knees 12 years after tearing their ACL, and 51% of those individuals met the criteria for radiographic knee osteoarthritis.[35] Another study found that 78% of male soccer players had radiographic changes in their injured knees 14 years after a torn ACL, and 41% of those individuals had more advanced changes.[36] Other studies report ranges from 12% up to 50 to 60% of patients 5 years post-ACL reconstruction displaying signs of osteoarthritis.[37] Instability of the joint caused by ACL tears also increases the chances of the development of osteoarthritis due to changes in the molecular structures. Cartilage and synovial fluid samples obtained post-ACL injury revealed a rapid onset of damage to type II collagen and an initial increase in proteoglycan content associated with osteoarthritis.[38] After ACL reconstruction, stability may be restored in one plane of motion, but it may not fix it in all other planes of motion because of graft structure, intra-articular graft placement, and initial graft tension.[39, 40] The development of osteoarthritis following ACL tear has not been clearly determined, but those with chronic ACL deficiency are at a significantly higher risk of secondary meniscal damage.[37] The combination of meniscal injuries at the time of ACL injury is most frequently associated with knee osteoarthritis.[41]

Other factors that play a role in the development of osteoarthritis in the knee are medial joint laxity, higher BMI (Body Mass Index) values, lesser quadriceps femoral strength, lesser knee flexion, greater knee adduction, and greater co-contraction of the quadriceps femoris and gastrocnemius muscles.[42, 43]

THE HIP

The hip joint is inherently more stable than the knee joint due to its ball-and-socket configuration and surrounding musculature. High load-bearing with or without joint trauma is the primary association with hip osteoarthritis. It is commonly associated with heavy manual labor and major musculoskeletal injuries. A 22-year follow-up study of adult Finns diagnosed 4.9% of subjects with hip osteoarthritis after working jobs that involved heavy manual labor. Men with high exposure to heavy lifting were at a higher risk of developing hip osteoarthritis and the risk increased as the weight of the loads increased. Also, a higher risk was associated with lifting heavier loads before the age of 30. Occupations of farming and construction work showed increased incidence rates of hip osteoarthritis due to superolateral migration of the femoral head.[25, 44-46] Similar results were also found in women who experienced high levels of physical work in their occupation and at home. Increased risk factors

include frequent stair climbing, physically demanding tasks outside of their occupation, and high-intensity sports activity.[47] Female physical education teachers had a higher prevalence of osteoarthritis in the hip when compared to a similar-aged control group.[48] Damage as a consequence of musculoskeletal injuries also was an independent predictor for the development of hip osteoarthritis.[25] Specific risks include high loads, sudden or irregular impact, preexisting abnormalities such as hip dysplasia, and labral tears.

Athletes are prone to hip injuries and later development of OA. Professional soccer players have a 10-fold risk of developing hip osteoarthritis compared to that of the normal population, even with the lack of an injury.[49] Similar findings emerged among former National Football League (NFL) players with 55.6% reporting arthritic problems in an NFL Players Association Survey in 2001.[50, 51] Repetitive low-grade impact from sport-related stresses can be enough to damage the soft tissue and surrounding ligament structure, weakening the joint, and starting the arthritic process.[52]

THE SHOULDER

By virtue of its shallow socket (glenoid) and great range of motion, the shoulder is very susceptible to connective tissue injury and instability leading to osteoarthritis. Osteoarthritis seen in the shoulder and elbow can be traced back to direct trauma or repetitive usage. Multiple studies have shown that repetitive high-stress activities involving the throwing arm in youth baseball players have led to the development of osteochondritis of the head of the radius and the capitulum of the humerus. Because of the presence of loose bodies floating in the joint, pain and eventual development of osteoarthritis can occur.[26, 50, 53] Recurrent dislocations, especially anteriorly, can also cause development of instability and osteoarthritis in the shoulder.

The development of glenohumeral osteoarthritis occurs at a point of maximum joint-reaction force where the humeral head meets the glenoid and when the arm is abducted 90 degrees. This wear and tear causes the glenoid to become flattened and eroded posteriorly and may increase the likelihood of posterior subluxation. The combination of years of dislocations and surgery tighten the joint capsule and produce fixed subluxations in the opposite direction of the dislocations, resulting in severe cases of degenerative arthritis.[54] Anterior instability has also been associated with the development of osteoarthritis. One study found shoulder osteoarthritis in the radiographs of 11.3% of subjects and CT scans revealed arthritic changes in 31.2%.[55] The number and frequency of dislocations and/or subluxations were significantly higher in the osteoarthritic joints when compared to the non-osteoarthritic joints. Rheumatoid arthritis, rotator cuff tears, and Lyme disease also increase the chances for development of osteoarthritis in the glenohumeral joint.[56]

THE ANKLE

The most common injury to the ankle is the ligamentous lesion to the lateral ligament complex as a result of an inversion ankle sprain. Ankle sprains have been shown to occur more frequently in individuals with clinical instability and are more common in those with previous ankle sprains.[57] Between 10% and 30% of patients that experience inversion sprains experience chronic ankle instability.[58, 59] One study from 1979 reported osteoarthritis in 78% of subjects associated with ankle instability after 10 years, but other research has shown that osteoarthritis does not result until 26 years after a single severe sprain and 38 years in recurrent ankle sprains.[60, 61] Post-traumatic osteoarthritis is the cause of more than 70% of the arthritis cases in the ankles.[60] The incidence rates of osteoarthritis in recent years have increased, in part due to an increase in sports injuries.

Subtalar instability is believed to be one cause for chronic functional instability in the foot and ankle. One study reported that damage to the bifurcate ligament results in a significant increase in both plantarflexion and dorsiflexion, while injury to the inferior extensor retinaculum resulted in a significant increase in inversion and eversion. Also, dissection of the calcaneofibular ligament increased the degree of internal and external rotation and also produced significant kinematic changes in all degrees of motion in the subtalar joint.[62] Other contributing factors that result in the development of osteoarthritis in the ankle are malleolar fractures, tibial pilon fractures, talus fractures, and distal tibial fractures.[60] Poor ankle biomechanics also increase the likelihood of the development of osteoarthritis. There is a strong association of OA with abnormal pronation and external rotation during heel-strike, as well as abnormal supination and internal rotation during the acceleration phase during the gait cycle.[63]

The connection between ankle ligament injury and instability with osteoarthritis is clear from these studies and, as with other joints, the incidence of OA is expected to increase with the aging of a more active population.

THE WRIST AND HAND

Osteoarthritis of the wrist is associated with traumatic injuries and is frequently seen in the athletic population. Scapholunate interosseous ligament injury is the most common form of carpal instability and is caused by excessive wrist extension and ulnar deviation in collision and contact sports.[64, 65] Without a proficient scaphoid ligament, the scaphoid falls into a flexed position that alters the articular contact areas and stress patterns within the wrist.

Osteoarthritis can also develop in a scaphoid non-union with advanced collapse because the "hump-back" deformity that results over time causes changes in the kinematic patterns that result in dorsal instability.[50] Distal radial fractures also have been linked to the development of osteoarthritis, especially in the younger populations. Failure to properly realign distal radial fractures caused 65% to 68% of subjects to develop post-traumatic osteoarthritis in 7 to 34 years following injury due to increased instability and weakness within the joint.[28, 50, 66] There was also an observed relationship between the narrowing of the joint space and extra-articular malunion. The reported number of cases of OA increases significantly when the displacement of intra-articular fractures are greater than two millimeters.[67]

Osteoarthritis is also very common in the joints of the hands, predominately the first carpometacarpal (CMC) joint and the distal interphalangeal (DIP) joints. Though these are not weight-bearing joints, the first CMC joint, in particular, is very mobile and therefore subject to cartilage breakdown from overuse or excessive forces. It is less clear whether hypermobility apart from injury is responsible for OA of the DIP joints where multiple and bilateral involvement is the norm. This would likely focus more attention to a genetic or heritable source for OA of the hands.

THE NECK AND LOW BACK

Osteoarthritis can also be found in the cervical spine and lumbar spine, which have both synovial and non-synovial elements. Causes are multifactorial and, like the appendicular joints, the axial joints possess many pain generators, including the disc annulus, the periosteum, the dura, muscles, tendons, ligaments, capsules, and the nerves when compressed or stretched. The eventual development of OA in the form of degenerative disc disease (DDD), degenerative facet joint disease (DJD), or spinal stenosis is the end-stage of these unresolved pain generators.

Osteoarthritis of the spine tends to first appear during the third decade of life and can be related to the general aging process or related to a person's type of work. Gender can also affect incidence rates of osteoarthritis with a higher prevalence in post-menopausal women, an indication that hormones play a role. Excessive weight also increases the likelihood of development of the disease because of the increased stress the joints must support in the lumbar spine. Excessive abdominal weight is almost entirely a biomechanical problem since the lordotic configuration of the lumbar spine is further taxed by an anterior shift in the center of gravity. The cycle of a sedentary lifestyle and weakened abdominal and spinal muscles, causes further strain on the spine, discs, and facet joint capsules. The ligament component of spinal stability is related to the support, health, and proper function of these tissues and often overlooked as a major, if not the major, source of back pain and ultimate degeneration. The case can be made that excess use or even complete dependence on the MRI has focused too much attention on the intervertebral disc and the vertebrae themselves to the exclusion of the ligaments and facet joint capsules. Ligaments do not often show themselves on MRI to be damaged in the way a disc would and, therefore, the history and physical examination are of ultimate importance to determine the presence of pain, injury, and dysfunction involving these connective tissue structures. (*See Figure 6.*)

Possible Signs and Symptoms of Ligament Injury:

- Balance Difficulties
- Decreased Joint Motion
- Dizziness
- Joint Cracking
- Joint Instability
- Muscle Spasm
- Numbness
- Pain
- Swelling
- Vertebral Subluxations
- Weakness

Figure 6. Ligament injury can produce diverse symptomatogy.

The iliolumbar ligament is the ligament of primary importance in the lumbar spine. It is the major stabilizing component between the vertebral spine and the pelvis. However, it is also the weakest of the three stabilizing ligaments and without an intact iliolumbar ligament there would be decreased stability of the vertebral column in relation to the pelvis and excess motion of both the sacrum and the vertebral column. Also, due to its attachment angle, this ligament has an increased susceptibility to injury, especially during flexion and lateral bending. Repetitive microtrauma to the iliolumbar ligament, due to poor posture, obesity or faulty physical mechanics, can push it past its physiologic limits and induce low back pain.[68] According to George Hackett, M.D., ligamentous laxity is caused by acute and/or repetitive trauma and this laxity puts tension on the intrinsic nerve fibers, causing pain.[69]

Repetitive strains from accidents, surgery, poor posture, and injuries increase the risk of development of osteoarthritis of the spine. Genetics, such as family history of osteoarthritis and congenital defects of joints and the spine, as well as leg abnormalities, can also play a role its development. Spinal osteoarthritis occurs between the facet joints in the posterior spinal column, as it does in any other synovial joints in the body, and often leads to mechanically-induced pain because of inflammation and induced frictional pain.[70] One study researched the prevalence of facet joint osteoarthritis in conjunction with lower back pain across age groups. The highest reported cases of osteoarthritis were reported in the 60-69 year old age group with 88.9% of males and 89.5% of females with reported lower back pain also showing signs of osteoarthritis on CT scans.[71] (*See Figure 7.*) The L4-L5 spinal level had significantly higher levels of osteoarthritis and is commonly associated with degenerative spondylolisthesis.[72] This may be due to increased stresses and forces which the low back is subjected to when lifting objects.[52] A gender difference was discovered in the Kalichman study, showing a significant difference in the prevalence of facet joint osteoarthritis between males and females at the L4-L5 level. Women had a higher prevalence and were found to be at a higher risk for the development of osteoarthritis in the spine, hands, and knees because cartilage is a sex-hormone-sensitive tissue.[73] The L5-S1 level is also vulnerable to facet degeneration due to its location at the base of the spinal column and greater angulation. This is also the reason for a greater incidence of degenerative disc disease at the L4-L5 and L5-S1 levels.

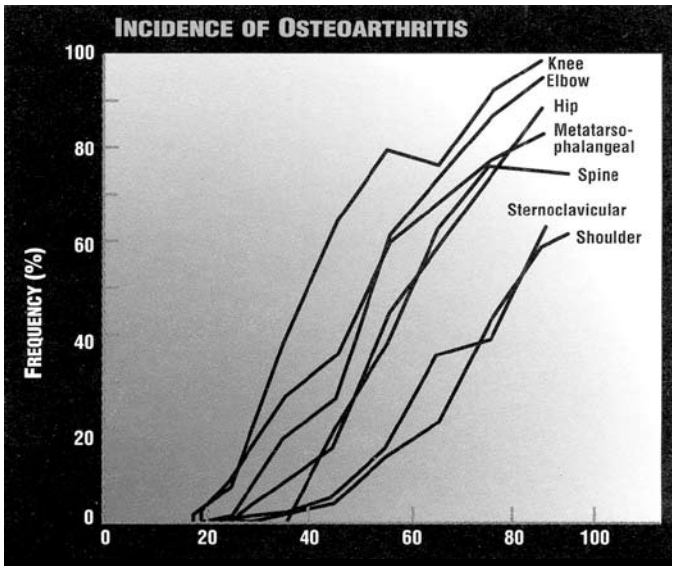

Figure 7. Incidence of osteoarthritis. Note the frequency of osteoarthritis involvement found on post-mortem examination in people of different ages. The underlying cause is typically ligament injury leading to excessive joint mobility.

Adapted from Osteoarthritis: Is it an Arrestable or Reversable Disease? By John H. Bland. Advances in Inflammation Research, Vol. 11, I.O. Merness, Raven Press, NY, ©1986, pp. 177-187.

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A consequence of spinal instability is the growth of bone spurs (osteophytes) at the entheses. Bone spurs are seen by some as part of the normal aging process and may not cause pain, but without question, instability is the most common etiology for spurs. These growths of bone are best thought of as traction spurs whereby repeated traction at ligament insertions result in microscopic tearing and bleeding. They can appear on the facet joints and on the spinal vertebrae and are the body's attempt to re-stabilize the joint. With continued growth they can cause irritation and even entrapment of nerves passing through the spinal structure due to foraminal narrowing.[70]

The cervical spine is also at risk for the development of osteoarthritis from various mechanisms of injury, including whiplash, fractures, dislocations, sprains and strains, repetitive stress, poor posture, all of which threaten the stability of the cervical spine and its neural contents. (*See Figure 8.*) The causes are similar to injuries of the lumbar spine but vary in degree in that lifting injuries and obesity, for example, are less common causes in the neck than the low back while motor vehicle accidents (whiplash) causes more neck injuries. With over 5.5 million car crashes in the United States every year, it is no surprise the most common mechanism of injury is whiplash.

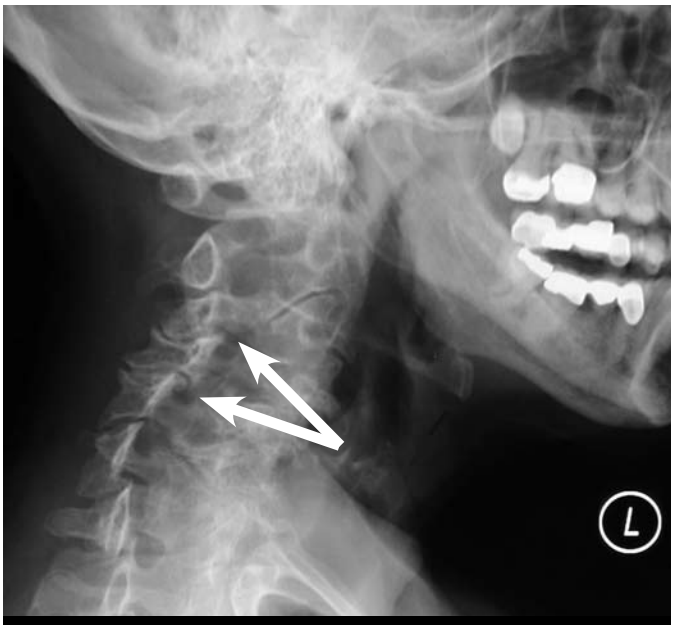

Figure 8. X-ray of the neck. This X-ray shows excessive degeneration causing neural foramina encrouchment.

By using high-speed technology, it was discovered that the cervical spine undergoes a sigmoid deformation as it is compressed by the rising trunk, with the lower segments undergoing extension while the upper segments flex around an abnormally located axis of rotation. There is also an observed anterior rotation to the upper elements of the cervical spine and a posterior rotation to its lower elements. Instead of the articular processes gliding by one another, the inferior processes chisel into the superior articular processes of the supporting vertebra.[74] This pattern of movement may lead to impaction fractures of the articular cartilage or articular processes, intervertebral discs may be torn or avulsed, and soft-tissue injuries may occur due to the abnormal separation of the vertebrae of the cervical spine, causing uneven forces to be applied to the surrounding joints. Also, altered joint mechanics and collagen fiber disorganization of and around the cervical facet joint capsule may imply ligament damage that has the potential to alter nerve fiber signaling and produce strained physiologic modifications, leading to pain and the development of osteoarthritis.[75]

CONCLUSION

The relationship of ligament injury and osteoarthritis is a convincing one. When there is insufficient ligament support to stabilize joint motion, the resultant increase in joint laxity leads to the development and acceleration of articular cartilage injury. The biomechanical abnormalities caused by joint instability greatly increase impact loading via increased shear and compression forces across areas of contact on opposing cartilage surfaces. Even with early recognition of ligament injury and deficiency, traditional medical interventions do not treat the etiology of the disease. It is for this reason that the prevalence of osteoarthritis will increase as will the number of joint replacements. ■

BIBLIOGRAPHY

1. McKinley M, et al. *Human Anatomy*. McGraw-Hill, Debuque, Iowa. 2006.
2. Zakhary B, et al. Joint Trauma and Osteoarthritis. *Arthritis MD*, retrieved from: http://www.arthritismd.com/joint-trauma_and_osteoarthritis.html. 2005.
3. Lawrence R, et al. Estimates of the prevalence of selected arthritis and musculoskeletal diseases in the United States. *Arthritis and Rheumatism*. 1998;41(5):778-799.
4. Lawrence R, et al. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States, part II. *Arthritis and Rheumatism*. 2008;58(1):26-35.
5. Lawrence R, et al. Estimates of the prevalence of selected arthritis and musculoskeletal diseases in the United States. *Journal of Rheumatology*. 1989;16(4):427-441.
6. Felson D, et al. Osteoarthritis: new insights. Part 1: the disease and its risk factors. *Annals of Internal Medicine*. 2000;133(8):635-646.
7. Petersson I, et al. Radiographic osteoarthritis of the knee classified by the Ahlback and Kellgren & Lawrence systems for the tibiofemoral joint in people aged 35-54 years with chronic knee pain. *Annals of the Rheumatic Diseases*. 1997;56(8):493-496.
8. Felson D, et al. The incidence and natural history of knee osteoarthritis in the elderly. The Framingham Osteoarthritis Study. *Arthritis and Rheumatism*. 1995;38(10):1500-1505.
9. HCUP Facts and Figures, 2006: AHRQ analysis of arthritis hospitalizations, retrieved from *Hospitalizations for Osteoarthritis Rising Sharply, USA*, Medical News Today, September 5, 2008, http://www.medicalnewstoday.com/article/120308.php.
10. Brody JE. Personal Health; A New Set of Knees Comes at a Price: A Whole Lot of Pain. *New York Times*. 2008. published February 8, 2005.
11. Revolution Health Group: How to cover – and Cut – the rising cost of osteoarthritis care, Updated July 17, 2007.
12. Lethbridge-Cejku M, et al. Hospitalizations for arthritis and other rheumatic conditions: data from the 1997 National Hospital Discharge Survey. *Medical Care*. 2003;41(12):1367-1373.
13. Gabriel SE. Direct medical costs unique to people with arthritis. *Journal of Rheumatology*. 1997;24(4):719-725.
14. Maetzel A, et al. Community Hypertension and Arthritis Project Study Team. The economic burden associated with osteoarthritis, rheumatoid arthritis, and hypertension: a comparative study. *Annals of Rheumatic Diseases*. 2004;63(4):395-401.
15. White AG, et al. Direct and indirect costs of pain therapy for osteoarthritis in an insured population in the United States. 2008.

16. Felson DT, et al. Osteoarthritis: new insights. Part 1: The disease and its risk factors. *Annals of Internal Medicine*. 2000;133 (8), 635-46.

17. Andriacchi TP, et al. A framework for the in vivo pathomechanics of osteoarthritis at the knee. *Annals of Biomedical Engineering*. 2004;32(3):447-457.

18. Buckwalter JA, et al. Athletics and osteoarthritis. *The American Journal of Sports Medicine*. 1997;25(6):873-881.

19. Buckwalter JA, et al. Joint injury, repair, and remodeling: roles in post-traumatic osteoarthritis. *Clinical Orthopaedics and Related Research*. 2004;(423):7-16.

20. Buckwalter JA. Sports, joint injury, and posttraumatic osteoarthritis. *The Journal of Orthopaedic and Sports Physical Therapy*. 2003;33(10):578-588.

21. Gelber AC, et al. Joint injury in young adults and risk for subsequent knee and hip osteoarthritis. *Annals of Internal Medicine*. 2000;133(5):321-328.

22. Rall KL, et al. A study of long term effects of football injury to the knee. *Missouri Medicine*. 1984;Jun;61:435-438.

23. Kujala UM, et al. Osteoarthritis of weight bearing joints of lower limbs in former elite male athletes. *BMJ (Clinical Research Ed.)*. 1994;308(6923):231-234.

24. Kujala UM, et al. Knee osteoarthritis in former runners, soccer players, weight lifters, and shooters. *Arthritis and Rheumatism*. 1995;38(4):539-546.

25. Juhakoski R, et al. Risk factors for the development of hip osteoarthritis: a population-based prospective study. *Rheumatology (Oxford, England)*. 2009;48(1):83-87.

26. Adams JE. Injury to the throwing arm. A study of traumatic changes in the elbow joints of boy baseball players. *California Medicine*. 1965;102:127-132.

27. Bennett GE. Shoulder and elbow lesions of professional baseball pitcher. *JAMA*. 1941;117: 510-514.

28. Forward DP, et al. Do young patients with malunited fractures of the distal radius inevitable develop symptomatic post-traumatic osteoarthritis? *The Journal of Bone and Joint Surgery, British Volume*. 2008;90(5):629-637.

29. Fleming BC, et al. Ligament injury, reconstruction, and osteoarthritis. *Current Opinion in Orthopaedics*. 2005;16(5):354-362.

30. Hart AJ, et al. Assessment of osteoarthritis after reconstruction of the anterior cruciate ligament: a study using single-photon emission computed tomography at ten years. *The Journal of Bone and Joint Surgery, British Volume*. 2005;87(11):1483-1487.

31. Liden M, et al. Osteoarthritic changes after anterior cruciate ligament reconstruction using bone-patellar tendon-bone or hamstring tendon autografts: a retrospective, 7 year radiographic and clinical follow-up study. *Arthroscopy*. 2008;24(8):899-908.

32. Neuman P, et al. Prevalence of tibiofemoral osteoarthritis 15 years after non-operative treatment of anterior cruciate ligament injury: a prospective cohort study. *The American Journal of Sports Medicine*. 2008;36(9):1717-1725.

33. Roos EM. Joint injury causes knee osteoarthritis in young adults. *Current Opinion in Rheumatology*. 2005;17(2):195-200.

34. Englund M. Meniscal tear – a feature of osteoarthritis. *Acta Orthopaedica Scandinavia Supplementum*. 2004;75(312):1-45.

35. Lohmander LS, et al. High prevalence of knee osteoarthritis, pain, and functional limitations in female soccer players twelve years after anterior cruciate ligament injury. *Arthritis and Rheumatism*. 2004;50(10):3145-3152.

36. Von Porat A, et al. High prevalence of osteoarthritis 14 years after an anterior cruciate ligament tear in male soccer players: a study of radiographic and patient relevant outcomes. *Arthritis and Rheumatism*. 2004;63(3):269-273.

37. Nebelung W, et al. Thirty-five years of follow-up of anterior cruciate ligament-deficient knees in high-level athletes. Arthroscopy: *The Journal of Arthroscopic and Related Surgery*. 2005;21 (6); 696-702.

38. Nelson F, et al. Early post-traumatic osteoarthritis-like changes in human articular cartilage following rupture of the anterior cruciate ligament. *Osteoarthritis and Cartilage/OARS, Osteoarthritis Research Society*. 2006;14(2):114-119.

39. Jonsson H, et al. Positive pivot shift after ACL reconstruction predicts later osteoarthrosis: 63 patients followed 5-9 years after surgery. *Acta Orthopaedic Scandinavica*. 2004;75(5):594-599.

40. Logan MC, et al. Tibiofemoral kinematics following successful anterior cruciate ligament reconstruction using dynamic multiple resonance imaging. *American Journal of Sports Medicine*. 2004;32(4):984-992.

41. Øiestad BE, et al. Knee osteoarthritis after anterior cruciate ligament injury. *The American Journal of Sports Medicine*. 2009;37(7), 1434-43.

42. Rudolph KS, et al. Age-related changes in strength, joint laxity, and walking patterns: are they related to knee osteoarthritis? *Physical Therapy*. 2007;87(11):1422-1432.

43. Schmitt LC, et al. Instability, Laxity, and physical Function in patients with medial knee osteoarthritis. *Physical Therapy*. 2008;88(12):1506-1516.

44. Bierma-Zeinstra SM, et al. Risk factors and prognostic factors of hip and knee osteoarthritis. *Nature Clinical Practice, Rheumatology*. 2007;3(2):78-85.

45. Hoaglund FT, et al. Primary osteoarthritis of the hip: etiology and epidemiology. *The Journal of the American Academy of Orthopaedic Surgeons*. 2001;9(5):320-327.

46. Jensen LK. Hip osteoarthritis: influence of work with heavy lifting, climbing stairs or ladders, or combining kneeling/ squatting with heavy lifting. *Occupational and Environmental Medicine*. 2008;61(1):6-19.

47. Vingard E, et al. Osteoarthritis of the hip in women and its relation to physical load at work and in the home. *Annals of the Rheumatic Diseases*. 1997;56(5):293-298.

48. White JA, et al. Relationships between habitual physical activity and osteaoarthrosis in aging women. *Public Health*. 1993;107(6):459-470.

49. Shepard GJ, et al. Ex-professional association footballers have an increased prevalence of osteoarthritis of the hip compared with age matched controls despite not having sustained notable hip injuries. *British Journal of Sports Medicine*. 2003;37(1):80-81.

50. Koh J, et al. Osteoarthritis in other joints (hip, elbow, foot, ankle, toes, wrist) after sports injuries. *Clinics in Sports Medicine*. 2005;24(1):57-70.

51. Callahan LF, et al. Osteoarthritis in retired National Football League players: The role of injuries and playing position. *Arthritis and Rheumatism*. 2002;(46):S415.
52. Hauser R, et al. *Prolo Your Sports Injuries Away*. Oak Park, IL: Beulah Land Press. 2001.
53. Stubbs MJ, et al. 3rd. Osteochondritis dissecans of the elbow. *Clinics in Sports Medicine*. 2001;20(1):1-9.
54. Neer CS, et al. Recent Experience in Total Shoulder Replacement. *Journal of Bone and Joint Surgery*. 1982;64 (3); 319-337.
55. Ogawa K, et al. Osteoarthritis in shoulders with traumatic anterior instability: preoperative survey using radiography and computed tomography. *Journal of Shoulder and Elbow Surgery*. 2006;15(1):23-29.
56. Woodward TW, et al. The painful shoulder: part II. Acute and chronic disorders. *American Family Physician*. 2000;61(11)3291-3300.
57. Dvorak J, et al. Football injuries and physical symptoms. *The American Journal of Sports Medicine*. 2000;28,S-3-S-9.
58. Hintermann B, et al. Medial ankle instability: an exploratory, prospective study of fifty-two cases. *American Journal of Sports Medicine*. 2004;32(1):183-190.
59. Karlsson J, et al. Chronic lateral instability of the ankle in athletes. *Sports Medicine (Aukland, N.Z.)*. 1993;16(5):355-365.
60. Valderrabano V, et al. Ligamentous posttraumatic ankle osteoarthritis. *The American Journal of Sports Medicine*. 2006;34(4):612-620.
61. Harrington KD. Degenerative arthritis of the ankle secondary to long-standing lateral ligament instability. *The Journal of Bone and Joint Surgery, American Volume*. 1979;61(3)354-361.
62. Weindel S, et al. Subtalar instability: a biomechanical cadaver study. *Archives of Orthopedic and Traumatic Surgery*. 2008;October 7, 2008 [Epub ahead of print].
63. Hashimoto T, et al. A kinematic study of ankle joint instability due to rupture of the lateral ligaments. *Foot and Ankle International*. 1997;18(11):729-734.
64. Jones WA. Beware the sprained wrist. The incidence and diagnosis of scapholunate instability. *The Journal of Bone and Joint Surgery, British Volume*. 1988;70(2):293-297.
65. Rettig AC. Epidemiology of hand and wrist injuries in sports. *Clinics in Sports Medicine*. 1998;17(3):401-406.
66. Knirk JL, et al. Intraarticular fractures of the distal end of the radius in young adults. *The Journal of Bone and Joint Surgery, American Volume*. 1986;68 (5):647-59.
67. Bradway JK, et al. Open reduction and internal fixation of displaced, comminuted intra-articular fractures of the distal radius. *The Journal of Bone and Joint Surgery*. 1989;71A:839-47.
68. Sims JA, et al. The role of the iliolumbar ligament in low back pain. *Medical Hypotheses*. 1996;46:511-15.
69. Hackett G. *Ligament and Tendon Relaxation Treated by Prolotherapy*, Third Edition. Springfield, IL: Charles S. Thomas. 1958.
70. Ray CD. Understanding Osteoarthritis of the Spine. *Spine Health: Trusted Information for Pain Relief*, retrieved from: http://www.spine-health.com/conditions/arthritis/understanding-osteoarthritis-spine. 2005.
71. Kalichman L, et al. Facet joint osteoarthritis and low back pain in the community-based population. *Spine*. 2008;33(23):2560-2565.
72. Vogt MT, et. al. Lumbar olisthesis and lower back symptoms in elderly white women. The Study of Osteoporotic Fractures. *Spine*. 1998;23(23):2640-2647.
73. Rosner IA, et al. Estrogens and osteoarthritis. *Clinical Orthopaedics and Related Research*. 1986;Dec:(213):77-83.
74. Bogduk N, et al. Biomechanics of the cervical spine part 3: minor injuries. *Clinical Biomechanics*. 2001;16:267-75.
75. Quinn KP, et al. Structural changes in the cervical facet capsular ligament: potential contributions to pain following subfailure loading. *Stapp Car Crash Journal*. 2007;51:169-87.