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REVIEW

Exercise and osteoarthritis

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Abstract

Exercise remains an extremely popular leisure time activity in many countries throughout the western world. It is widely promoted in the lay press as having salutory benefits for weight control, disease management advantages for cardiovascular disease and diabetes, in addition to improving psychological well-being amongst an array of other benefits. In contrast, however, the lay press and community perception is also that exercise is potentially deleterious to one's joints. The purpose of this review is to consider what osteoarthritis (OA) is and provide an overview of the epidemiology of OA focusing on validated risk factors for its development. In particular the role of both exercise and occupational activity in OA will be described as well as the role of exercise to the joints' tissues (particularly cartilage) and the role of exercise in disease management. Despite the common misconception that exercise is deleterious to one's joints, in the absence of joint injury there is no evidence to support this notion. Rather it would appear that exercise has positive salutory benefits for joint tissues in addition to its other health benefits.
Key words diarthrodial joint; exercise; osteoarthritis.

Introduction

Exercise remains an extremely popular leisure-time activity in many countries throughout the Western world and has for many become part of the modern lifestyle. It is widely promoted in the lay press as having salutory benefits for weight control, disease management advantages for cardiovascular disease and diabetes, and for improving psychological well being amongst an array of other benefits. In contrast, however, the lay press and community perception is also that exercise is potentially deleterious to one's joints, in particular those of the lower extremities. A previous review in this journal has focused on the effects of exercise on healthy articular cartilage, specifically its functional adaptation to loading, both short and long term (Eckstein et al. 2006). The purpose of the current review is to consider the potential effect of exercise on the onset and progression of joint disease, specifically to:

1 address the question of what osteoarthritis (OA) is;
2 provide an overview of the epidemiology of OA, focusing on validated risk factors for its development, in particular the role of both exercise and occupational activity;
3 consider the role of exercise on the joint tissues (particularly cartilage) and the role of exercise in disease management.

We hope by the end that the reader will have a clear perception of the role of exercise in OA that will dispel the common misconception that exercise is damaging to joints. This is a narrative review in which the authors have selected and considered the pivotal articles in constructing this manuscript. The literature on this topic predominantly pertains to the knee and this is reflected in our review.

What is osteoarthritis?

Early investigators tended to regard OA as an isolated disease of articular cartilage. Although cartilage loss is a prominent feature of OA, contemporary models recognize that the entire joint organ is affected by OA. OA can be viewed as the clinical and pathological outcome of a range of disorders that result in *structural and functional failure of synovial joints* with loss and erosion of articular cartilage, subchondral bone alteration, meniscal degeneration, a synovial inflammatory response, and bone and cartilage overgrowth (osteophytes) (Nuki, 1999). OA occurs when the dynamic equilibrium between the breakdown and repair of joint tissues becomes unbalanced (Eyre, 2004). This progressive joint failure may cause pain and disability (Guccione et al. 1994), although many persons with structural changes consistent with OA are asymptomatic (Hannan et al. 2000). The source of pain is not particularly well understood and is best framed in a biopsychosocial

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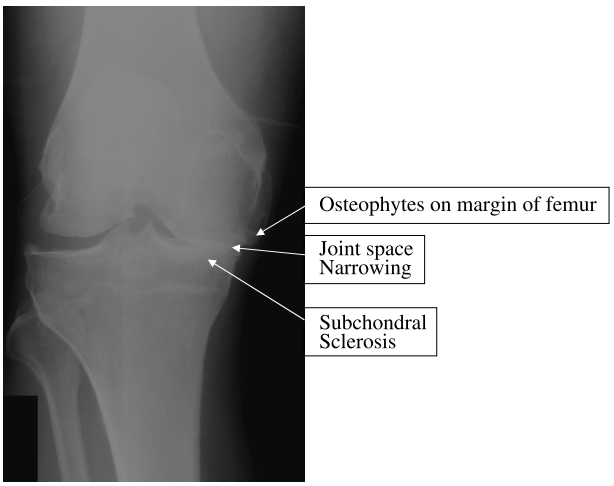


Fig. 1 A weight-bearing plain radiograph of the knee depicting the characteristic features seen in OA: joint space narrowing, osteophytosis and subchondral sclerosis.

framework (posits that biological, psychological and social factors all play a significant role in pain in OA) (Dieppe & Lohmander, 2005). The etiology of pain, however, is not the focus of this review. OA can occur in any synovial joint in the body but is most common in the knees, hips and hands.

In epidemiological investigations, OA is typically defined using radiographs, and less frequently self report. The characteristic features of OA scored on radiographs are osteophytes (osteocartilaginous growths), sclerosis and joint space narrowing (Fig. 1).

Understanding this definition becomes important when we consider the impact of exercise on development of OA, as osteophytes (typically the first feature identified on radiographs) are not necessarily a deleterious finding and may represent an effort on the part of the joint to promote stability. They are important, however, if they represent a source of symptoms, and yet most of the positive evidence that suggests an association between exercise and OA is based on the presence of self-reported OA or radiographic osteophytes and not on symptomatic OA (the concomitant presence of pain and radiographic features). It is the presence of symptomatic OA that is important clinically, not simply the radiographic identification of osteophyte formation or self-reported OA (where misclassification is even more problematic than the commonly used radiographic OA definition).

Epidemiology of OA

OA is the leading cause of disability in older people (Centers for Disease Control and Prevention (CDC) 2001). The reported prevalence of OA varies according to the method used to evaluate it. In most epidemiological studies it is commonly assessed by radiography. Marked osteoarthritic damage must be present, however, to detect characteristic changes with plain radiographs, and they are therefore not sensitive diagnostic tests. About 6% of adults aged > 30 years (Hunter & Felson, 2006) and 13% of persons aged 60 and over (Lawrence et al. 1998) have frequent knee pain and radiographic OA. Although OA is common in the knee, it is even more prevalent in the hands, especially the distal (DIP) and proximal (PIP) interphalangeal joints and the base of the thumb. When symptomatic, especially so for the base of thumb joint, hand OA is associated with functional impairment (Cunningham & Kelsey, 1984; Zhang et al. 2002). OA of the thumb carpo-metacarpal joint is a common condition that can lead to substantial pain, instability, deformity, and loss of motion (Armstrong et al. 1994). Over the age of 70 years, approximately 5% of women and 3% of men have symptomatic OA affecting this joint with impairment of hand function (Zhang et al. 2002). The prevalence of hip OA is about 9% in Caucasian populations (Felson & Zhang, 1998). The prevalence of symptomatic hip OA is approximately 4% (Lawrence et al. 1998).

The prevalence of OA is expected to increase as the population ages and the prevalence of obesity rises (this being an important risk factor; see below). By 2020, it is expected that the number of people with OA may have doubled (Centers for Disease Control and Prevention (CDC), 1994; Badley & DesMeules, 2003).

Risk factors for OA

OA is perhaps best understood as resulting from excessive mechanical stress applied in the context of systemic susceptibility (see Fig. 2 for an overview).

Susceptibility to OA may be increased in part by *genetic inheritance* (a positive family history increases risk), *age, ethnicity, diet* and *female gender* (Felson, 2004a).

In persons vulnerable to the development of knee OA, local mechanical factors such as abnormal joint congruity, malalignment (varus- or valgus deformity), muscle weakness or alterations in the structural integrity of the joint environment (such as meniscal damage or ligament rupture) facilitate the progression of OA. Loading can also be affected by obesity and joint injury (either acutely as in a sporting injury or after repetitive overuse such as occupational exposure), both of which can increase the likelihood of development or progression of OA.

Mechanical factors increasing risk for progression

Local mechanical factors such as the *adduction moment*, *malalignment*, *meniscal damage*, *bone marrow lesions*, and *altered quadriceps strength* potentially put the knee joint at increased risk of progression of OA (Felson, 2004b). Local mechanical factors also mediate the impact on the knee of more systemic factors such as obesity (Felson et al. 2004). Body weight and habitual activity determine how much overall load the joint must routinely sustain and the

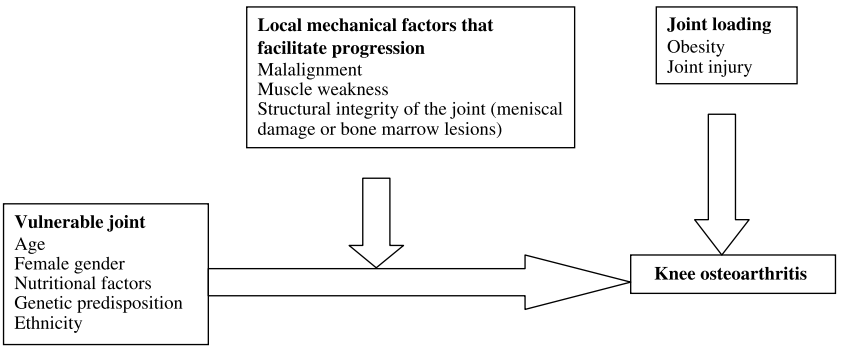


Fig. 2 Schemata demonstrating the risk factors for OA.

mechanical factors determine how this is distributed through the joint. For example, in the knee the relative alignment of the femur, tibia, and patella determines the manner in which this load is distributed over the medial and lateral joint surfaces. As little as 5° of genu varum (bow-legged) malalignment results in an estimated 70–90% increase in compressive loading of the medial knee compartment (Tetsworth & Paley, 1994). This dramatic increase in compressive load corresponds to a four-fold increase in the risk of worsening medial knee OA over 18 month (Sharma et al. 2001). Conversely, genu valgum (knock-kneed) malalignment markedly increases compressive load on the lateral compartment of the knee and elevates the risk of lateral OA progression five-fold (Sharma et al. 2001).

When instability accompanies knee malalignment, as it often does in moderate to severe cases of knee OA, whatever malalignment is present in quiet standing can become exaggerated during walking. Among knees with genu varum in standing, the presence of a varus thrust during walking further elevates the risk of medial OA progression three-fold (Chang et al. 2004). A recent study using quantitative MRI (Sharma et al. 2008a) showed that varus malalignment increased the risk of cartilage loss in the medial femorotibial compartment (tibia and femur) after adjusting for age, sex, BMI, medial meniscal damage and extrusion, and lateral laxity. This analysis (Sharma et al. 2008b) further showed that medial meniscal damage predicted cartilage loss in the medial femorotibial compartment and lateral meniscal damage predicted cartilage loss in the lateral femorotibial compartment. Medial and lateral meniscal extrusion, and medial and lateral laxity, in contrast, did not show consistent relationships with quantitative cartilage outcomes in models fully adjusted for other local factors. This analysis used recently developed regional approaches to cartilage segmentation (see Fig. 3) (Eckstein et al. 2007).

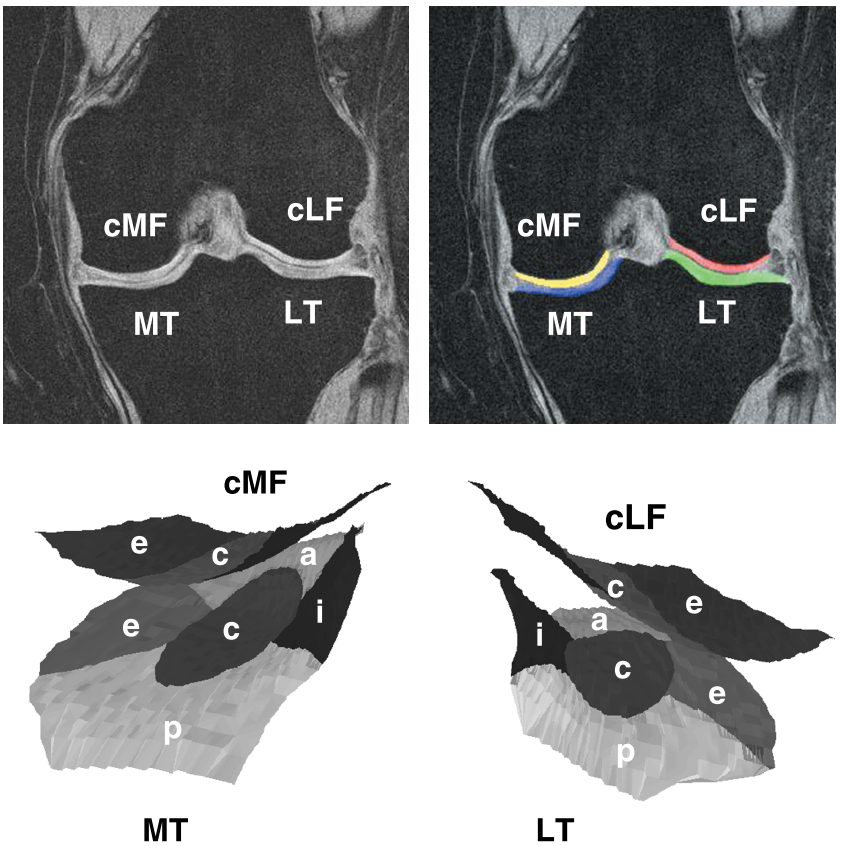
The menisci have many functions in the knee, including the equal distribution of stress between the relatively incongruous femorotibial joint surfaces. Other functions may include stability enhancement and lubrication (Seedhom et al. 1974). Until relatively recently, surgical interventions on the meniscus consisted largely of resection, rather than preservation strategies. Identification of a tear on an MRI in a person with OA is not an indication for surgery, as meniscal tears in persons with OA are almost universal and do not appear to be associated with increased symptoms (Bhattacharyya et al. 2003). Further damage to the morphology of the meniscus and alteration in its position predicts progression of cartilage lesions on MRI (Hunter et al. 2006a).

Bone marrow lesions (BML) are irregular hypointense signals in the subchondral bone on T1- and hyperintense on T2-weighted fat-saturated MRI images. They frequently occur in subjects with trauma or OA, have been reported to be associated with pain (Felson et al. 2001), and were found in one study (Hunter et al. 2006b) to predict compartment-specific OA progression. Further they infrequently regress and their effect appears to be mediated by alignment.

Muscle weakness, in particular the quadriceps with regards to the knee joint, may be an important risk factor for knee OA in women. In a study by Slemenda et al. (1997) the quadriceps muscle was found to be on average 20% weaker (even after controlling for body mass and other covariates) among those with radiographic signs of OA and appeared to predate the onset of disease in women. Weakness is present even before the knee becomes painful, suggesting that deficient quadriceps strength may be a risk factor for the subsequent development of symptoms. One consequence of quadriceps weakness is that the knee is rendered less stable during risky occupational or recreational activities. To the extent that they are able to improve the dynamic stability of the knee, quadriceps exercises may offer some protective advantage to patients who are routinely engaged in high risk activities.

Obesity

From a public health perspective the largest modifiable risk factor for knee OA is body weight. During gait, body weight is transferred onto the knee with substantial leverage, so that each additional kilogram of body mass increases the compressive load over the knee by roughly 4 kg (Messier et al. 2005). Even modest weight reduction is therefore capable of achieving sizable decreases in the compressive stress over the knee. Where body weight is



MT = medial tibial condyle, LT = lateral tibial condyle
cMF = central (weight-bearing) medial femoral condyle
cLF = central (weight-bearing) lateral femoral condyle
e = external subregion
i = internal subregion
a = anterior supbregion
p = posterior subregion
For definitions of subregions, please see Wirth et al. 2008

Fig. 3 Regional division of tibiofemoral joints for cartilage morphometric analysis and further subregional delineation.

excessive, weight loss interventions have been found to lessen the risk for developing symptomatic disease and also decrease the likelihood that existing knee OA will worsen (Messier et al. 2004; Christensen et al. 2005).

Joint injury

Occupation

When considering the role of exercise on OA, the effects of activity on load-bearing joints can also be investigated by exploring insights from occupational activity and its respective impact on OA joints. Microtrauma is known to result from the repetitive application of high mechanical stresses to vulnerable structures within the joint. Current evidence would suggest that OA is more common in those who perform heavy physical work and particularly in those whose jobs involve knee-bending, kneeling or squatting. For example, among carpenters, miners and others with a history of occupational exposure to repetitive squatting or kneeling, knee OA is far more prevalent than in the general population (McMillan et al. 2005).

A recent systematic review of the literature suggested a strong positive relationship between work-related knee bending exposure and knee OA (range of odds ratio: 1.4–6) (Maetzel et al. 1997). Estimates of the attributable proportion for these occupational exposures suggest that between 5% (Cooper et al. 1994) and 20% of all symptomatic knee OA may result from occupations involving repetitive knee use.

Previous studies of occupational groups exposed to repetitive use of small hand joints have shown an excess of hand OA in joints that were used repetitively (Kellgren & Lawrence, 1952; Lawrence, 1961; Hadler et al. 1978). In Lawrence's classical surveys, coal miners were shown to have more OA than dockers who, in turn, had more OA than office workers (Kellgren & Lawrence JS, 1952). Different types of hand function may predispose to OA in different

articular areas, with studies of repeated pincer grip and generally increased workload demonstrating a predisposition to distal interphalangeal joint OA (Hadler et al. 1978; Bergenudd et al. 1989; Nakamura et al. 1993).

Joint injury from sports

Consideration of the role of exercise in OA needs to pay heed to the potential overlap with sports injury. Among both genders, a past history of injury to the stabilizing or load-bearing structures of the knee renders the joint highly vulnerable to OA in subsequent years. This is distinct from the degenerate meniscal tears that occur frequently as part of the OA disease process that was described earlier.

Knee injury/trauma has been identified as the most important modifiable risk factor for subsequent knee OA in men, and is second only to obesity in women (Felson et al. 2000). It typically occurs in the younger population and as such leads to prolonged disability and economic cost (Yelin & Callahan, 1995), largely due to work loss. A large prospective cohort study revealed that joint injury substantially increased the risk for subsequent knee OA [relative risk, 5.17 (CI, 3.07–8.71)] (Gelber et al. 2000).

The high incidence of knee OA in the years following anterior cruciate ligament (ACL) (Roos et al. 1995; Lohmander et al. 2004) or meniscal injury (Roos et al. 1995; Englund et al. 2003) is well-documented, and credible evidence suggests that current arthroscopic procedures, including ACL reconstruction and meniscectomy, are not sufficient to fully restore normal joint mechanics or neutralize the long-term risk of OA (Englund & Lohmander, 2004; Andriacchi et al. 2006).

Meniscal injury and subsequent meniscectomy is often accompanied by cartilage degeneration and the onset of OA because of the high focal stresses imposed on articular cartilage and subchondral bone due to excision of the meniscus (Tapper & Hoover, 1969; Johnson et al. 1974). A number of recent publications have documented long-term follow-up of radiographic changes after meniscectomy (Roos et al. 1998; Macnicol & Thomas, 2000; McNicholas et al. 2000). Whilst the observed incidence of secondary OA varies, Roos et al. (1998) reported mild radiographic changes in 71% of knees, with more advanced changes, comparable with a Kellgren and Lawrence grade 2 or higher in 48%. Further analysis suggested that surgical removal of a meniscus following knee injury represented a significant risk factor for radiographic tibiofemoral OA, with a relative risk of 14.0 after 21 years. Older age at the time of trauma or surgery appears to predict a more rapid deterioration to OA of the involved knee.

Certain competitive athletes, including hockey players, whose sporting activities repetitively stress the stabilizing structures of the knee and who are at great risk for knee injury also have very high rates of early-onset knee OA (Sandmark et al. 1999).

Risk of OA from sports participation

Recent years have witnessed an enormous increase in the popularity of recreational exercise. There is a plethora of evidence supporting participation in regular exercise, including recreational activities or competitive sports, as it improves general health and may increase longevity. Individuals with normal joints frequently ask whether their exercise programs increase the risk of developing OA.

The initial studies that evaluated the relationship between regular recreational weight-bearing exercise and OA of the knee generally found no ill effects on the joints from exercise participation (Lane et al. 1986; Panush et al. 1986). More recent studies that assessed the longitudinal effects of aging and exercise on OA of the hip and knee after 5 and 8 years of follow-up also found no increased risk of developing OA in runners, compared with age-similar controls (Hannan et al. 1993; Panush et al. 1995). What is clear from the data is that the risk of subsequent OA relates more to the intensity of the level of participation, the performance level (e.g. elite vs. recreational) and the concomitant presence and or likelihood of joint injury. In this light we have considered recreational vs. elite separately, paying particular attention to the presence or consideration of joint injury.

A study of female physical education teachers compared with population-based controls found no increase in rates of radiographic hip or knee OA, and actually found lower rates of knee OA (White et al. 1993). Potentially in contrast to the former study, 571 graduates from a single Swedish training facility had higher rates of self-reported OA knee and knee injury than the comparator group (Sandmark & Sandmark, 2000). The graduates had a current median age of about 57 and were matched with people from a population register. However, radiographs were not taken in this study and therefore the prevalence of radiographic OA is not certain. Further, the concomitant presence of joint injury likely inflated the risk of subsequent OA.

A number of population-based studies have studied the effects of participation in sport on rates of OA. These studies provide a clearer picture of overall associations in communities but tend to rely upon recall of sporting participation. A British team of investigators (Cooper et al. 2000) examined a large cohort with knee radiographs taken 5 years apart and a median age at follow-up of 75.8 years. They found that a history of regular sport participation was associated with an increased risk of incidence (but not progression) of knee OA. The risks for hip joint replacement from retrospective case-control studies of Swedish male (Vingard et al. 1993) and female (Vingard et al. 1998) population-based cohorts found that in those with the most hours of recalled sporting activities (over 800 h in total based on recall of all sporting involvement up till the age of 50), men had a relative risk of 4.5 and women 2.3 of developing OA of the hip, compared with

the lowest activity groups. Importantly, men who had performed jobs with high joint loading as well as high levels of sport had almost double the relative risk. Lane et al. (1999) examined the association of reported exercise and radiographic OA hip in a population of 5818 women with a mean age of about 72. Women who reported regular recreational physical activities (defined as one to five times a week) as teenagers, at age 30, and at age 50 had significantly greater odds of moderate to severe hip OA than those who performed no recreational activities. In all of these population-based studies, a major confounding issue has been the separation of the impact loading aspects of the sport from the associated injuries which undoubtedly predispose to OA. A retrospective, case-control study from the UK used 216 subjects (drawn from a larger national survey of 4316 persons) with self-reported OA knee and matched each of them with four controls (Sutton et al. 2001). Extensive data on lifetime exercise involvement was obtained. The only strong risk factor for knee OA was a reported prior knee injury (odds ratio 8.0); most of these knee injuries were related to sport.

Therefore, the results from these studies show that individuals who had normal joints and participated in low-impact exercises did not have an increased risk of developing OA of the knee or hip as they aged, independent of joint injury. Whilst there is some evidence suggesting an increased risk with activity this has not been adequately disentangled from injury. In this light there is *no good evidence supporting a deleterious effect of exercise on joints in the setting of normal joints and moderate activity*.

In contrast, there does seem to be an association between elite sports participation and an increased risk of OA. However, the nature of the sport is very important to the degree of risk. The sports with major risk are those that involve repetitive, high intensity, high impact forces through the affected joints, especially where there is a high associated risk of injury. Categorizing exercise into different levels of impact is somewhat arbitrary but relates to the extent of compressive loading during the activity. Common examples of high-impact exercise include running, dance exercise, tennis, racquetball, and squash. This is in contrast to low- to moderate-impact exercises such as walking, swimming, stair climbing, rowing, and cross-country skiing.

A Finnish group examined hospital admission rates over 21 years for OA (hip, knee or ankle) for over 2000 male ex-athletes (Kujala et al. 1994). They divided the cohort into endurance (long-distance running, cross-country skiing), team (soccer, ice hockey, basketball, track and field), and power (boxing, wrestling, weight-lifting, throwing) sports. All three groups had higher incidences of OA-related admissions, with power and team sports having admissions at an earlier age. Soccer players and weight-lifters had higher risk, which was due at least partly to knee injuries in the former and high body mass index at age 20 in the latter (Kujala et al. 1995). The former power-sport competitors had the highest odds ratios for hip disability, whereas only the team-sport players had a high risk for knee disability, again perhaps reflecting the injury-prone nature of these sports (Kettunen et al. 2001).

A Swedish retrospective cohort reported both hip and knee radiographic OA associations in 71 elite and 215 non-elite players with a mean age of 55 and compared the rates with those of 572 age-matched controls (Lindberg et al. 1993; Roos et al. 1994). For the hip, the prevalence of OA was 14% for elite players compared with 4.2% in non-elite and control groups (Lindberg et al. 1993). For the knee, the prevalence of OA was 15.5%, 4.2%, and 1.6% in elite, non-elite, and control groups, respectively (Roos et al. 1994).

The data from studies of runners indicate that the distance run and intensity may play a role. In a retrospective cohort study, examinations were performed in 1973 and 1988 on a number of former athletes: 27 long-distance runners (averaging 60 miles per week in 1973), nine bobsleigh riders, and 23 controls (Marti et al. 1989). The athletes had a mean age of 42 years at the second examination. Radiographic hip OA was found in 19% of the runners, but in neither of the other groups. Age and number of miles run per week in 1973 were the positive predictors of radiographic OA. Spector et al. (1996) compared 67 female elite middle- and long-distance runners and 14 female tennis players (aged 40–65) with a large matched control group. Radiographic hip and knee OA rates were significantly higher in the former athletes compared with controls of a similar age, with a tendency to more patellofemoral OA in the runners. No clear risk factors were seen within the ex-athlete groups, although the tennis players tended to have more osteophytes at the tibiofemoral joints and hip, but the runners had more patellofemoral joint disease. In contrast, in a retrospective cohort study, the rates of radiographic hip OA in 60 ex-marathon runners were not higher than in controls, although the timing of radiographs differed for the two groups (Puranen et al. 1975). A study comparing 504 university level cross-country runners with similar-level swimmers found no difference in levels of hip and knee pain but x-ray examinations were not performed (Sohn et al. 1985). A small prospective study of 17 male runners (nine were marathon runners) compared with controls found no difference in radiographic OA at the hip, knee, ankle or feet (Panush et al. 1986).

Lane et al. (1986, 1993, 1998) have presented a series of reports from a well-described, prospective cohort of subjects from a long-distance running club – perhaps closer to recreational runners than the elite athletes studied above. At baseline, 41 runners aged 50–72 years and averaging 25 miles a week were compared with controls matched for age, sex, years of education, and occupation (Lane et al. 1986). There were no differences in clinical and radiographic OA findings in the knee and lumbar spine. Follow-up of

this cohort at 5 (Lane et al. 1993) and 9 (Lane et al. 1998) years showed significant within-group progression of both osteophytes and total knee radiographic scores ($P = 0.01$ for runners and $P = 0.05$ for non-runners) and joint space narrowing in non-runners ($P = 0.01$), with no significant between-group differences. For the runners, regression analysis disclosed that the predictors of progression of radiographic knee OA were baseline radiographic score and a faster pace per mile. Hip radiographs taken 9 years later also showed no differences in OA between the groups (Lane et al. 1998). Another study of 30 long-distance runners, 90% of whom averaged 12.5–25 miles a week over a median of 40 years, found no differences in rates of radiographic OA at the hips, knees or ankles when compared with controls matched for age, body mass index, and occupation (Konradsen et al. 1990).

Thus elite athletes who perform their activities with high impact and high stress to the joints appear to have an increased risk for OA in the hips and knees compared with age-matched controls (Kujala et al. 1995; Spector et al. 1996). Again the concomitant presence or likelihood of joint injury increases the risk of developing OA.

Exercise and cartilage

When considering the impact of weight-bearing activity on the joint tissues there is a preponderant focus on cartilage as this pertains to OA. In general, though, the effect of exercise on load-bearing joints extends beyond cartilage, and exercise is known to have advantageous trophic effects on periarticular bone and muscle in particular, and also tendon, at least in men (Magnusson et al. 2007).

Hyaline articular cartilage provides the articulating surface of synovial joints. Its complex composition facilitates the even transfer of forces from one subchondral plate to the other (Mow & Setton, 1998). Many studies suggest that articular cartilage is mechano-adaptive; that is, the biosynthetic activity of chondrocytes is responsive to mechanical stimuli and can alter the morphology and composition of cartilage (Carter et al. 2004). Other studies suggest that excessive mechanical force can have a deleterious effect on the prevalence of OA (Kujala et al. 1995; Spector et al. 1996). Due to the pluripotent effects of mechanical loading on articular cartilage, physical activities may play an important role in either the causation of or protection against OA. Prolonged immobilization in animals leads to reductions in articular cartilage thickness, although it does not necessarily become osteoarthritic (surface remains smooth and no osteophytes or erosions develop) (Vanwanseele et al. 2002b). Similarly, in humans the absence of normal joint loading due to spinal cord injury results in rates of cartilage thinning that are higher than those observed in persons with OA (Vanwanseele et al. 2002a, 2003). Thus cartilage undergoes atrophy in the absence of mechanical stimulation.

In animal studies, physical activity has been shown to have varying effects on articular cartilage (Jurvelin et al. 1986; Lammi et al. 1993; Newton et al. 1997). Overexercised animals have been found to develop glycosaminoglycan depletion (Komulainen et al. 1999). In humans, there are data supporting both directions of effect of physical activity, and it is not understood how both could be true. To test this effect in humans, Jones and colleagues studied 92 children ranging in age from 9–18 and evaluated their cartilage thickness on MRI initially cross-sectionally, followed by a longitudinal assessment of 74 of the same cohort (Jones et al. 2000, 2003). They reported that self-reported activity prior to the MR examination was related to articular cartilage volume and that the effect of physical activity was mediated, in part, by its relation to muscle strength. The longitudinal observation suggested that participants who were above the median for average intensity of sport gained more cartilage than did those below the median. It should be noted that these observations were in children and that analysis of cartilage volume does not permit separation of the effect on cartilage development (thickness) from bone growth (epiphyseal joint area). Vigorous self-reported activity in children was associated with greater accrual of cartilage in tibial but not patellar cartilage compared with children with no reports of vigorous activity (Jones et al. 2003).

What may be more relevant to disease occurrence is whether activity in middle or older years affects cartilage thickness at a time when the person is at highest risk of disease. Eckstein et al. (2002) has demonstrated that in 18 triathletes their knee joint surface area was larger and cartilage thickness the same as 18 controls who had never been physically active for more than 2 h per week, but were not obese. This may suggest that load bearing may have a greater influence on the articular surface area than on cartilage thickness. Gratzke et al. (2007) reported no increased cartilage thickness (but also no increased joint surface areas) in weight-lifting and sprinting top athletes compared with non-athletic controls. Animal studies corroborate the evidence that weight-bearing physical activity appears to protect against the development of OA (Otterness et al. 1998) but, as stated above, human studies investigating the influence of physical activity on cartilage thickness have reported somewhat conflicting findings.

A cross-sectional study in athlete runners, occasional runners and sedentary controls found an increased Delayed Gadolinium Enhanced MRI of Cartilage (dGEMRIC) index in knee cartilage in those who exercised (Tiderius et al. 2004) and a brief exercise intervention in persons at high risk for the development of OA was reported to have a favorable effect on the dGEMRIC index, indicating increased proteoglycan, compared with sedentary individuals not participating in exercise (Roos et al. 2005). dGEMRIC relies on intravenous injection of a negatively charged MR contrast agent and the acquisition of a T1 map after equilibration

of the contrast agent in the cartilage, to estimate the glycosaminoglycan distribution within cartilage.

This finding may suggest a protective effect on the development of OA in persons at high risk of developing OA, but another study found no significant difference in the *in vivo* deformational behavior of cartilage between athletes (weight-lifters, sprinters) and non-athletic controls (Eckstein et al. 2005). In a longitudinal study of younger community-based participants, Foley et al. (2007) identified that physical work capacity was negatively associated with cartilage loss and thus appeared to be protective.

Exercise as a tool in the therapeutic armoury

The benefits of recreational exercise are not distinct from that prescribed as part of a therapeutic intervention. Exercise has been a central component of any effort to conservatively manage OA. Exercises can be prescribed to facilitate weight loss, preserve joint range of motion, improve strength, improve functional performance, and reduce symptoms (Ettinger et al. 1997; Bennell et al. 2005). Persons with OA capable of exercise have been recommended to be encouraged to partake in a low-impact aerobic exercise program (walking, biking, swimming or other aquatic exercise) (Roddy et al. 2005b). Aquatic exercise is preferable to land-based exercise as the body's buoyancy greatly limits the compressive load that the knee must sustain. Seated bicycling can similarly partially unload the knee and keep it stable while it is exercised through a large range of motion. The rationale behind the promotion of low-impact exercise is that it will encourage the benefits of exercise whilst avoiding the potentially damaging influences of high-impact activities.

Quadriceps weakness is common among patients with knee OA, in whom it had been believed to be a manifestation of disuse atrophy which develops because of unloading of the painful extremity (Hurley, 1999). Some studies, however, have indicated that quadriceps weakness may be present in persons with radiographic changes of OA who have no history of knee pain, and in whom lower extremity muscle mass is increased, rather than decreased (Slemenda et al. 1997). Quadriceps weakness was hence considered a risk factor for the development of knee OA, presumably by decreasing stability of the knee joint and reducing the shock-attenuating capacity of the muscle (Slemenda et al. 1998; Hurley, 1999). The role of exercise therapy was the subject of a systematic review that concluded that there is evidence of beneficial effects of exercise therapy in patients with OA of the knee (van Baar et al. 1999). Quadriceps strengthening exercises were found to lead to improvements in pain and function. Most strengthening exercise regimens were recommended to begin with isometric exercises and then advance to isotonic resistance exercises as tolerated.

It is important to individualize exercise therapy for hip or knee OA, particularly considering individual patient preference, and ensure that adequate advice and education to promote increased physical activity is provided (American Geriatrics Society Panel on Exercise and Osteoarthritis 2001, 2003; Roddy et al. 2005a). As adherence is the main predictor of long-term outcome from exercise in hip or knee OA, strategies to improve adherence should be adopted, such as long-term monitoring. Similarly, patients should do exercise they enjoy to promote long-term participation. Some exercises are likely to be harmful in the long term, particularly those that involve high velocity impact (running, step aerobics, etc.) on an already injured joint surface; thus these should be actively discouraged.

Concluding remarks

Based upon current evidence, individuals with normal joints and no joint injury should be actively encouraged to exercise regularly both for benefits as they pertain to the joints and other health benefits. There is no strong evidence to suggest that vigorous low-impact exercise is associated with an accelerated rate of development of OA. The current evidence in persons who participate in elite sports activity, particularly in sporting groups susceptible to joint injury, suggests that these groups are at increased risk for OA as a result of their participation, but it is unclear whether participation in the absence of injury is harmful. When considering the individual risk of OA development it is important to consider the type of sports participation, its intensity and extent of joint impact, the existence of concomitant joint injury, family history of OA and body weight, as well as occupational risk. Exercise has, and will continue to play, an important role in both the pathogenesis and management of OA.

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