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Effects of Exercise on Osteoarthritis: A Review

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ABSTRACT

Osteoarthritis is a disease that degenerates articular cartilage and can cause debilitation in muscular strength and aerobic capacity. This review examines current research to determine whether certain activities increase the risk for developing OA, and discusses recent exercise studies involving OA patients. Research has shown that the risk for developing OA appears to increase with repetitive trauma as experienced in sports or when there is a history of joint injuries. However, moderate levels of running do not seem to increase symptoms or incidence of OA. It appears that not only can the OA population benefit from the typical effects of aerobic exercise such as increased work capacity and reduced CHD risk, but also from analgesic relief and the possibility of decreased medication. Resistance training studies found significant increases in strength and improvement in symptoms of OA. Other benefits of resistance training include pain reduction, increased mobility and walking speed, and decreased stiffness. Since moderate levels of exercise did not appear to increase the symptoms or risk of developing further OA, exercise would seem warranted for people with OA.

Key Words: joint injury, running, aerobic exercise, resistance exercise

Introduction

Osteoarthritis (OA) is a degenerative disease that results in the breakdown of articular cartilage and eventually involves the surrounding tissues, synovium, and bone. As cartilage is damaged by excessive abnormal stress or impaired nutrition, degenerative enzymes are released to dissolve damaged cells. This process continues until the exposed bone experiences added stress. New bone formation will occur in the form of sclerosis and/or cysts. Finally, as both the bone and cartilage break down, chronic inflammation begins to occur in the synovium of the joint (17, 18). During this process, friction in the joint increases, shock absorption decreases, and the impact loading increases.

OA, rarely seen in patients under the age of 40, can affect a wide range of joints. The onset of OA is usually insidious and has little or no joint effusion. Usual symptoms are pain on motion (early), pain at rest (later), aggravation of pain by prolonged activity, and localized stiffness. Common signs are localized tenderness, crepitus on motion, mild joint enlargement, synovitis (less common), and possibly gross deformity in later stages (32). These signs and symptoms of OA usually are not seen until there is significant damage to the cartilage. Lack of symptoms in early stages of OA is due to the low level of neural innervation in the cartilage (17, 18). The symptoms surface once the disease has affected surrounding tissues that have a very strong supply of nerves (17, 18).

Epidemiological studies show that OA is common. About 80% of individuals over 65 years of age show some evidence of the disease (10). Since OA can cause debilitating physical condition and affects a great majority of our older population, preventive and intervention strategies such as exercise are warranted.

The purpose of this review is to describe recent studies in patients with OA which address the risk of developing OA, and changes in outcome variables resulting from exercise. This paper also assesses the strengths and weaknesses in this field of research and makes recommendations regarding future research.

Functional Capacity of People With OA

When looking at older populations affected by OA, one concern even with asymptomatic individuals is the functional decline that occurs. It is generally known that elderly persons who are asymptomatic need a higher $\dot{V}O_2$ than younger individuals at the same workload. Increases in submax $\dot{V}O_2$ for elderly individuals can be as much as 10% in treadmill exercise. Martin et al. (27) postulated that this increase in $\dot{V}O_2$ demand may be due to the different muscle recruitment patterns resulting from the muscle loss that occurs with aging. This increased submaximal $\dot{V}O_2$ of older people can also be attributed to other conditions besides aging. Declines in $\dot{V}O_2$ max have been attributed to increased inactivity, fat mass, and total body weight, all of which may play a greater role than age itself (13, 16, 36).

The decrease in functional capacity caused by increased inactivity typical of asymptomatic older persons is further hampered when chronic conditions such as OA occur. Minor et al. (30) found the $\dot{V}O_2$ max of

patients with rheumatoid arthritis and OA (mean age 61 yrs) to be 19.1 ml · kg^{-1} · min^{-1}. This figure is approximately 20% lower than values in healthy asymptomatic elderly patients (15, 20, 38). In further support of this finding, Philbin et al. (35) compared asymptomatic age-matched controls to patients with hip and knee OA and found significantly decreased cycle ergometry $\dot{V}O_2$ max scores ($p < 0.0005$ for knees, $p < 0.0001$ for hips). The $\dot{V}O_2$ max averages for hip and knee OA patients were 14.9 and 12.8 ml · kg^{-1} · min^{-1}, respectively, with the control groups having 19.0 and 17.6 ml · kg^{-1} · min^{-1}, respectively. Their study also reported significantly more manifestations of coronary heart disease (CHD) in OA patients versus controls (27 vs. 13%, $p < 0.05$).

Muscular Strength and Joint Flexibility

Muscular function decline in OA patients has also been documented in several studies. Nordesjo et al. (34) determined the isometric strength of OA patients and healthy controls in knee flexion and extension. They found a significant decrease of knee flexion and extension in the OA patients; the range was 55–70% of that of healthy controls. Madsen et al. (26) reported significant decreases in isokinetic and isometric strength (40 and 15%, respectively, $p < 0.01$ for both) in 20 women with gonarthrosis (knee OA) when compared to 26 healthy controls.

Messier et al. (28) reported significant decreases in patients with OA in regard to knee flexibility when compared to controls ($p = 0.005$ for extension and $p = 0.04$ for flexion). OA patients had 9.6° less knee flexion and 15.9° less knee extension when compared to healthy controls. Furthermore, knee angular velocity measured by a one-camera kinematic setting was significantly lower while walking ($p < 0.05$).

Decreases in aerobic capacity and muscular function of this population can be a great burden and could obviously accelerate physical decline. Regular exercise could help OA patients regain or retain muscular and aerobic capacities. However, if exercise exacerbates the OA symptoms, the benefits of exercise may not be worth the increased progression of this degenerative disease.

To determine whether exercise is detrimental to OA, one must examine the causes of this condition. The complete etiology of OA is still unknown, but it has been found to be multifaceted in origin. Factors associated with the disease are metabolic, genetic, mechanical, and environmental (3, 19, 29).

Sports Participation and OA

The fact that exercise participation, through mechanical or environmental influences, could cause or exacerbate OA is of concern. Numerous studies have attempted to determine whether prolonged participation in sports and in running increases the risk of developing OA. Spector et al. (40) estimated the risk of developing OA in 81 female former elite athletes (67 runners, 14 tennis players) compared to 977 age-matched controls. From radiographic information on the tibiofemoral (TF), patellofemoral (PF), and hip joints, the study reported that the former athletes had a higher incidence of osteophyte formation at all sites as well as joint space narrowing at all sites except the tibiofemoral joint. Correcting for weight and height, odds ratios were calculated to analyze the risk of developing OA at each joint. Overall, the risk for osteophyte formation increased by twofold to well over threefold at all three joints, and there was a higher incidence of narrowing of the patellofemoral joint.

In further support of this study, 38 female former professional dancers were examined for incidence of OA in the ankle, subtalar, and first metatarsophalangeal (MTP) joints and then compared with a control group. The dancers' careers averaged 37 years (range 13 to 54) and their mean age was 59 years (range 50 to 66). The results revealed significant decreases in joint space of the ankle and MTP joints of the dancers ($p < 0.05$). Furthermore, the amount of arthrosis in the subtalar, MTP, and ankle joints was found to be significantly higher in dancers (33).

Kujala et al. (23) studied 117 male former elite athletes (28 long-distance runners, 31 soccer players, 29 weight lifters, and 29 shooters) to determine the prevalence of TF and PF OA. They found that 29% of the soccer players, 14% of the runners, 31% of the weight lifters, and 3% of the shooters had OA. Comparing age-adjusted risk ratios of the shooters to soccer players, weight lifters, and runners, the risk ratios were 12.3, 12.9, and 4.8, respectively. In other words, these 3 groups were 4 to 12 times more likely than shooters to develop OA. Finally, if the subjects had previous knee injuries, the odds ratio was 4.73.

Accelerated Progression of OA With Previous Injuries

This last finding is interesting since it shows that past injury may in turn raise the risk of developing OA. It has been thought that ligamentous instability, prior injury, or meniscal removal could play a major role in the development of OA. In support of this point, for 4-1/2 years Warren and Marshall (41) followed 86 patients who had undergone surgery to repair an injured anterior cruciate ligament and/or medial collateral ligament (81% from sports injuries). In the final follow-up examination, 42% had chondromalacia patellae and 20 to 52% had radiologic abnormalities.

Furthermore, Funk (11) examined patients who had reconstructive surgery to repair meniscal injury and the anterior cruciate ligament simultaneously. The number of meniscal tears at first visits was 42%. For patients who declined surgery the first year and continued to participate, the number of meniscal tears in the second

Table 1
Summary of Aerobic Training Studies

Study	*N*	Subjects	Mode & Intensity	Frequency	Duration	Results
Morey et al. (31) (1989)	69	70.0 yrs M only	Walking (65–75% max HR), cycling & water aerobics	3 ×/wk (45 min)	4 mos	Max MET level increased from 7.1 to 8.3 ($p < 0.001$); submax HR decreased from 123.7 to 118.8 bpm ($p < 0.001$); RHR decreased from 68.1 to 63.3 bpm ($p = 0.005$)
Minor et al. (30) (1989)	120 (80 OA)	54.3 yrs M & F	Walking (W), water aerobics (A) (60–80% max HR), & ROM exercise in groups	3 ×/wk	12 wks	50-ft walk time, aerobic capacity, and exercise endurance improved signif. in W & A groups vs. ROM group ($p < 0.001$); pain also decreased nonsignif. in 2 exercise groups ($p = 0.216$)
Kovar et al. (22) (1992)	102	70.4 yrs M & F	Walking (W) & nonexercising controls	3 ×/wk (30 min)	8 wks	Signif. improvement in timed walking distance ($p < 0.0001$), 27% pain reduction ($p = 0.003$), and in amount of medication used ($p = 0.08$) in W group vs. controls
Beals et al. (1) (1985)	20	50.2 yrs M & F	Cycle ergometry	n.a.	n.a.	No exacerbation of arthritic symptoms during progressive maximal cycle ergometry testing
Ettinger et al. (2) (1997)	439	69.6 yrs M & F	Walking (50–70% HRR) & calisthenics or health education	3 ×/wk (1 hr)	18 mos	Peak $\dot{V}O_2$ increased ($p = 0.03$), 10% decrease in durability ($p < 0.001$); 12% decreased pain score ($p = 0.001$), increased distance in 6-min walk ($p < 0.001$), and increased KF strength ($p = 0.004$)

Note. HRR = heart rate reserve, KF = knee flexion.

year doubled. This damage to menisci could definitely increase the chance of developing OA, since it is this very tissue the disease attacks.

The acceleration of OA with prior injury is further supported by Kujala et al. (23). In a study on former elite male athletes, they observed that 50% of those with a history of knee injuries had knee OA whereas only 13% of those without knee injuries had OA of the knee ($p = 0.0003$). These studies suggest there is an increased risk for developing OA when participating in sports over many years or when knee injuries have occurred. However, most of these studies used small samples and different rating scales for radiological findings, which may have altered the results. Also, the studies examining OA risk during sports were all retrospective, or nonexperimental, and did not look at changes prospectively. Thus the logic for a cause and effect relationship in these studies is limited.

Running and the Risk of OA

Several studies have examined the effects of long-distance running on the incidence of OA (20, 23, 24, 37). Sohn and Micheli (39) compared 504 (mean age 57 years, range 23–57) former varsity cross-country runners to a control group of 287 former college swimmers. The runners ran an average of 25.4 miles per week over 11.3 years. The percentage of runners and swimmers reporting severe hip or knee pain was 2.0 and 2.4, respectively. With such a small percentage reporting knee pain, it appears that moderate levels of running over time did not increase the incidence of OA.

Lane et al. (24) examined 34 runners (mean age 59.8 ±0.9 yrs) and compared the roentgenographic results for presence of OA at baseline and 2 years later. After 2 years both the controls and the runners had significantly increased signs of OA ($p < 0.001$), but there were no differences between groups. Furthermore, women runners had a significant increase in spur formation ($p < 0.001$).

Lane et al. (25) examined 498 long-distance runners (age 58.6 ± 0.3 yrs) and compared them to 365 controls for musculoskeletal disability, heart rate, blood pressure, and rate of disability. Runners showed less physical disability ($p < 0.01$) and maintained more functional musculoskeletal capacity than controls ($p < 0.001$). Heart rate and blood pressure were also significantly lower in the runners ($p < 0.001$ for diastolic BP and heart rate and $p < 0.05$ for systolic BP).

Konradsen et al. (21) examined 27 long-distance runners (age 58 yrs, range 50–68) who had been competitive in the 1950s and still ran 12 to 24 miles a week. No significant difference was found between the matched controls (age 57 yrs) and the runners in joint alignment, ROM, pain levels, cartilage thickness, grade of degeneration, or osteophytosis in the hip, knee, or ankle joints.

Overall, these studies show no concrete evidence that long-distance running increases the incidence of OA or its symptoms (with the possible exception of increased spur formation in women). Furthermore, it should be noted that one study (25) reported less disability and more functional capacity in runners compared to controls, which would appear to support the rationale for exercising in the OA population.

Effects of Aerobic Exercise

Because moderate or light aerobic activity does not seem to either increase the development of OA or hasten its progression, it would appear that exercise should be encouraged in this population. The primary goals of such a program should be to increase functional capacity in the muscle and soft tissues surrounding the major joints and to increase aerobic capacity and flexibility in these joints.

Table 1 summarizes the findings of recent key studies of OA patients involved in aerobic training programs (1, 2, 22, 30, 31). The major finding from these studies is that aerobic training modes of walking and water aerobics significantly increases aerobic capacity. Increased aerobic function should lead to a decreased morbidity/mortality of CHD, which have been shown to be higher in OA patients (35).

These studies show significant increases in aerobic capacity and walking distance as well as decreases in submaximal and resting heart rate. Except for the study by Beals et al. (1), all the studies in Table 1 had subject groups of moderate size. Of the 3 studies that assessed pain level, 2 found significant decreases in pain (2, 22). Another trend in the Kovar et al. (22) study was the decrease in use of medications ($p = 0.08$) by OA patients after exercise training. These results suggest an analgesic effect from exercise that may prove clinically useful.

These studies shed light on the effects of aerobic exercise in the OA population, but further studies are needed to more fully explore the long-term benefits and the possibility of pain reduction. More attention needs to be given to functional assessments such as pain reduction, comfort, and ability to perform everyday tasks. Also, to make the programs more comparable, the details of exercise programs should be presented. For example, the Kovar et al. (22) study reporting significant decreases in pain did not state the intensity level of aerobic exercise. Such details are essential in determining exercise guidelines for patients with OA.

Table 2
Summary of Resistance Training Studies

Study	*N*	Subjects	Mode & Intensity	Frequency	Duration	Results
Morey et al. (31) (1989)	69	70.0 yrs M only	Isotonic exercises (for total body)	3 ×/wk	4 mos	Abdominal strength increased signif., 16.5% ($p <$ 0.001)
Fiatarone et al.(5) (1990)	10	90 yrs M & F	NK table isotonic (50–80% 1-RM)	3 ×/wk (3 × 8)	8 wks	KE strength increased 174% ($p < 0.001$); muscle size (via CT scan) increased 9% ($p = 0.05$); walking time decreased by 48% from 43.4 to 29.6 min ($p = 0.05$)
Fisher et al. (9) (1991)	15	67.6 yrs M only	Isometrics & progressive isotonic exercises	3 ×/wk	15 wks	Signif. increase in strength (35%), endurance (35%), & angular velocity (50%); signif. decreases in dependency (10%), difficulty (30%), & pain (40%) ($p < 0.05$)
Green et al. (14) (1993)	47	66.8 yrs M & F	Hydrotherapy (H) &/or H & HEP	2 ×/wk (H) 2 ×/day HEP	6 wks	No signif. differ. in work done, power, or endurance between groups; a conclusion of no real need for hydrotherapy; a limitation was short duration of the study
Fisher et al. (6) (1993)	40	66.0 yrs M & F	Isometrics & progressive isotonic exercises	3 ×/wk	3 mos	Muscle strength signif. increased for hamstrings (9 & 19%) & quadriceps (8 & 24%) for men & women, respectively; walking time improved; difficulty & pain upon activity decreased ($p < 0.05$ for all listed variables)
Fisher et al. (7) (1994)	19	67.4 yrs M & F	Isometrics, flexibility, & isotonic exercises	5 ×/wk	3 mos	Signif. increases in exercise group of isometric strength at 45° KF and at 60° & 120° of HE (35%) ($p < 0.05$); no increases in functional capacity
Fisher & Pendergast (8) (1994)	12	63 yrs (F) 71 yrs (M)	Isometrics, flexibility	3 ×/wk	3 mos	29% increase in isometric KE strength at all tested angles, $p < 0.05$; increase in 90-sec endurance test for KE at all tested angles, $p < 0.05$
Schilke et al. (37) (1996)	20	64.5 yrs M & F	Isokinetic (90°/sec) 6 sets of 5 reps	3 ×/wk	8 wks	Signif. increases in exercise group in KF (30%), pain, mobility ($p < 0.001$), stiffness, & KE ($p < 0.01$)
Ettinger et al. (2) (1997)	439	69.6 yrs M & F	Calisthenics & isotonic exercises	3 ×/wk (1 hr)	18 mos	8% decrease in disability ($p = 0.003$), 8% decrease in pain score ($p = 0.02$), increase in 6-min walk distance ($p = 0.02$), and KF strength increase ($p = 0.02$)

KE = knee extension; HEP = home exercise program; H = hydrotherapy; HE = hip extension; KF = knee flexion.

Effects of Resistance Training Exercise

Decreased muscular strength, which has been shown in patients with OA, has been closely linked with immobility, frailty, and falls (4, 12, 42). It is important to determine whether OA patients can benefit from strength training programs, not only to increase muscular strength but also to possibly decrease the incidence of falls and/or fractures.

Table 2 summarizes recent resistance training studies that involved OA patients (2, 5, 9, 14, 31, 37). The results in all the studies except for the one by Green et al. (14) is that strength can increase significantly in OA patients. Training ranged from completely isotonic exercise protocols (2, 5, 31), isometrics and progressive isotonic exercises (6–9), to isokinetic exercises (37).

Further results included three studies showing not only increases in muscular strength but also significant decreases in pain (2, 6, 9, 37). The benefits of resistance training appear to go beyond strength enhancement. Other findings of significant decreases in dependency (2, 9), increased ease of movement (2, 9), and decreased stiffness and increased mobility (38) show that resistance training can be used to improve functional capacities in these patients.

Also, one strength training study (8) observed a significant increase in maximal treadmill walking speed from 2.0 to 2.4 mph ($p < 0.05$) and increased exercise time on the treadmill from 9.2 to 11.2 minutes. Further results displayed significant decreases in submaximal exercise heart rate and systolic blood pressure following the program. These results are appealing in that the level of aerobic capacity for osteoarthritic patients may be partly dependent on their muscular function, which suggests that increases in muscular strength can improve aerobic ability in patients with OA.

The results from the resistance training studies are informative but more research is needed. A major concern is that the studies cited here used a wide variety of resistance equipment and programs. Several programs had no set training regimen for each patient, but did mention that they were individualized for each patient's symptoms. Further studies should describe training regimens in detail.

Another limitation is the small sample size of most studies. Excluding the Ettinger et al. (2) study, the majority had some degree of attrition ranging from 1 to 18 subjects. However, in the aforementioned study (8) it should be noted that only 3 of the 40 patients did not finish the program because of discomfort ($n = 1$) or exacerbation of OA symptoms ($n = 2$). Unfortunately, concerning the Ettinger et al. study, 75 participants left the study and there was no explanation as to why; however, the retention percentages for the exercise and health education groups did not differ significantly. Regardless of these findings, it is obvious that more data need to be collected in a systematic manner to determine the effects of resistance training exercises.

Practical Application

Osteoarthritis is a degenerative disease that attacks articular cartilage and results in decreased muscular strength and aerobic capacity. Moderate levels of resistance training on the part of OA patients have been found to yield significant increases in strength, increased mobility, decreased stiffness, and even pain reduction. Furthermore, moderate exercise does not appear to increase the risk for OA or exacerbate it. Therefore exercise would seem warranted for persons with OA.

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