Effects of Exercise Interventions in Older Adults with Knee Osteoarthritis

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Received: 31 August 2011/Accepted: 2 December 2011/Published online: 18 January 2012
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Keywords aerobics · resistance training · nonpharmacologic OA trials

Introduction

Knee osteoarthritis causes a decrease in physical function that is associated with mobility impairment. Mobility impairment has the potential to accelerate decline from independent living to disability and assisted living. One goal of nonpharmacologic interventions is to flatten out the slope of the disability curve to provide older adults with knee osteoarthritis more years of independent living and a good quality of life.

Results of Clinical Trials Investigating the Role of Exercise in OA

The first large-scale, multicenter trial of exercise and knee osteoarthritis was the Fitness Arthritis and Seniors Trial (FAST) [1]. FAST compared two forms of exercise, aerobic walking, and weight training to a health education control group. The intervention was 18 months in duration and consisted of 3 months of center-based exercise followed by 15 months of home-based exercise. Intensity of the weight training exercise was low to moderate using loads that were in the range of 60% of each patient's one repetition maximum. Both intervention groups had significantly less self-reported disability (the primary outcome) than the control group after 18 months. In addition, they had less pain (Fig. 1); performed better on physical performance measures which included a 6-min walk, stairs climbing, lift and carry, and car tasks; and walked faster than the control group. Hence, FAST showed that low- to moderate-intensity exercise is a safe and effective nonpharmacologic therapy

that can slow the decline in physical function due to knee osteoarthritis and should be prescribed as part of the standard of care for older adults. That exercise has a larger effect size for pain than either acetaminophen or NSAIDs [9] also supports its use in standard arthritis care.

Weight loss is another important component of the nonpharmacologic approach to osteoarthritis patient care. The lifetime risk of painful knee osteoarthritis increases with increased levels of BMI [7]. The ADAPT clinical trial compared exercise and weight loss, both separately and in combination, to a healthy lifestyle control group and found that in spite of no difference in medial joint space width between the groups, the combination of a 5.7% weight loss plus exercise was superior to a control group in pain and self-reported function [5]. Mechanistic results favored weight loss in reducing knee joint loads and systemic levels of proinflammatory cytokines. Taken together, it appears that weight loss impacts both the mechanical and inflammatory disease pathways, but little evidence exists that low- to moderate-intensity exercise has any effect on these pathways.

Most exercise studies have used low to moderate intensity. Of great interest are the large effect sizes of the few high-intensity strength training studies which would use loads greater than 60% of the one repetition maximum [3]. A pilot study from our lab showed that 16 weeks of high-intensity strength training decreased pain and improved function compared to a low-intensity strength training group. The average participant in our current clinical trial (IDEA) [4] has a thigh volume that is 51% fat. Preliminary data show that total thigh fat is significantly related to knee joint loads and that this relationship is driven by thigh subcutaneous fat. Also, the greater the amount of quadriceps intramuscular fat, the greater is the knee joint loads. Hence, changing thigh muscle and fat mass may decrease knee joint loading. Taaffe et al. [8] showed that high-intensity strength training significantly decreases fatty infiltration of the quadriceps in 65- to 83-year-old adults. We suggest that high-intensity strength training can impact the mechanical disease pathway to knee osteoarthritis by

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Fig. 1. Pain level decreases both with weight training and with aerobic exercise. Adapted with permission from Messier et al. [6] Copyright © 1997 American Medical Association. All rights reserved

increasing thigh muscle function and decreasing thigh fat depots, resulting in long-term improvement in pain and function and possibly attenuate disease progression.

Summary

In summary, low- to moderate-intensity exercise effects are known: they reduce pain and improve function, but have no effect on disease progression. Primary and secondary prevention studies using nonpharmacologic interventions such as exercise and weight loss are needed to clarify how exercise protocols can retard or even prevent the onset of osteoarthritis. These studies should include clinical, mechanistic, and structural outcomes, such as assessments of pain and ambulatory function, measures of actual joint loads applied by the patients, and assessment of disease progression. The interventions should be long term (≥ 18 months), and retention and compliance are of the utmost importance [2].

Disclosures The author certifies that he has no commercial associations (e.g., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted article.

The author certifies that his institution has approved the reporting of these cases, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participating in the study was obtained.

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