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Editorial

Running-related Osteoarthritis Caused by a Combination of Joint Vulnerability and Joint Loading

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Editorial

Running is an excellent activity to promote general health and well-being. However, running-related injuries are common, and concern is sometimes raised that running might lead to osteoarthritis (OA) in weight-bearing joints [1-2]. Therefore, the benefits of participating in running need to be balanced against the risks of developing OA. Although the existing evidence on whether running causes OA is currently insufficient for researchers to draw unequivocal conclusions, the preponderance of data seems to indicate that moderate level, or recreational running do not increase the risk of OA of the knees and hips for healthy people and that this activity might even have a protective effect [3-13]. On the other hand, however, under many circumstances, running may be associated with an increased risk of developing OA [14-19].

Although much attention has been paid on the relation between running and OA risk, it remains uncertain about the cause of running-related OA. Since OA has long been considered a "wear and tear" disease leading to loss of cartilage, many healthcare professionals consider it as the major cause of exercise-related OA. This hypothesis predicts that any type of impact, such as running would increase OA, or worsen it once developed. Nevertheless, "wear and tear" alone may not be the cause of running-related OA. For one thing, recent evidence has led to a new view that OA pathophysiology should be perceived in the context of the entire joint with involvement of several tissues [20-21]. More importantly, the clinical evidence suggests that recreational running does not increase the risk of OA, or even is beneficial to cartilage health [3-13]. Shrier [22] thought OA in high intensity or elite runners could be due to a threshold effect, and "wear and tear" only occurs after a threshold.

In 1999, Hurley reviewed the basic science evidence (although largely indirect evidence) and proposed that motor and sensory dysfunction of muscle is the most important modifiable mediating factor for primary OA [23]. This muscle dysfunction hypothesis is based on the finding that muscle fatigue increases the impact forces crossing a joint [24-25], which suggests that properly contracting muscles are the main absorber of force. Later, after reviewing the published data about the association between sports activities and hip/knee OA, Shrier supported the contention that muscle dysfunction is the primary cause of exercise-related OA [22]. Nevertheless, direct evidence was not provided supporting or refuting either framework as causal. Furthermore, in addition to muscle, many other tissues, such as tendons, ligaments, joint capsule, and mechanoreceptors, are also responsible for protecting cartilage from injury during daily activities. OA often occurs slowly and is attributable not just to failure of one joint protector, but the failure of several in combination. Hence, as a multi-factorial disease, one mechanism alone might not fully explain the complex interaction between mechanical and neuromuscular factors in contributing to running-related OA [26].

OA has a multi-factorial etiology and can be considered the product of interplay between systemic and local factors. In a review, Felson [27] created a new paradigm for better understanding these risk factors, using joint vulnerability and loading as a framework, and clarified them as those increasing joint vulnerability (malalignment, muscle weakness, genetic and ethnic predispositions, aging), and those that cause excessive loading (obesity, certain physical activities). In this regard, it appears that running-related OA is a combined result of joint vulnerability and joint loading. As noted above, running may be associated with an increased risk of developing OA under many conditions, which may be due to the increased joint vulnerability and/or joint loading.

High level or competitive running: A number of studies focusing on ex-elite long-, or middle-distance runners suggested a link between running and OA risk [14-18]. Besides, a significantly higher incidence of OA was found in male recreational runners who were involved in high levels of running [19]. For one thing, high level running may induce high running-induced loading, which can be harmful to the joint, if magnitudes exceed the physiologic tolerance level for an individual. Secondly, high level running may be linked with a higher incidence of joint injury [28]. Thirdly, the high pain threshold [29] or the muscular fatigue in elite runners may lead to the impairment of the subtle synchronization between the felt impact and the muscle response, and thus a higher risk for OA [30].

Increasing age: With ageing, the deformation of femoral and tibial cartilage decreases during running [31], indicating that the collagen fibrils are more prone to fatigue failure [32-33]. Furthermore, ageing may also change kinematic and ground reaction force data during running, suggesting relatively poor shock absorbency [34]. Such changes may be reflected by the increased OA risk with increasing age in both recreational runners [10] and ex-elite runners [14].

Joint injury: It was suggested that the increased risk of lower limb OA in participants of repetitive and high impact sports is strongly associated with joint injury [35]. Specifically, a body of evidence demonstrated that the increased incidence of knee OA in the runners may be attributed to a higher incidence of knee injuries [16-18]. Previous joint injury may mediate the running–

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OA relationship due to its effects on several potential mediators of cartilage degeneration, including secondary muscular weakness, joint asymmetry, biomechanical malalignment, ligamentus instability, and neurological deficits [36]. The associated abnormal loading patterns, coupled with impaired muscle function to absorb force, result in degenerative changes of the joint [37].

Joint malalignment: Unlike those with normal joint mechanics and neuromuscular function, in persons with joint malalignment, high impact loading from running may increase particular contact stress and cause running-induced degeneration [38-39]. Not surprisingly, a significant association between degenerative changes and the presence of genu varum was reported [18].

Obesity: A number of studies confirmed that BMI can modify the relationship between running and OA risk [6, 10]. Obesity means greater loading on joints of lower limbs. Additionally, the added weight means that muscles must absorb even more force and therefore must be stronger and have greater endurance. Unfortunately, obesity is usually associated with physical inactivity and therefore relative muscle dysfunction [40]. Such 'relative muscle dysfunction' in obese/overweight runners may explain their high OA risk [22].

As stated above, factors often implicated in the development of running-related OA comprise those that increase joint vulnerability (including increasing age, previous joint injury, joint malalignment, etc) and those which increase joint loading (including obesity and high level running). Not surprisingly, with moderate loading, running-related OA unlikely develops on healthy joints. However, depending on the degree of joint vulnerability, moderate loading (recreational running and/or normal weight) may put vulnerable joints at risk for disease development. On the other hand, excessive loading (competitive running and/or overweight) may also put healthy joints at OA risk. This hypothesis should have important implications for OA prevention. Efforts should be made to identify those with joint vulnerability and joint loading, and measures should be taken to have those factors and/or their running programmes modified before commencing or continuing to run safely.

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