Muscle dysfunction versus wear and tear as a cause of exercise related osteoarthritis: an epidemiological update

Ian Shrier

There are two main hypotheses for the cause of exercise related osteoarthritis: wear and tear of the articular cartilage and muscle dysfunction. This is a review of the clinical literature to see which hypothesis has the greatest support. Clinical studies support the muscle dysfunction hypothesis over the wear and tear hypothesis.

Osteoarthritis (OA) often limits activities of daily living—for example, climbing stairs, dressing—and can prevent participation in the labour force for younger patients. Patients seek advice from family doctors, internists, rheumatologists, and orthopaedic surgeons.

Recently sponsored symposia in both the United States and Canada (co-sponsored by the Canadian Institutes of Health Research and the Canadian Arthritis Network, Toronto, Ont, April 2002) suggest that OA is a complex syndrome—that is, constellation of symptoms and signs with multiple causes—that involves the balance between cartilage synthesis and degradation, and affects all tissues surrounding the joint. That being said, the question remains as to which factors are directly related to the cause of OA and are modifiable so that doctors may counsel patients appropriately.

In the case of primary OA—that is, excluding genetic diseases, severe biomechanical abnormalities, post-septic arthritis, etc—many healthcare professionals believe the major cause of OA is “wear and tear”—that is, gradual thinning of the articular cartilage due to repeated weight bearing activity of the joints—and that therefore OA is caused and worsened by exercise. However, in 1999, Hurley reviewed the basic science evidence and proposed that properly contracting muscles are the main force absorber for the joint, and that muscle dysfunction is the most important modifiable mediating factor for primary OA. Because regular exercise improves muscle function, this hypothesis predicts that exercise would not increase the incidence of or worsen OA. Hurley also suggested that whereas the wear and tear hypothesis predicts that cartilage thinning will be the first sign of OA, the muscle dysfunction hypothesis predicts that sclerosis would be the first sign. Finally, in the case of injury, the muscle dysfunction hypothesis predicts that injuries to muscles in a leg may increase the risk of OA in joints not immediately adjacent to the injured muscle because impact forces are not being properly absorbed. The wear and tear hypothesis suggests that injuries would only increase the risk of OA if articular cartilage injury occurs at the time of injury, or is more likely to occur after injury—for instance, anterior cruciate ligament (ACL) instability. The specific objective of this systematic review is to determine the clinical evidence in support of and against the hypotheses that exercise related OA is caused by (a) wear and tear or (b) muscle dysfunction.

The reader should not forget that OA is multifactorial and that there are other causes of OA. As such, there are two important limitations to the scope of this article. Firstly, it focuses on both hip and tibiofemoral OA and does not discuss patellofemoral OA, or OA in other areas of the body. Secondly, regardless of the initiating event of OA in a particular patient, the articular cartilage is eventually destroyed. The mechanism of articular cartilage destruction is also beyond the scope of this article.

MATERIALS AND METHODS

A systematic review of the literature was carried out. Medline and SportDiscus databases were searched using the strategy (osteoarthritis or osteoarthrosis) AND (activity or exercise or injury). Based on titles and abstracts, all potentially pertinent articles were retrieved and reviewed. The bibliographies of all articles retrieved were reviewed for additional references, and a search of Citation Search Index was conducted to find any article that may have cited one of the key articles previously retrieved. Data were abstracted by one person using a standardised form, and verified with a second reading by the same person at least four weeks later. This review is limited to exercise related primary OA, and studies investigating OA secondary to injury or previous surgery were not included in the results.

Results are presented as odds ratios (OR) or relative risks (RR) or hazard ratios (HR) with 95% confidence intervals (95%CI) in parentheses unless otherwise specified. Because many studies lacked the necessary power to determine if the differences were statistically significant, relying on p values or confidence intervals might result in a Type II error (incorrectly indicating that the differences between groups were not important). Therefore, the emphasis in this review is on the

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Abbreviations: OA, osteoarthritis; OR, odds ratios; RR, relative risk; HR, hazard ratio; 95%CI, 95% confidence intervals; ACL, anterior cruciate ligament; Exp, exercise group; Con, control group
direction and magnitude of the changes—that is, are the changes clinically relevant?— rather than whether a study had significant results.

Because the clinical studies reported different outcomes, used widely differing methodology, etc, a qualitative synthesis was more appropriate than an attempt to provide an overall summary statistic for the estimate of the effect.

RESULTS

Twenty three clinical articles (representing 18 studies) related to exercise and OA were retrieved. Table 1 presents studies on running, table 2 presents studies on football, and table 3 presents studies on other sports. Where studies reported on more than one type of exposure, the relevant details are repeated under each section and the duplication noted.

### Table 1

<table>
<thead>
<tr>
<th>Article</th>
<th>Population</th>
<th>Design</th>
<th>Results</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Munt (n = 59) | Males: age range not reported. | Historical cohort with 15 yrs followup. Information obtained by recall. | Mean values | Response rate 27/27 runner, 9/12 bokelgh and 23/26 controls.
OA scored separately as joint space, sclerosis and osteophytes, and also as a composite score ranging from 0-9.
Although composite score worse for runners, the mean score was only 1.4 of a total score of 9. Still, 4/27 runners had joint space <3 mm whereas there were no bokelgh or control subjects with this limited amount of joint space.
Running pace was a better predictor of radiological hip OA.
Hip pain was present in 30% of the runners, but 0% of bokelgh or control groups. |
| Chang (n = 16,961) | Males (n = 12,888) and females (n = 4,073). Ages 20-87. | Historical cohort with mean (SD) followup time = 10 (8) yrs. Information obtained by survey. Self-reported exposure and outcome so no binding. | Hazard ratios for self-reported OA adjusted for BMI, alcohol, smoking, caffeine preference category and sedentary. | The inclusion of people as young as 20 for an outcome of OA may be inappropriate. The mean follow-up time suggests skewed distribution and we are not sure of minimum follow-up time. Some subjects in mod or high categories may have been exclusions instead of inclusion. |
| Speir (n = 1058) | Women, ages 40-65. | Historical cohort (1.5-45 yr followup). OA status by x-ray and exposure status by recall. | OR for sub vs controls for different joints (adjusted for age, height and weight). | Response rate 81/117 elite athletes and 977/1003 controls.
Adequate information on physical activity in controls available only in 585/977 controls.
OA assessed by joint space narrowing and osteophytes, but no total score. Among the control population with different levels of physical activity, the OR for joint space narrowing among the people with a past history of long-term physical activity was close to that of the elite athletes. The respective OR for osteophytes (among controls) was much higher than that for joint space narrowing. They were not reported here for space limitations. Note that the importance of osteophytes is not yet clear. |
| Vingled (n = 569) | Males, ages 50-70. Cases: Total hip replacement 2nd idiopathic OA in 4 Swedish hospitals [n = 267 partial participation, 323 partial participation]. Controls: Swedish men living in area of some 4 hospitals [n = 322 partial participation, 302 complete participation]. | Case-control Information by recall. Binding of evaluators for exposure not reported. | OR for total hip replacement among runners, adjusted for age, BMI, smoking and physical activity at work. | Response rate 233/253 [92%] for full participation among cases, and 302/392 [78%] among controls. Slightly greater partial participation rates for both groups. Relative risks estimated from OR. Exposure information obtained by recall during interview. Authors note that recall in myocardial infarction patients and controls faced no increased RR and they suggest that makes recall not a problem. However, if regular physical activity is protective against infarction, and recall not an issue, then physical activity should have been protective. |

**Table 1** Details of studies related to running exposure (95% CI in parentheses)
Overall, the three cross-sectional running studies concluded that exercise is not associated with OA,4–6 and the three case-control running studies found mixed results but overall suggested that some higher intensity activities may be associated with the development of OA.7–9

With respect to historical cohort studies on running, there was no increased risk of OA in runners in four of seven historical cohort studies. This was true for (a) 27 elite Danish male orienteering runners compared with hospital controls,10 (b) 60 Finnish male elite runners compared with hospital controls,11 (c) 504 US college varsity cross country runners compared with varsity swimmers,12 and (d) 1282 Finnish endurance athletes and 777/1010 (77%) controls. The total number of approached subjects (n = 2528) represented 60.8% of original cohort (39.2% died before start of study).

In one study showing a possible increased risk of OA in runners,16 running pace was a better predictor than running mileage even though the wear and tear hypothesis would predict that OA should increase with each vertical impact—that is, step—more so than horizontal speed. Horizontal speed would be important if the running technique was suboptimal, and the runner placed the foot in front of the body at heel strike, thereby creating a large braking force.
therefore would not be expected to correlate with running speed.

Another historical cohort study suggested an increased risk in runners younger than 50 who run >20 miles a week. An effect of mileage was not seen in subjects older than 50, which again is contrary to what would be predicted by the wear and tear hypothesis. In the remaining study showing a possible increase in OA, osteophytes were associated with elite exercise, but the OR for joint space narrowing was close to 1 for the knee (1.2, 95% CI 0.7 to 1.9) and for the hip (1.6, 95% CI 0.7 to 3.5). Within the control population, moderate exercise was not associated with joint space narrowing of the hip or knee, although there was a trend toward decreased joint space of the hip in the higher participation category (1.8, 95% CI 0.73 to 3.48).

In the only prospective study, Lane and colleagues found no difference in the development or progression of OA between 41 runners and matched controls after two, five, or nine years. In another study that simply categorized exposure as “sport participation”, there was again a lack of progression of OA.22

Besides pure running, team sports such as soccer have also been implicated as a cause of OA. Although Klunder et al23 found a higher proportion of radiographic hip OA in soccer players, 13/30 patients with OA had previous injuries compared with only 3/19 controls. Lindberg et al24 found hip OA was higher only in the elite soccer players (14.1% for elite, 4.2% for non-elite, and 4.2% for control).

In summary, these findings suggest that moderate intensity impact sports do not cause or worsen OA. OA in high intensity or elite sports could be due to a threshold effect—that is, wear and tear only occurs after a certain level, and therefore the “matched” controls would have lower occupational loads than normal population.

Table 1 Contd

<table>
<thead>
<tr>
<th>Study</th>
<th>Mean age</th>
<th>Men: 48/25 females</th>
<th>Women: 45/24 females</th>
<th>Cross-sectional OR for OA of hip due to sport</th>
<th>OA grade on loss of joint space, sclerosis and osteophytes</th>
</tr>
</thead>
<tbody>
<tr>
<td>OA</td>
<td>Structures</td>
<td>Osteophytes</td>
<td>Hip cartilage (mm)</td>
<td>Degeneration in knee (%)</td>
<td></td>
</tr>
<tr>
<td>Runners</td>
<td>7.6 (5.7)</td>
<td>4.6 (3.8)</td>
<td>4%</td>
<td>6%</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>8.4 (4.6)</td>
<td>4.9 (2.5)</td>
<td>13%</td>
<td>17%</td>
<td></td>
</tr>
<tr>
<td>Knee OA was all Grade 1, and control was split equally between Grades 1–3. There was no degeneration in the hips or ankles of either group.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>Hip</td>
<td>Knee</td>
<td>Ankle</td>
<td>Foot</td>
<td></td>
</tr>
<tr>
<td>Runners</td>
<td>23%</td>
<td>29%</td>
<td>12%</td>
<td>5%</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>11%</td>
<td>22%</td>
<td>5%</td>
<td>0%</td>
<td></td>
</tr>
</tbody>
</table>

In the only prospective study, Lane and colleagues found no difference in the development or progression of OA between 41 runners and matched controls after two, five, or nine years. In another study that simply categorized exposure as “sport participation”, there was again a lack of progression of OA.22

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Elite sports

Kuhla’s group13–15 found that the risk of hip or knee disability was only increased in elite team sports (previous injury not controlled for). When the same cohort of athletes was compared with 1403 controls without controlling for previous injury,14 OA was increased in all types of athletes (OR range 1.73–2.17), but the greatest increase occurred in wrestling (OR 2.73, 95%CI 1.63 to 4.64), weight lifting (OR 2.74, 95%CI 1.27 to 5.9), soccer (OR 2.1, 95%CI 1.2 to 3.8), and ice hockey (OR 4.2, 95%CI 2.2 to 8.0). Three of four of these exposures do not involve impact, suggesting that wear and tear is not a likely cause. In a subsequent study of a subgroup of the same population but now controlling for previous injury,15 the risk was now considerably less (OR 1.2, 95%CI 1.0 to 2.3) and much less than the risk associated with previous injury (OR 6.0, 95%CI 1.3 to 27.8). The presence of previous injury may also partially explain the higher rate of OA in the previously mentioned Lindberg study.24 Using the same population, the
subsequent publication noted that 33% of elite soccer players with previous meniscectomy or ACL tear developed knee OA compared with 11% in those without these injuries. The same may also be true for hip OA, but this type of analysis has yet to be published.

**DISCUSSION**

The results of this literature review strongly suggest that regular mild-moderate impact exercise does not increase the risk of OA, and that there is some evidence that it does not increase symptoms in patients with mild-moderate OA. This evidence supports the muscle dysfunction hypothesis as a cause of OA over the wear and tear hypothesis.

Running/soccer

The wear and tear hypothesis predicts that any type of impact such as running would increase OA, or worsen it once developed. However, the clinical evidence suggests that recreational running and soccer do not increase the risk of OA. In the basic science literature, canine cartilage adapts favourably to moderate running, and running did not worsen immobilisation induced OA in rabbit knees, which is consistent with the prospective study reported by Lane et al. In addition, the finding that degeneration occurs with forced exhaustive running in dogs is also consistent with the muscle dysfunction hypothesis because exhaustion will prevent the muscles from absorbing force. Although some might believe that marathon running could be analogous to forced exhaustive exercise in dogs, most marathon training is done at much lower mileage. Although subjects may be tired, they are not exhausted. The actual marathon is run only a few times a year, whereas the dogs were run to exhaustion regularly.

Most of the subjects in the clinical studies in this review had intact menisci, and presumably no major malalignment. In subjects with previous meniscectomy, Roos et al reported no effect of exercise on the incidence of OA. This contradicts the basic science finding that running increased the risk of OA in meniscectomised sheep. Although there were no studies on the effect of exercise in subjects with malalignment, Sharma et al reported that disease progression occurs more rapidly in this population. How does the muscle dysfunction hypothesis relate to these populations? The wear and tear hypothesis predicts that cartilage damage precedes bone sclerosis. However, the reverse occurred in adult rabbit knees subjected to one hour impulse loading a day. The sclerosis was associated with numerous healing trabecular fractures, suggesting that the principle force absorber in anaesthetised animals is not cartilage but bone. This is supported by in vitro findings suggesting that articular cartilage does not absorb force, but does redistribute force. If enough microtrabecular damage occurs over a short period of time, sclerosis would occur as an adaptation—that is, damage would be less likely in sclerotic bone. Within this paradigm, malalignment and meniscectomy could increase the risk of OA because they prevent the normal redistribution of force—that is, even in normal knees, the muscles do not absorb 100% of the force—which makes micro-damage more likely to occur. Finally, the sclerotic changes in underlying bone stiffness may increase the stress on articular cartilage, which would lead to increased degenerative changes in both meniscal and articular cartilage.

**Elite sports**

Although the findings suggest that recreational sports are innocuous with respect to developing OA, they do suggest that participation in elite sports increases the risk of OA. This

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Table 2 Contd

<table>
<thead>
<tr>
<th>Lindberg9 (n = 858)</th>
<th>Males, ages 40–88</th>
<th>Cross-sectional study but exposure likely preceded outcome by 15–23 yrs</th>
<th>% Hip OA</th>
<th>Response rate not given.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exp: soccer players, with 7/2946 being elite</td>
<td>Information obtained from hip exams over 38 yrs</td>
<td>Older group</td>
<td>Younger group</td>
<td></td>
</tr>
<tr>
<td>Con: 572 males from same city population records, matched for age (soccer history unknown).</td>
<td>Binding of evaluators not reported.</td>
<td>14.7%</td>
<td>13.5%</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nonelite</td>
<td></td>
<td>3.1%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control</td>
<td></td>
<td>6.1%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Salomon6 (n = 76)</th>
<th>Males, ages 18–37 for Exp and 18–68 for control.</th>
<th>Cross-sectional as all Exp group still active soccer players.</th>
<th>Hip No OA in either group.</th>
<th>Response rate 36/60. As they were currently active and the reason for refusal was not provided, the subjects who refused may have been trying to hide their injuries.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exp: 76 active soccer players with 5–23 yrs experience (many national teams).</td>
<td>Information obtained by recall</td>
<td>Knee 28% of soccer players mild/moderate OA at Patella joint</td>
<td>Fem-Tib</td>
<td>Fem-Pat</td>
</tr>
<tr>
<td>Con: 40 subjects with acute lower extremity injury and no known history of soccer.</td>
<td>Binding of evaluators not reported.</td>
<td>Exp</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fem-Pat</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Talocrural</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>

73% of soccer players had moderate/severe knee injuries, and 80% had severe ankle injuries.

All studies suggested soccer players were at risk of OA, but where studied, only in elite sports or those without injury. Studies are sorted by design.

- OA: osteoarthritis
- Exp: Exercise group
- Con: Control group

- Historical cohort: For the purpose of this review, any observational study for which the exposure clearly predates the outcome was considered either a cohort study (groups assembled based on exposure history) or a case-control study (groups assembled based on presence or absence of OA). Although some historical cohort studies might be considered cross-sectional studies by other authors (e.g. when all information obtained by interview at one point in time), we felt that the important distinction between these two categories is the ability to determine if the exposure preceded the outcome (i.e. historical cohort study).
occurred in impact sports, such as soccer, and also in non-impact sports, such as weightlifting and hockey. Unlike the wear and tear hypothesis, the muscle dysfunction hypothesis predicts these results through the increased risk of injury that occurs with elite sports and the subsequent muscle dysfunction that occurs with injury. In support of these findings, others have found that young adults with previous knee injury are more likely to develop OA, and previous hip injury increases the risk of hip OA.

There are three possible mechanisms by which previous injury could increase the risk of OA. Firstly, the damage may occur at the time of the injury and OA develops over the subsequent years. Secondly, the associated ligamentous instability with injury leads to recurrent articular cartilage damage. Finally, the associated muscle dysfunction with injury leads to recurrent articular cartilage damage because the impact forces are no longer being absorbed appropriately.

If damage occurred at the time of injury, and the wear and tear hypothesis is correct, then articular cartilage damage should occur early. In support of this finding, others have found that young adults with previous knee injury are more likely to develop OA, and previous hip injury increases the risk of hip OA.

The possibility of "sub-clinical articular damage" remains theoretical at the present time. Finally, Felson et al recently found a strong correlation between location of bone marrow oedema on magnetic resonance images and progression of OA. If bone marrow oedema is indeed a strong predictor of progression, it suggests that bone injury is an early sign of damage. Future research should
examine the subgroup of patients who had osteophytes without joint space narrowing at baseline to see if bone marrow oedema preceded the articular cartilage damage.

If ligamentous instability of the joint after an ACL tear causes OA, then ACL reconstruction should minimise the risk of OA. However, clinical studies (albeit with their limitations) suggest that it may not.43 44 Other authors have suggested that it is the underlying bone injury that occurs at the time of ACL rupture that is the cause of OA. Yet, OA is produced in dogs and cats by isolated transection of the ACL without associated bone damage at the time of injury.36 45 46 In the muscle dysfunction hypothesis, the loss of proprioception information from the ACL would result in increased force transmission to the bone, and increased OA. Further, evidence from biomechanical studies reveal an increase in loading of the non-transected knee, which does not develop OA,77 which again suggests that normal muscles can absorb the regular amounts of stress and strain across a joint and that “wear and tear” is not the cause of OA in uninjured limbs.

The muscle dysfunction hypothesis is based on the finding that muscle fatigue increases the impact forces crossing a joint,48 49 which suggests that properly contracting muscles are the main absorber of force. Whether the muscle cannot contract properly because of age or fatigue or disuse atrophy, or because of injury induced weakness (strains) or loss of proprioception (ACL tears), the effect is the same; more force is transmitted to the bone, which leads to increased microtrabecular damage, which leads to sclerosis, which could lead to changes in the stresses and strains across the articular cartilage, and then joint space narrowing. The added stress would then lead to the characteristic changes observed in periarticular tissue. Note that this hypothesis would predict an increased risk of OA with less severe injuries than are usually accounted for in studies—for example, quadriceps contusion could lead to increased risk of OA even though there was no ligamentous damage—and also the greater risk of hip OA compared with knee OA in soccer players23—that is, groin strains occur often in soccer but rarely with running. In addition, it would predict a higher rate of hip OA in

### Table 3

<table>
<thead>
<tr>
<th>Study</th>
<th>Method</th>
<th>Participants</th>
<th>Cohort</th>
<th>Exposure</th>
<th>Outcome</th>
<th>Results for activity not shown. Article does say &quot;other risk factors for OA in this study.&quot;</th>
<th>OR for OA (adjusted for age, sex, BMI, knee pain at baseline and Heberden’s nodes)</th>
<th>Response rate not reported.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rosen6</td>
<td>Exp: 71 elite male soccer players, Exp: 215 non-elite male soccer players.</td>
<td>Men: 40-88,</td>
<td>Historical cohort with 15-63 yr follow-up.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>There was no standard follow-up and not all subjects had x-rays. Rather, x-ray records were pulled from radiology sources (only 253/858 had x-rays). OA defined by joint space narrowing &gt;50% of other knee compartment or contralateral knee, or joint space less than 3 mm. The lower prevalence of OA in non-elite compared with elite suggests higher rates of OA in soccer mostly only in elite players. The difference in injury rates may be the mechanism (only ACL and meniscus recorded).</td>
</tr>
<tr>
<td>Cooper22</td>
<td>Exp: westley participation in sports for at least 10 yrs after leaving school</td>
<td>Males (n = 99) and females (255) with mean age 75 6 yrs (BOE = 69 5-80 9).</td>
<td>Historical cohort with 5 yr follow-up.</td>
<td>Exposure information obtained by interview and outcome by x-ray.</td>
<td></td>
<td></td>
<td></td>
<td>Response rate 107/123 in Exp group. For control group, 214 originally designated but 16 excluded. Of remaining, 198, 83 refused initially (leaving 115). Of those remaining, 40 excluded as double-contacts, six refused to continue and 1 was excluded. OA required joint space narrowing. Power analysis suggested only needed 60 controls.</td>
</tr>
</tbody>
</table>

Studies are sorted by whether the results suggest an association between exercise and OA, and by study design within each category.  

- **OA:** osteoarthritis  
- **Exp:** Exercise group  
- **ACL:** anterior cruciate ligament  
- **Con:** Control group

### Table 3 Cont'd

<table>
<thead>
<tr>
<th>Category</th>
<th>% OA Total group</th>
<th>% Unjured</th>
<th>% Major surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elite</td>
<td>15.5%</td>
<td>11%</td>
<td>9%</td>
</tr>
<tr>
<td>Nonelite</td>
<td>4.2%</td>
<td>3%</td>
<td>2%</td>
</tr>
<tr>
<td>Control</td>
<td>1.6%</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>

OA defined by joint space narrowing >50% of other knee compartment or contralateral knee, or joint space less than 3 mm. The lower prevalence of OA in non-elite compared with elite suggests higher rates of OA in soccer mostly only in elite players. The difference in injury rates may be the mechanism (only ACL and meniscus recorded).
subjects with knee injuries, and vice versa because the muscles of the thigh would be expected to absorb force across both joints. However, this analysis has not yet been published.

Other activity and obesity

The objective of this article was to assess the risk of OA with exercise. Although a detailed discussion of the risk of OA with exposure to various occupations is beyond its scope, the muscle dysfunction hypothesis can explain findings in this area as well. Briefly, if a person is forced to work when fatigued or injured—for example, a farmer—the muscles no longer absorb the forces crossing a joint and there would be an expected increase in microtrabecular damage, then sclerosis, and then OA. For example, in the study by Lau et al., for those subjects with occupational exposures that required climbing 15 flights of stairs or more, the OR for developing OA was 5.1 (95% CI 2.5 to 10.2) for women and 2.5 (95% CI 1.0 to 6.4) for men in the entire study, but 34.0 (95% CI 4.7 to 248.4) overall for those with previous injury. Similarly, the OR for developing OA in those subjects with occupational exposures that required lifting ≥10 kg more than 10 times a week was 2.0 (95% CI 1.2 to 3.1) for the entire group and 25.9 (95% CI 8.1 to 82.4) for those with previous injury.

Finally, obesity is a well recognised risk factor for OA. The muscle dysfunction hypothesis explains this relation as well. The added weight means that muscles must absorb even more force and therefore must be stronger and have greater endurance or there will be a “relative dysfunction”. However, obesity is associated with physical inactivity and therefore relative muscle dysfunction. With respect to mortality, most of the evidence suggests that obesity is not related to mortality if there is adjustment for physical fitness. Future studies should explore whether the relation between obesity and OA is similar to that between obesity and mortality.

CONCLUSIONS

The muscle dysfunction hypothesis that was originally proposed based on basic science evidence is supported by the clinical literature as well. This includes:

- Strengthening and endurance exercise relieves symptoms in patients with mild and moderate OA, and poor knee proprioception is associated with increased disability in patients with OA.
- Regular running increases joint space width whereas forced exhaustive running—that is, fatigue—decreases joint space width.
- Major injuries are associated with a high rate of OA.
- Because muscles provide the “dynamic” joint stability during movement, some signs of OA—that is, osteophytes and capsular thickening—may be an attempt by the body to increase joint stability in the presence of muscle dysfunction induced dynamic instability.
- A wide variety of elite sports, but not recreational exercise, are associated with OA. This effect is greatly reduced when major injuries are controlled for. Because elite athletes often play while injured—that is, on weak muscles—the muscle dysfunction hypothesis predicts that there would still be an increase in risk if minor injuries are not controlled for—for example, groin strain in soccer and hip OA.

The most important implication of the muscle dysfunction hypothesis is that proper rehabilitation after an injury may be important in the prevention of OA. A study designed to definitively test the role of muscle dysfunction would require detailed prospectively collected data, controlling for proper rehabilitation after major and minor injuries using appropriate strength testing and close supervision. That being said, the hypothesis that best explains the evidential relation between exercise and OA currently available today is the muscle dysfunction hypothesis.

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Ian Shrier

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