Obesity and Vocational and Avocational Overload of the Joint as Risk Factors for Osteoarthritis

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As we examine the epidemiology of osteoarthritis (OA) and, in particular, the relationship of vocational and avocational activities and of obesity to OA, it is important to define our terms: I discuss both radiologic OA and symptomatic OA. These are by no means identical. Radiographic OA is often the focus of epidemiologic investigations and is usually defined as the presence of a definite osteophyte on the radiograph. The problem is that some subjects with radiographic OA have symptoms and others do not. Obviously, of greater interest to clinicians and those interested in public health is the entity of symptomatic OA, which is usually defined as the presence of symptoms and radiographic evidence of OA.

How do we position vocational and avocational loading of the joint and obesity as risk factors in the broad context of the etiology of OA? As shown in Figure 1, a variety of systemic factors affect the vulnerability of a joint to development of OA: e.g., age, sex, genetic susceptibility, or nutritional factors. These may, themselves, cause systemic problems or they may increase the local vulnerability of the joint to damage from, e.g., meniscectomy, periarticular muscle weakness, varus-valgus malalignment, or a proprioceptive defect. The combination of systemic and local vulnerabilities may lead to OA. Loading factors act on this vulnerable joint, among the best studied of which are vocational and avocational joint use and obesity.

This discussion will focus on 2 loading factors — vocational and avocational physical activity – and their effect on the propensity of the knee or hip to develop OA. I will present evidence to support the following hypothesis: in a vulnerable joint, participation in sports and even normal daily activities may lead to OA because the vulnerable joint is incapable of withstanding such activities. In contrast, if the joint is normal, a great deal of activity — perhaps more than most of us subject our joints to on a regular basis — is required to cause joint damage and disease. What is the evidence to support this hypothesis?

The Chingford study[1] examined middle-aged women in England who were considered to have joints that were not vulnerable to OA. Odds ratios for osteophytosis and joint space narrowing, 2 individual features of OA, were not significantly increased for any of 3 types of physical activity: walking, vocational activity, or participation in sports, i.e., none of these activities was associated with an increase in OA.

What about running? Are normal joints capable of withstanding running without developing OA? The answer is, generally, yes. Several studies published between 1985 and 1998, some with a duration of followup longer than others, but all employing a control group of non-runners, examined the relationship between running and knee OA[2]. Generally speaking, among recreational runners, the risk of knee OA in these studies was not increased. However, 2 studies of elite, nationally competitive runners — people in whom running was essentially an occupation — found an increase in the prevalence of knee OA in those subjects, in comparison with non-runners[3,4]. Thus, beyond a certain level of intensity, running may become a problem, i.e., the ability of the normal knee to withstand repeated impulsive loading may be exceeded.

As for hip OA, although several investigators have found no increase in OA among elite runners, Puranen, *et al*[5], Marti, *et al*[6], and Vingard[7] noted that hip OA was more prevalent in runners than in the controls. Further, in studies by Vingard, *et al*[8] and by Kujala, *et al*[9], even recreational running increased the risk of hip OA, albeit modestly.

What is the effect of running on a vulnerable joint? In a small uncontrolled study, McDermott and Freyne[10] described 20 runners with knee pain, all of whom were in their thirties. Six had radiographic evidence of knee OA, 4 of whom had already had a history of major joint injury, indicating they were running on a joint whose vulnerability to OA was increased. Thus, running at a level of activity that would not have an adverse effect on a normal joint may lead to OA if the joint is vulnerable.

How much running is needed to cause OA? In a study in which thousands of men and women were put through a rigorous evaluation of their physical capacity and then followed over time for development of self-reported OA, Cheng, *et al*[11] found that as the level of running increased, so did the risk of knee OA. For both men and women, running more than 20 miles per week increased the risk. In a review of data on the risk of knee or hip OA with sports participation, Lane and Buckwalter[12] found no association between recreational athletics and OA in the absence of injury. However, athletic competition seemed to increase the risk of OA (Table 1).

What is the relationship between vocational activity and

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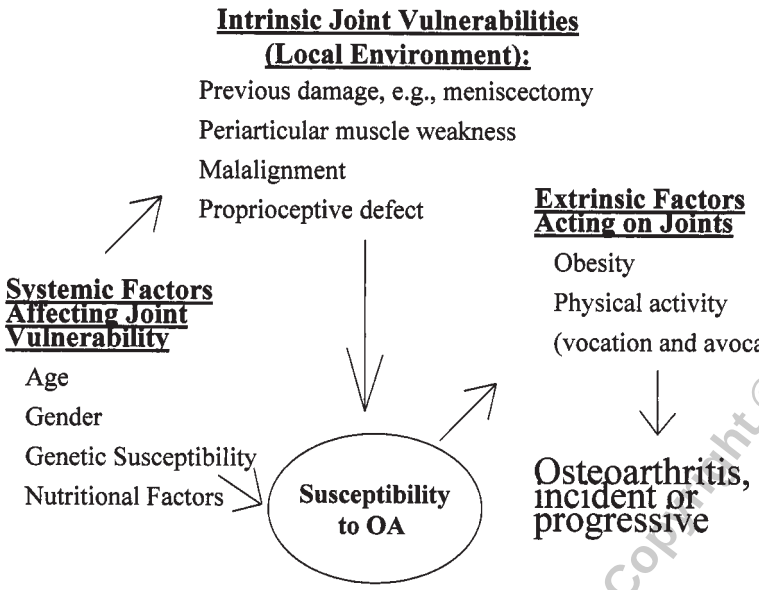

Figure 1. Concept of etiopathogenesis of osteoarthritis.

Table 1. Participation in sports related to high risk of knee/hip OA. Data from Lane N, Buckwalter J. Curr Opin Rheumatol 1999;11:413-8.

	Recreational Activity	Participation in Competitive Sports
Normal joints		
Distance running	Probably No ↑	Probably ↑
Soccer	Slight ↑	↑↑
Racket sports	Probable ↑	↑↑
Dystrophic and/or injured joint		
All sports	↑↑	↑↑

OA? In most cases, when we consider the relationship of vocational activity to OA it is reasonable to assume that joints are essentially normal at the start of the vocational activity, rather than being inherently vulnerable to OA. With activities that are performed day in and day out, 8 hours a day, 40 hours a week, 52 weeks a year, over many years, however, the repetitive activity may exceed the point at which muscles protecting the joint fatigue. A number of studies have shown that repetitive activities of laborers lead to OA (e.g., in finger joints of cotton mill workers; knees and spines of miners; hip joints of farmers; and elbows, wrists, and metacarpophalangeal joints of jackhammer operators).

To understand how activities might lead to OA, it is helpful to examine some relevant biomechanical data. McGibbon *et al*[13] described a patient who at the time of total hip arthroplasty was fitted with a prosthesis that contained multiple pressure sensors and was then studied postoperatively in the gait laboratory under a variety of experimental conditions. Figure 2 illustrates the pressures measured at various topographic sites on the prosthetic hip when the subject carried a 10 lb load. The message is simple: merely placing a 10 lb weight in either hand produced an increase in pressure in the hip.

The contact area of the tibiofemoral compartment changes in relation to the angle of knee flexion: with increasing flexion, e.g., squatting, as required in many occupational activities, the load across the joint is concentrated on a diminishing area of the articular surface, leading to an increase in stress[14].

Does vocational activity lead to OA? Coggon, *et al*[15] suggested that lifting, kneeling, and squatting increase the risk of knee OA, in contrast to occupational activities that do not increase the load on the knee, e.g., sitting (Table 2). A job that requires kneeling, squatting, and heavy lifting over many years increases the risk substantially. Notably, this is not merely an issue of radiographic OA: among people exposed to a medium or high level of force acting on their joints, the risk of obtaining a disability pension is dramatically increased in comparison with that in subjects with low exposure (Table 3). This results in a substantial cost to society.

In summary, with respect to the relationship of occupation to OA, specific activities increase the risk for hip and knee OA. Ergonomic modification of the workplace may lessen the societal burden of OA, but the payoff for any change in the job may be difficult to detect because OA develops slowly. Hence, any alteration in vocational activities that lowers the risk of OA might not produce obvious results for years. However, the fact that OA is likely to be a compensable disorder and is expensive may promote further research in this area.

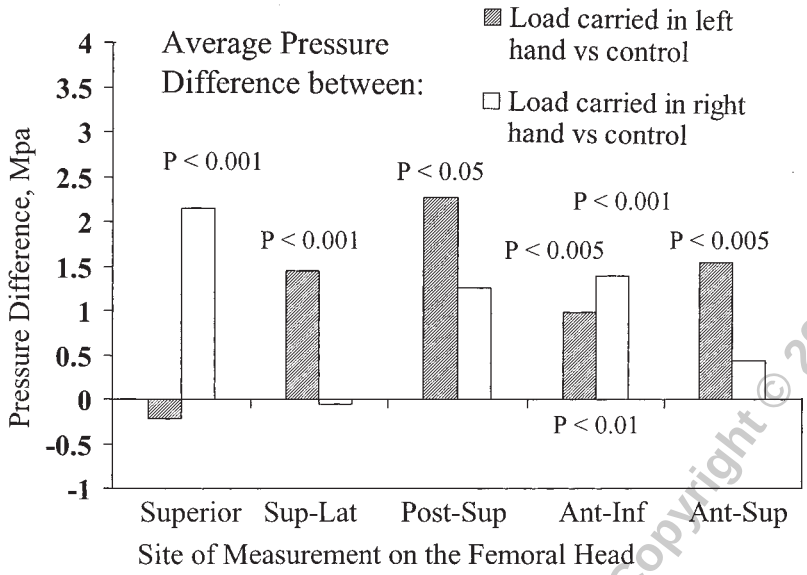

Figure 2. Effect on hip joint stresses of carrying a load (10% body weight), as detected by sensors in the prosthesis of a patient who underwent arthroplasty of the left hip. Sup-Lat: Superolateral, Post-Sup: posterosuperior, Ant-Inf: anteroinferior, Ant-Sup: anterosuperior. With permission from McGibbon C, *et al*. Arthritis Care Res 1997;10:300-7.

Table 2. Association of specific occupational activities with knee OA. With permission, from Coggon D, *et al*. Occupational physical activities and osteoarthritis of the knee. Arthritis Rheum 2000;43:1443-9.

	OR (95% CI)
Lifting ≥ 25 kg more than 10 times/week	1.7 (1.2, 2.6)
Sitting ≥ 2 h/day	0.7 (0.5, 1.0)
Kneeling > 1 h/day	1.8 (1.2, 2.6)
Squatting > 1 h/day	2.3 (1.3, 4.1)
Walking > 2 miles/day	1.9 (1.4, 2.8)
Climbing ladder or stairs > 30 times/day	1.5 (1.0, 2.3)

OR: odds ratio; CI: confidence interval.

Table 3. Risk of a disability pension due to musculoskeletal disorders for persons with medium and high level occupational exposure to forces acting on hip, knee, neck/shoulder, and low back. Comparison with the risk in those with low level of exposure.

Diagnosis	Medium Exposure RR (95% CI)	High Exposure RR (95% CI)
OA of hip	4.1 (2.4–7.1)	12.4 (6.7–23.0)
OA of knee	4.5 (2.6–7.6)	14.3 (8.1–25.4)
Low back disorders	2.3 (1.5–3.4)	8.6 (6.0–12.2)
Neck/shoulder disorders	1.3 (0.7–2.3)	9.4 (5.4–16.4)

RR: relative risk; CI: confidence interval.

What are the effects of obesity on knee and hip OA? Table 4 summarizes the results of a study in which we examined data from the National Health and Nutrition Examination Survey (NHANES)[16]. It depicts the prevalence of radiographic evidence of OA in women in various age groups in relation to body weight. Fifty percent of older women who were obese — but almost none of the relatively young women of normal weight — had radiographic evidence of OA. The effect of weight is dramatic and is seen in every cross-sectional study addressing this issue.

Table 4. Obesity and knee OA in Caucasian females. Percentage of subjects with radiographic knee OA (K-L grade ≥ 2).

	Weight Status		
	Normal, %	Overweight, %	Obese, %
Age, yrs			
25-34	0	0.3	2.2
35-44	0	0.3	11.1
45-54	0.5	1.9	13.2
55-64	2.6	5.2	17.5
65-74	5.8	17.7	49.0

With permission, from Anderson, *et al*. Am J Epidemiol 1988;128:179-89.

One of the questions about such cross-sectional data, however, is that of cause and effect. What about the possibility that the subject who developed symptomatic OA became sedentary for that reason and then became obese because of inactivity? To answer this question, we used the Framingham database to examine longitudinally the relationship between baseline weight and the development of OA in serial radiographs, based upon the development of new osteophytes. In women, for every 5 unit increase in body mass index (BMI), the odds of incident OA increased by 1.8. In men, however, no association between BMI and

incident OA was apparent. This is true also in other studies; the relationship, if one exists, is not nearly as strong in men as it is in women. We found that a change in weight was also a significant risk factor. If the subject gained weight, the risk of incident disease increased; if she lost weight, the risk diminished. Data from longitudinal studies of people with OA indicate that obesity increases the risk of radiographic progression in subjects who already have knee OA. Thus, it is a risk factor not only for incident OA but also for OA progression.

Does weight loss by an obese subject with knee OA benefit the joint disease? A definitive answer to this question is not available, despite recommendations that the treatment of OA incorporate weight loss. A small placebo-controlled study of an appetite suppressant showed that weight loss was associated with symptomatic improvement in patients with knee OA but not hip OA. A small trial of exercise and diet versus exercise alone showed greater improvement in ambulation in the group treated with diet alone. Finally, in an uncontrolled study of morbidly obese individuals who underwent a gastric stapling operation followed by weight loss of 100 pounds or more, the prevalence of joint pain was strikingly reduced. In the aggregate, these data suggest that dramatic weight loss may have a great effect on OA symptoms. Whether this is true also for less dramatic weight loss is unclear. Evidence of a relationship between obesity and hip OA is not nearly as convincing as that for the relationship to knee OA. Studies of the NHANES data by Tepper and Hochberg[17] and by Nevitt, *et al* from the Study of Osteoporotic Fractures[18] indicate that the risk for the association of obesity with unilateral hip OA is unimpressive, although some relationship appears to exist in patients with bilateral radiographic hip OA.

If we consider *clinical* hip OA, a clearer picture begins to emerge from the analysis of several studies. In general, odds ratios are ≥ 2, suggesting that the risk of clinical hip OA is increased among those who are obese. If the analysis is restricted only to subjects who have incident total hip arthroplasty, the risk is much greater.

Thus, people who are overweight are clearly at increased risk of developing knee OA and, to a lesser extent, hip OA. Weight loss may prevent OA in these joints and may be effective treatment for those who already have disease.

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