

Osteoarthritis Revisited: A Contemporary Review of Aetiology

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ABSTRACT Osteoarthritis is among the most common pathological conditions in skeletal collections and is the most frequent musculoskeletal disorder in contemporary populations. Jurmain (1991) has previously published in this journal a brief review of skeletal perspectives on osteoarthritis. Subsequent studies by osteologists and medical researchers have added considerably to understanding of the aetiology and patterning of osteoarthritis. Thus, it is timely to present an updated review that expands and supports conclusions discussed in the earlier review. In short, osteoarthritis aetiology is multifactorial, with age being the main influence on the onset and severity of osteoarthritis. Genetic influences also play a large role in the severity of osteoarthritis, especially in the lower limbs. Weight, although playing a significant role for modern populations, seems to have had very minimal effects on prehistoric populations. Sex differences may often be a consequence of hormones, body size and anatomy, rather than activity related. Finally, intense activity starting at a young age still may influence osteoarthritis, especially in the upper limbs. Future directions discussed include within-body comparisons, animal studies, and examining patterns in large populations. Copyright © 2007 John Wiley & Sons, Ltd.

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Introduction

Other than dental diseases, osteoarthritis is the most ubiquitous pathological condition in skeletal collections. Approximately 15 years ago, one of us (Jurmain, 1991) published in this journal a brief review of skeletal perspectives on osteoarthritis. Because so many further studies have since been conducted by osteologists and medical researchers, many of which have added considerably to our understanding of the aetiology and patterning of this condition, we feel it is timely to present an updated review.

'Osteoarthrosis' is frequently used as the preferred terminology, although in North America, 'osteoarthritis' is more widely used. This latter terminology has been questioned because it implies an inherently inflammatory condition. Given that inflammation was traditionally *not* viewed as a primary aspect in the pathogenesis of the condition, alternative terms such as 'osteoarthrosis' or 'degenerative joint disease' were suggested (Bennett *et al.*, 1942; Hough, 1993). However, there has been criticism of this latter usage also (Dieppe, 1987). In addition, clinical perspectives relating to the nature of the disease are shifting; a large proportion of contemporary researchers now regard inflammation as 'crucial to the pathogenesis of OA' (Punzi *et al.*, 2005). In sum, while there is still not full consensus regarding the most appropriate terminology, based on the most common usage, as well as

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new insights concerning aetiopathogenesis, we prefer 'osteoarthritis'.

Biological anthropologists have studied osteoarthritis extensively. Studies of archaeological samples have focused primarily on peripheral joints, but several investigations of the spine have also been completed (e.g. Hooton, 1930; Anderson, 1963; Chapman, 1972; Swedborg, 1974; Jurmain, 1990; Bridges, 1994; Knüsel *et al.*, 1997). For comprehensive reviews of osteoarthritis studies of archaeological materials, see Bridges (1992) and Jurmain (1999). Nevertheless, much is still unknown.

Many causes of osteoarthritis have been discussed, such as age, weight, and mechanical loading (e.g. Merbs, 1983, 2001; Waldron, 1997; Derevenski, 2000; Kahl & Smith, 2000; Solano, 2002; Weiss, 2005, 2006). Anthropologists working on reconstructing activity patterns have focused especially on the role of repetitive mechanical loading. Defining the cause of osteoarthritis as resulting from repetitive mechanical loading has led to conclusions that severe osteoarthritis scores on specific joints are the result of continued use of specific muscles and joints in daily and repetitive tasks. Such an approach has led several researchers to consider osteoarthritis as ideal for reconstructing lifestyles. This perspective has a considerable history, but was given much impetus in the 1960s and 1970s with the work of J. Lawrence Angel (e.g. 1966, 1971) and Calvin Wells (e.g. 1962, 1963, 1972). Ortner's (1968) pioneering analysis of elbow osteoarthritis also stimulated wide interest.

Other important contributions included Merbs' (1983) analysis of the Saldermiut Inuit, Jurmain's (1978) work on Pecos Pueblo and Alaskan Inuit samples, Thould & Thould's (1983) study of a Romano-British collection, Bennike's (1987) analysis of Danish remains, Waldron's (1992) analysis of a London Black Death cemetery, and Webb's (1995) comprehensive study of Native Australians.

Using this basic palaeopathological/palaeoepidemiological approach, several researchers incorporated analyses of osteoarthritis into a broader bioarchaeological framework – most notably Larsen and colleagues (Larsen, 1990, 1995; Larsen & Ruff, 1994) and Phillip Walker and colleagues (Walker & Holliman, 1989). An

important focus of this research investigated possible links between subsistence economy and prevalence and patterning of osteoarthritis (see, e.g., Cohen & Armelagos, 1984, but also see Bridges, 1992 for a critique). Additionally, questions regarding whether males and females differed in activity patterns, whether groups differed in specific activities related to food production and trade, plus many more, have at least sometimes been tentatively answered using osteoarthritis scores (e.g. Lovell & Dublenko, 1999; Derevenski, 2000; Slaus, 2000).

It is important to note, however, that many anthropologists are cautious when trying to reconstruct specific activities; that is, they acknowledge the complex aetiopathogenesis of osteoarthritis. The most supported findings in the anthropological osteoarthritis literature relate to age differences (see references within Jurmain, 1999). Researchers using osteoarthritis scores to reconstruct past lifestyles frequently take age differences into account to enable more accurate reconstructions, but controls beyond age may be important as well (e.g. Waldron, 1997; Kahl & Smith, 2000; Merbs, 2001; Weiss, 2005, 2006). In all age-controlled investigations (and agreeing with clinical data), a very high correlation of spinal involvement with age was observed. In fact, Knüsel *et al.* (1997: 494) concluded that 'the vertebral column may not be an ideal structure to study markers of occupational stress'.

Beginning in the late 1990s, skeletal analyses focusing on osteoarthritis have become considerably less common. This is unfortunate since, even if behavioural interpretations are usually tenuous, understanding the skeletal pattern of osteoarthritis can be important in interpreting the disease itself (for good examples of the possible productivity of such an approach, see Baetsen *et al.*, 1997, Waldron, 1997). While a focused anthropological interest in osteoarthritis has declined, many more general skeletal studies (which incorporate osteoarthritis evaluation) still fail in their research designs to appreciate the inherent complexities of the disease process (see, e.g., Steckel & Rose, 2002, especially the methodological chapter authored by Goodman & Martin, another good example of persistence of such simplification is Cope *et al.*, 2005). This review will provide information on the most

recent medical and anthropological research on the effects of genes, hormones, anatomy, body mass and behaviour on osteoarthritis, to better use osteoarthritis scores in understanding past populations.

Recent research

Osteoarthritis aetiology is multifactorial. While anthropologists have concentrated on the effects of repetitive mechanical loading along with ageing as the main factors influencing the onset and severity of osteoarthritis, clinical research has been able to identify other factors that influence osteoarthritis patterns. The most recent medical literature can be separated into three main non-activity related aetiological groupings: genes, anatomy, and body mass index (or weight). In addition, medical epidemiologists, as reported in the occupational and sports literature, have also intensively investigated populations ostensibly at risk for developing osteoarthritis; these results bear directly on the issues of aetiology and mechanical loading (see below for further discussion). This medical research has consequences for anthropologists in terms of reconstructing activity patterns, especially with regard to sex and group differences. Although there is some overlap in these categories, for most purposes they can best be discussed in the above-mentioned categories.

Genetic influences

Genetic studies have recently gained considerable momentum in the medical literature relating to risk factors of specific diseases, including osteoarthritis. Two main forms of genetic studies to determine heritability are family studies, which examine frequency of different traits within specific familial lines, and twin studies, which compare monozygous (identical) twins with dizygous (non-identical) twins. Both familial and twin studies find support for genetic influences on the formation of osteoarthritis (e.g. Jonsson *et al.*, 2003; Manek *et al.*, 2003; Spector & MacGregor, 2004; Zhai *et al.*, 2004; Min *et al.*, 2005). Results from various twin and familial studies show that overall heritability of osteoar-

thritis averages around 0.50; in other words, 50% of phenotypic variability in osteoarthritis can be accounted for by differences in genotype (e.g. Ingvarsson *et al.*, 2000; Lanyon *et al.*, 2000; Jonsson *et al.*, 2003; Manek *et al.*, 2003; Spector & MacGregor, 2004). However, it is important to note that there is research suggesting that twin studies may overestimate heritabilities (Zhai *et al.*, 2004). Owing to the nature of the measure, heritability estimates can vary significant from population to population. Since heritability is an estimate of the genotypic contribution to *overall* phenotypic variation, when the environmental contribution varies, so too will the calculated heritability. When compared with prior populations (or contemporary non-Western, non-urban ones), where environmental influences can be much more extreme, twin studies might well produce considerably inflated heritability values.

Looking more closely at these twin studies, different joints seem to be more or less affected by genetics. The spine and the hip, for example, have the highest heritability estimates, ranging from 0.60 to 0.70 (Sambrook *et al.*, 1999; Spector & MacGregor, 2004), but hand and knee joints have considerably lower heritabilities of about 0.40 (Spector *et al.*, 1996). It is important to note that some of these studies already control for age, body mass index, bone mass density and activity levels (Spector & MacGregor, 2004).

With the knowledge that genes influence osteoarthritis, researchers have begun to use molecular techniques to identify candidate loci and to isolate polymorphisms at these sites (e.g. Uitterlinden *et al.*, 2000; Bergink *et al.*, 2003; Min *et al.*, 2005). Up to nine likely loci identified so far have significant effects on osteoarthritis (Uitterlinden *et al.*, 2000; Spector & MacGregor, 2004; Min *et al.*, 2005). It is a complicated picture, with different loci influencing different joints, possible pleiotropic effects, and sex and population differences confounding the factors (Spector & MacGregor, 2004; Min *et al.*, 2005). Moreover, there seem to be differential effects on various parts of the joint (Valdes *et al.*, 2004). For example, some loci (e.g. Vitamin D receptor loci) seem to influence osteophyte development, while others (e.g. COLZA1) primarily involve joint space narrowing (i.e. cartilage loss) (Spector &

MacGregor, 2004; Valdes *et al.*, 2004). Since spinal involvement, especially lumbar disk disease, is nearly ubiquitous in modern populations, and, as seen osteologically, the lumbar syndesmoses are also the most commonly involved spinal joints, specific genetic evidence relating to degenerative lumbar disease is especially illuminating. Recent work has shown that individuals with a specific single nucleotide mutation (an SNP) in the *CILP* locus are at considerably higher risk for developing degenerative lumbar disk disease (Seki *et al.*, 2005). This gene has also been implicated in influencing osteoarthritis in other joints (Serra *et al.*, 1997; Yan *et al.*, 2001). This apparently common polymorphism of the *CILP* gene influences both cartilage production and maintenance. Further research on the frequency and physiological effects of this variant (and others) might help explain why spinal degenerative disease has such high heritabilities and, secondly, why some populations are more affected than others. Lastly, this research reminds us again that spinal degenerative disease is very probably *not* a good indicator of activity.

It also appears that genetic influences from these molecular studies find no or little heritability of the presence versus absence of osteoarthritis, but genes affect the severity of the osteoarthritis present (Spector & MacGregor, 2004). As an aside, it seems important to note that many studies find that the heritability is higher in females than in males, which could be due to the role that oestrogen receptor genes have in osteoarthritis (e.g. Wilson *et al.*, 1990; Bergink *et al.*, 2003; Spector & MacGregor, 2004).

To end this section on a more positive note for anthropologists, there may still be exceptions when mechanical loading is extreme, such as seen in farm workers and athletes (Manek *et al.*, 2003; Thelin *et al.*, 2004). As noted, in most cases, heritability estimates have been obtained from more broadly sampled contemporary urban populations, which for the most part do not experience extreme mechanical loading, thus these estimates may not easily be extended to past populations.

Anatomical influences

Anthropologists often concentrate on the loads and stresses that bones and joints experience

through activity patterns, but they seldom consider the anatomical variance that can affect joints differently. Some individuals have anatomical variances that put more torque on a joint than other individuals, which can in turn influence the onset and severity of osteoarthritis. One such example of anatomical variance and the effect it has on osteoarthritis is knee height; it appears that having a long shin (or a high knee) increases the prevalence of osteoarthritis of the knee, especially in females (Hunter *et al.*, 2005). The indirect cause may be due to an increase in external knee adduction movement, which will affect the distribution of forces on the knee joint. Females may especially show this patterning because they lack the greater knee-stabilising quadriceps strength found in males (Hunter *et al.*, 2005). Thus, a sex difference here is related to anatomy rather than activities.

Another example is that of acetabular dysplasia and osteoarthritis of the hip. Individuals with acetabular dysplasia, or shallow hip-sockets, have an increased risk of hip osteoarthritis (Reijman *et al.*, 2005). Again, this effect is found to a greater extent in females and increases with a higher body mass index. It is also important to note that mechanical factors or activity have also been shown to have an additive effect with hip osteoarthritis and acetabular dysplasia (Reijman *et al.*, 2005). Other examples come from population differences, such as variance in knee alignment and an increase in knee osteoarthritis in Chinese peoples compared with Caucasians (Felson *et al.*, 2002).

Normal anatomical variance coupled with weight and activity thus influences the onset and severity of osteoarthritis, especially in the lower limb and in females. It is interesting to note that females tend to have more osteoarthritis in the lower limbs than in the upper limbs. As stated before, this may be in part due to genetics and in part due to anatomy (of course, the latter could easily be partly the result of genetic variation) (e.g. Wilson *et al.*, 1990). Biological sex differences, however, are often confused with cultural sex differences in skeletal samples, especially when trying to reconstruct activity patterns.

A much more general anatomical/biomechanical influence very frequently leads to onset of degenerative spinal diseases. Indeed, the

evolution of the human spine has produced a species-specific pattern, with similar involvement found systematically among most samples. It appears that a bipedal gait with co-evolved spinal curves and concomitant compressive forces produce this common pattern (Merbs, 1983; Kilgore, 1990; Bridges, 1994). While the human intra- or inter-population distributions of spinal degenerative disease do not seem to provide much assistance in evaluating behavioural or other differences among humans, a more general comparison with other primates does offer the potential for insight into the evolution of bipedality (Jurmain, 2000).

Body mass index influences

Heavier people have more severe osteoarthritis than lighter people (Heliövaara *et al.*, 1993; Tepper & Hochberg, 1993; Dumond *et al.*, 2003; Manek *et al.*, 2003). Both mechanical and systemic effects seem to play a part in the influence that body mass index has on osteoarthritis. For example, even non-weight-bearing joints are negatively affected by a high body mass index (which is a calculation of an individual's weight as a ratio of their height), but weight-bearing joints experience the highest influence (Dumond *et al.*, 2003; Manek *et al.*, 2003). The mechanical effects are straightforward; joints that are exposed to heavy loads are mechanically stressed and this causes degeneration of the cartilage, especially at the knees (Manek *et al.*, 2003). Systemic effects relating to body mass are more difficult to explain. The relationship of body mass to osteoarthritis does not appear to be mediated by action of a single gene or a combination of a few genes; rather, such systemic influences may be due to greater leptin (fat) in the cartilage that causes osteophyte formation, or they may be related to changes in hormones that arise secondarily from changes in body fat (Dumond *et al.*, 2003). In either case, it is clear that high body mass index, especially in overweight and obese females, correlates with osteoarthritis. The sex difference may in part be due to the smaller size of the joints experiencing higher loads than in males and/or related to hormonal effects.

Jurmain (1991) and Weiss (2005, 2006) have conducted osteological studies to determine whether body mass or size affects osteoarthritis. For example, Jurmain (1991) used four simple body size measurements and found no significant body size correlations with osteoarthritis on a large sample. He also attempted to see what effect weight, as recorded in the dissecting room sample, might have; here, the correlations were negative, but not significantly so. Weiss (2005, 2006), on the other hand, found that when age was controlled for, osteoarthritis scores correlated with body mass and body size. Her results were negative, which meant that smaller individuals experienced more osteoarthritis than larger individuals. Interestingly, while Jurmain's (1991) earlier findings did not establish a significant (negative) correlation with body size, the new data do show such significant negative correlations; so the direction is still the same. Additionally, Weiss (2006) used body mass as calculated from various skeletal elements to determine whether the negative correlation found in her previous study would appear in a large sample size ($n = 114$). She found that with age and sex controls, hip osteoarthritis correlated negatively with body mass (Weiss, 2006). Of note, body mass and sex were highly correlated in both studies (Weiss, 2005, 2006). Thus, body size may also be intermingled with sex differences, and it is important to take a closer look at whether observed sex differences are related to some combination of activity patterns, hormones or size.

In summary, the recent osteological studies by Weiss (2005, 2006) show that smaller and lighter individuals have greater osteoarthritis scores than larger individuals, which seems to contradict the clinical literature mentioned above on weight. Body size as measured on skeletal remains, however, is not body weight, and it is unlikely that the individuals in this sample were overweight. One could hypothesise that the smaller the linear size of an individual, the smaller their joints and that, if they add extra pounds to their frame, this would affect a smaller individual more than a larger individual. The difference between the medical literature and the anthropological literature may reflect the fact that high body mass index seems to be a modern

phenomenon. Thus, the effect of body mass may not be as significant an issue for anthropologists looking at pre-modern samples as it is for contemporary populations.

Mechanical influences: the epidemiological data

Anthropologists who investigate osteoarthritis from the perspective of behavioural (activity) reconstruction have often referred especially to one area of biomedical research, that is, epidemiological studies of at-risk populations participating in particular occupations or sports activities. Unfortunately, references to this rich literature source have sometimes been superficial, with only a few sources cited—and these appear sometimes to be selected to reinforce the preconception that osteoarthritis is caused primarily by activity. It is fair to note, however, that almost everyone who has done such osteological research has been guilty of such selective culling of this literature (e.g. Jurmain, 1977, 1978, 1980).

The earlier review (Jurmain, 1991) attempted to correct this bias, discussing evidence from the occupational and sports literature that both supported *and* failed to support a link between activity and prevalence of osteoarthritis. Nevertheless, this was still quite a superficial treatment, citing a total of nine studies (five finding a positive relationship of osteoarthritis with activity, and four finding no significant relationship). Occupations discussed included pneumatic tool users, cotton mill workers, weavers, coal miners, long-

shoremen, and physical education teachers. Also, a few sports-related activities were mentioned, including those performed by ballet dancers, soccer players and parachutists. In addition to this limitation the sources were quite outdated (with 70% pre-dating 1970, including all those showing a positive relationship).

Later in the 1990s, a much more extensive review of the occupational/sports epidemiological data was done by one of us (Jurmain, 1999). A summary of the studies covered by that review, expanded to include more recent work, is shown in Tables 1 and 2. As is evident from this more complete survey, the evidence is far from clear-cut. Both for the more general studies of overall activity as well as the more specific studies focusing on particular occupational or sports activities, results are mixed. Those data relating to overall increased levels of activity and prevalence of osteoarthritis show no obvious trend. Indeed, barely half of the samples evaluated (a total of 41) found a consistent and significant association.

When looking at the epidemiological studies focusing on specific risk groups of individuals engaged in presumably mechanically stressful activities, results are slightly more encouraging—but not overwhelmingly so. For these varied samples, including a wide range of occupational and sports activities and numerous joint areas (although most analyses concentrated on the hip and/or knee), a modest trend is seen. At least a majority of the samples (~2/3) did show a positive correlation of increased prevalence of osteoarthritis compared with controls. Some interesting patterns are also seen, especially in the most recent investigations. For example, it is

Table 1. Epidemiological studies of population samples at risk for developing osteoarthritis^a studies investigating correlation of osteoarthritis with general levels of activity

	Prior total ^b	Current total ^c
Samples in which positive correlation was found (Yoshimura <i>et al.</i> , 2000; Flugsrud <i>et al.</i> , 2002; Manninen <i>et al.</i> , 2002; Zhang <i>et al.</i> , 2004)	18	22
Samples in which no significant correlation was found (Sutton <i>et al.</i> , 2001; Jones <i>et al.</i> , 2002)	11	14
Samples in which results were mixed (Coggon <i>et al.</i> , 1998; Cvijetic <i>et al.</i> , 1999; Rossignol, 2004)	2	5

^aUpdated with new citations in italics.

^bSee Jurmain (1999) for full listing and discussion.

^cTotals include 1999 survey plus updated sources.

Table 2. Epidemiological studies of osteoarthritis^a—samples investigated for correlation of osteoarthritis with specific occupational/sports activities^b

Occupation/sports activity	Studies finding positive correlation	Studies finding no correlation	Mixed results
Textile workers	1	1	
Pneumatic drill users	6	2	
Foundry workers	1	0	
Chainsaw users	0	1	
Lumberjacks	0	1	
School cooks	1	0	
Sailors ('seafarers')	1	0	
Parachutists	0	1	
Karate instructors	0	1	
Baseball pitchers	4	0	
Soccer players	5	2	1
	(Shepard <i>et al.</i> , 2003)		
American football players	3	0	
Runners	2	6	
		(Lane <i>et al.</i> , 1998)	
Ballet dancers	3	2	
Professional dancers	1		
	(Teitz & Kilcoyne, 1998)		
Wrestlers	1	0	
Weight lifters	2	1	
Swimmers	1	0	
Porters	0	2	
Rock climbers	1	1	
		(Schoffl <i>et al.</i> , 2004)	
Floorlayers	1	0	
	(Jensen <i>et al.</i> , 2000)		
Carpenters	1	0	
	(Jensen <i>et al.</i> , 2000)		
Rowers	0	1	
		(Teitz <i>et al.</i> , 2003)	
Javelin throwers (elite)	1	0	
	(Schmitt <i>et al.</i> , 2004)		
High jumpers (elite)	1	0	
	(Schmitt <i>et al.</i> , 2004)		
Truck drivers	1	0	
	(Rossignol <i>et al.</i> , 2003)		
Housekeepers	1	0	
	(Rossignol <i>et al.</i> , 2003)		
Building and construction workers	1	0	
	(Holmberg <i>et al.</i> , 2004)		
Farmers	8	0	1
	(Thelin <i>et al.</i> , 2004)		
	(Rossignol <i>et al.</i> , 2003)		(Holmberg <i>et al.</i> , 2004)
	(Tuchsen <i>et al.</i> , 2003)		
	(Sandmark <i>et al.</i> , 2000)		
	(Thelin <i>et al.</i> , 1997)		
Totals ^c	48	22	2

^a Updated with new citations in italics.

^b See Jurmain (1999) for full listing and discussion.

^c Totals include 1999 survey plus updated sources.

now becoming increasingly clear that individuals engaged in farming are at significantly higher risk for developing hip osteoarthritis; fewer studies have focused on knee involvement, and results have been mixed. In two Swedish studies,

one (Sandmark *et al.*, 2000) found that knee osteoarthritis was more common among both men and women participating in farming, but in the other more recent investigation (Holmberg *et al.*, 2004), results were mixed (females showed a

significantly higher prevalence than the general population, but males did not; see also Walker-Bone & Palmer, 2002, for a good review of musculoskeletal disorders among farmers).

Osteologists have also noted the epidemiological trend seen among agricultural workers (Waldron, 1997; Jurmain, 1999) and have suggested a possible explanation. Individuals involved in agriculture frequently begin stressful activities *early in life* (often while still children). To this point, contemporary epidemiological studies of individuals engaged in agricultural work have generally not consistently controlled for age of onset of mechanical stress. Some of this research has suggested a link of increased risk with an early age at which the activities began (Cooper *et al.*, 1996; Thelin *et al.*, 1997; Rossignol *et al.*, 2003); however, a recent, well-controlled analysis found 'no support for any relationship between "work at a young age" and the development of osteoarthritis' (Thelin *et al.*, 2004: 208). It may be that the age of onset of mechanical stress is at least as important as the amplitude of stress and probably more influential than duration as measured in adults. Similar observations and conclusions have recently been forcefully stated by Pearson & Lieberman (2004) as pertaining to general capacity for bone remodelling in subadults as compared with adults.

From an osteological perspective, what then can we learn from these epidemiological data on osteoarthritis? It seems that osteoarthritis is not an 'ideal' indicator of the overall level of activity, nor is it at all a good predictor of specific activities. However, in *some* cases, osteoarthritis can become much more likely to develop—when the stresses are high in amplitude, *and* they begin quite early in life. These conclusions have obvious relevance for interpretation of osteoarthritis in archaeological samples. But caution must be used; the effects will not pertain to all joints nor to all (or even most) populations.

Proper differential diagnoses of the medical research for anthropologists

Some possible consequences for skeletal evaluation of osteoarthritis, considering the information from the medical fields, are:

- (1) There are many aetiologies of osteoarthritis and there may actually be more than one 'disease' that is being defined as osteoarthritis.
- (2) Since different joints vary with regard to the effects of genes and environment, anthropologists should be cautious of aggregate scores when it comes to osteoarthritis.
- (3) Genotypic influences involve several loci, most of which are demonstrably polymorphic (among individuals). It remains to be determined whether polymorphism also exists at the population level (if so, we are even more constrained in making behavioural interpretations). That is, differences between populations could reflect differences due to age, body mass and/or genotypes. The total variance accounted for by these factors could be over 80%. It is, however, possible that 80% is an over-estimate, since clinical studies of modern people often consist of individuals living in homogeneous modern environments.
- (4) Sex differences that have been considered due to activity patterns may actually be related to hormones, body size, genes and anatomy. Anthropologists need to take care to dissect the causes of sex differences instead of assuming that they are cultural in nature.
- (5) The biomedical literature concerning osteoarthritis is extremely broad and growing dramatically. Moreover, results often continue to be contradictory. Systematic evaluation of this rich and invaluable resource presents a challenge for those of us doing osteological research.

Osteological perspectives on osteoarthritis: diagnostic criteria

Skeletal evaluation of osteoarthritis has used both marginal changes (i.e. osteophytes) as well as alterations to articular surfaces. Regarding the latter, both 'porosity' and eburnation are widely used (e.g. see the *Standards* publication (Buikstra & Ubelaker, 1994)). Osteoarthritis scores for individual joints thus usually represent composite variables, and totals of limb or vertebral segments, or even entire skeletons, have been further combined into aggregate scores (e.g. Jurmain, 1977, 1978, 1980; Weiss, 2005).

We suggest it is perhaps timely to reevaluate these approaches. As noted above, the genetic/physiological mechanisms influencing margins of joints may be separate from those operating on joint surfaces. Using multivariate analyses, some of our skeletal research (Jurmain, 1980) also suggests it is most prudent to evaluate an individual joint's marginal changes separately from those of the articular surface.

Consistent results of our research have shown that osteophytes develop primarily as a correlate of biological ageing and, thus, do not seem to be a reliable indicator of the severity of the disease. Other researchers, both from a clinical perspective (e.g. Hernborg & Nilsson, 1977; Duncan, 1979; Moscovitz, 1993) as well as osteological observations (Rogers & Waldron, 1995; Nagy, 1996; Rogers *et al.*, 1997), have reached similar conclusions.

Aggregation of data for several joints probably compounds the problems further. Aggregation may not be the best method to use in osteoarthritis studies, but some earlier researchers used aggregation in part due to the high level of correlation between joint osteoarthritis scores within an individual. As previously mentioned, both authors have used such aggregate osteoarthritis measures in the past; we are currently doing a systematic analysis to see how results may differ from our earlier results when specific areas within joints are evaluated.

Given that articular surface modifications are probably the best indication of the severity of osteoarthritis, we still must be cautious regarding *which* surface changes are used. Certainly, in both in both clinical and osteological analyses eburnation is regarded as a clear indicator of severe disease. However, surface pitting (also called 'porosity') has also frequently been used as an ostensibly accurate indicator of osteoarthritis (e.g. Buikstra & Ubelaker, 1994; Jurmain & Kilgore, 1995; Weiss, 2005). Nevertheless, there is now good reason to suspect that these small surface pits may be unrelated to osteoarthritis. As carefully researched by Woods (1995) and summarised by Rothschild (1997), the small 'holes' or 'coalesced pits' occur independently and in different joint areas from the later, more diagnostic surface alterations (i.e. eburnation). In fact, areas of such porosity appear to be most

common in articular areas *not* in regular joint contact. Woods (1995), therefore, concluded that these changes might reflect some secondary effect due to vascular invasion to supply under-nourished cartilage.

Future directions

As noted in the 1991 review, we re-emphasise that controlled palaeoepidemiological comparisons are essential to discern populational differences in the prevalence of osteoarthritis. That is, samples must be stratified by both age and sex—thus necessitating large initial samples. Further, it is important to record data relating to joint involvement in a precise manner. As argued above, and as suggested by the *Standards* publication (Buikstra & Ubelaker, 1994), separate scoring of marginal changes and surface alterations is very useful; noting especially severe involvement by the presence of eburnation will assist in making comparisons between studies more accurate and more meaningful. Thus, in publishing severity frequencies of osteoarthritis involvement, it is helpful to report more than simply 'slight, moderate, and severe'. A further listing, in tabular form or as an appendix, of fine-tune evaluations will prove very useful as well. Only in this way can full and more *consistent* epidemiological comparisons be made.

Additionally, anthropologists may want to consider aetiologies that are not reliant on activities. Possible animal studies, such as those done on bone remodelling with sheep and treadmills that are summarised in Pearson & Lieberman (2004), could increase our knowledge of osteoarthritis. These studies could control activity levels, diet, be age-specific, and possibly even look at the specific molecular genetics of osteoarthritis. Although bone remodelling and osteoarthritis differ in aetiology and speed of occurrence, animal studies often aid in understanding the complexities of biology in a way that is not possible in human studies due to ethical issues, time constraints and cost.

To end on a positive note, systematic and multiple within—body comparisons are probably the best measures for reconstructing activity patterns. That is, asymmetry evaluations of

several variables (including osteoarthritis, musculoskeletal markers, cross-sectional geometry, and body size) show the most promise. Asymmetry measures do not need to be standardised for body size; the whole body is experiencing the same nutrition and age, and so any differences between limbs are more likely to reflect activity patterns. Currently, we are investigating these asymmetry variables to a fuller degree in order to see whether they offer more reliable and effective measures to reconstruct activity patterns. We remain mindful of the variety of confounders that may affect the patterning of these variables, whether singly or more systemically. So while emphasising that there is substantial reason to pursue productive avenues of research further, caution is always required. We encourage others to return to systematic evaluations of osteoarthritis as part of a more complete bioarchaeological understanding of past populations.

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