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# The relationship of age, activity, and body size on osteoarthritis in weight-bearing skeletal regions

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ABSTRACT

This study examined the simultaneous impact of multiple underlying factors on OA expression in weight-bearing joints of the vertebrae and lower limb of a modern European skeletal sample (Lisbon and Sassari). OA was evaluated using standard ranked categorical scoring; composite OA scores derived through principal component analysis. Body size was calculated from postcranial measurements; torsional strength (*J*) of the femoral midshaft was calculated from three-dimensional surface models, size standardized and used as a proxy for activity. A standard multiple regression was applied.

In all regions, the linear combination of age, body mass, stature, and *J* was significantly related to differences in OA. Across all joints, age was the strongest predictor; neither body size, nor activity variables demonstrated a statistical relationship with OA at the lumbar or knee; *J* demonstrated a negative correlation with pelvic OA.

Variation in OA can be explained by age, stature, body mass, and structural adaptation related to habitual use. The negative correlation between femoral torsional strength with OA suggests that long-term, repetitive physical work capacity in childhood may be protective against OA development later in life. The multifactorial aetiology of OA requires incorporating multiple lines of evidence to interpret individual or population health from bone samples.

## 1. Introduction

Osteoarthritis (OA) is the most common joint pathology in human populations, studied extensively for more than 60 years by both clinicians and osteologists to produce an amazingly deep and rich literature base (Rogers et al., 1987; Jurmain, 1999; Karsenty, 2003; Arden and Nevitt, 2006; Waldron, 2012). Clinical research has focused largely on cellular destruction of articular cartilage, but new diagnostic tools (e.g., MRI, bone scans), and biomarker discoveries have revealed four important results: (1) OA is not entirely a cartilage problem and can affect varied joint tissues, (2) OA is not a purely degenerative disorder, but also a reparative one, (3) OA is not necessarily progressive with potential to stabilize from changes in joint anatomy, and (4) OA is a focal pathology occurring in habitually loaded joint areas (Felson and Nevitt, 2004; Dieppe, 2011; Waldron, 2012). If OA is joint failure driven by cumulative or abnormal joint loading, rather than disease, then paleoepidemiological analyses of OA have much to contribute to a discussion of how environmental factors influence human biology via questions regarding mobility, habitual behaviours, and aging in archaeological populations.

Despite major advancements in knowledge, the aetiopathogenesis of OA is complex and still poorly understood (Felson et al., 2000). The interplay between systemic influences (e.g., age, sex, hormones, nutrition, genetics) and local biomechanical risk factors (e.g., muscle weakness, obesity, and physical activity) requires further study to determine whether OA represents a single state or a heterogeneous cluster of conditions that share a common final pathway (Felson et al., 2000; Sowers, 2001). Research on OA prevalence has consistently demonstrated that it increases with age (Jurmain, 1991; Loeser, 2010) and that sex-specific differences are evident (Oliveria et al., 1995), but complexities of bone biology, limited samples, and fragmentary preservation have restricted behaviourally-oriented research in the archaeological record, and osteologists are rightfully cautious in interpreting both causal mechanisms and specific activities from bone pathologies (Weiss, 2015; Weiss, 2006; Jurmain et al., 2012). Studies that consider joint changes of OA as a function of both the natural aging process and of mechanical stress (wear-and-tear) are necessary to recognize diverse aetiologies of OA, particularly in relation to clinical diagnoses, risks, and treatment, but also to uniquely document bony lesions associated with OA biology that can only be studied through

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direct observation of articular bone surfaces.

The relationship between joint OA and excessive mechanical stress can be assessed through variation in long bone diaphyseal shape. The distribution of cortical bone reflects its loading history; therefore, skeletal remodelling of the long bone diaphysis is a useful measure of behavioural inference (Ruff and Hayes, 1983). The seminal research by Ruff and Hayes (1982, 1983, 1984), Ruff (2000), Ruff et al. (2006), Stock and Shaw (2007), and Sparacello and Pearson (2010) have shown how measures of bone robusticity address long standing anthropological inquiries regarding adaptive subsistence practices, variability in skeletal growth and development, and relative limb strength and locomotion. As such, long bone cross sectional geometric (CSG) properties (e.g., strength) may also be useful to investigate reasons for the onset and development of musculoskeletal disorders, such as OA, that are driven by biomechanical forces as much as by genetic influence and biological aging. Though OA does not coincide with the period during which diaphyseal cross-sectional morphology of the limbs is established, cumulative or abnormal loading of the skeleton demands that weight-bearing joints respond and adapt to mechanical stresses and strains, which may eventually be compensated by arthritic changes to bone (Stürmer et al., 2000; Berenbaum and Sellam, 2008). Since remodelling of the long bone diaphysis in humans is greatly reduced after young adulthood, a great deal of cross-sectional morphology reflects habitual activity during younger life (Allen and Burr, 2013). It is important to consider whether arthritic joint lesions (e.g., osteophyte formation) observed in adulthood are simply a functional adaptation to loads imposed on the plastic skeleton to promote joint stability (Dieppe, 2011), generated by different lifestyles.

Body size estimates are important to reconstruct various attributes of past populations that include demographic characteristics, to assess health, and to recognize patterns of skeletal morphology affected by activity loading patterns (Steckel and Rose, 2002; Ruff et al., 2006; Cohen and Crane-Kramer, 2007). Large body size correlates with generalized bone hypertrophy and with OA characteristics, of which osteophytes are the most consistent variable (Spector et al., 1996). Body weight is distributed primarily in the lower limb (Ruff, 2000), and moveable joints such as the hip and knee, are highly affected by OA (Larsen, 1997; Felson et al., 2000; Wearing et al., 2006; Jiang et al., 2011). Forces transmitted across the knee joint during normal gait range between two-and-three times body weight, a load effect that explains the increased risk for OA among overweight persons (D'Lima et al., 2012). Sanford et al. (2014) recently found that joint loads in the hip and knee increase approximately linearly with body mass, which strengthens the findings that body mass could be a factor linking obesity to OA (Oliveria et al., 1999; Powell et al., 2005; D'Lima et al., 2012); among other likely causes that include hormone production by adipose tissue (e.g., leptin and adiponectin) and increased muscle loading to resist greater loads (Pottie et al., 2006). An understanding of structural factors/limitations that contribute to both hip and knee OA is advancing rapidly (Lane et al., 2000).

Effects of stature on arthritic development are less well understood, although new research using biological markers (i.e., single nucleotide polymorphisms, SNPs) to identify genetic variants associated with height has identified a link between short stature and susceptibility to OA (Sanna et al., 2008). Gene polymorphisms may be linked to increased susceptibility to OA, because mutations that reduce the functional levels of cartilage matrix proteins, or alter key interactions in their assembly and function, could be expected to compromise the biomechanics of joint cartilage (Kannu et al., 2009). Genome-wide association studies represent a promising way to study complex, common, chronic disorders like OA that incorporate body size variables highly influenced by both genes and the environment. For paleopathologists, the relationship between overall body size and the mechanical stress threshold of weight-bearing joints may be specifically important factors to control for in evaluating joint failure and arthritic patterning in once-living populations.

The purpose of this study is to test the hypothesis that differences in adult patterns of age, activity, and body size are reflected in joint arthritic changes through an examination of OA in weight-bearing regions of the lumbar spine, pelvis, and knee for a modern 19th-20th century European skeletal population. Explaining population variation in OA by age, activity, or body size is relevant because OA aetiology is multifactorial. This bioarchaeological study examines the simultaneous impact of multiple underlying factors on the expression of idiopathic OA in weight bearing joints of the lower limb from a skeletal series; results of which aim to improve behavioural reconstruction and interpretation of disease determinants in the past.

## 2. Material and methods

### 2.1. Sample

The study sample (n = 124) was derived from two large modern European identified skeletal populations from Portugal (Luís Lopes, University of Lisbon) and Italy (Sassari, University of Bologna), grouped together based on a shared population history of Southern Europe with similar activities and lifestyle at the turn of the 20th century (Cardoso, 2006; Belcastro et al., 2008). Both populations represent a low socio-economic class, evidenced by documented occupations in rural farming/manual trades (males) and managing the family household (females). Exhumed from municipal cemeteries in the last 50 years, complete skeletons for individuals who died between 1912 and 1970 were evaluated and known sex and age-at-death were recorded for each individual from documents accompanying the collections. Ages-at-death were collected from coffin plates, cemetery records, and in many cases, the individual death certificate. Summary data for age and sex of the test sample are provided in Table 1.

Sample individuals were selected randomly, but only individuals presenting with the requisite morphology were evaluated. Specimens affected by OA related to fractures were excluded, as were individuals with gross pathological evidence of infectious disease to avoid confusion with secondary OA that forms as a result of pre-existing abnormalities in joint tissues, and which may also influence diaphyseal morphology. Other criteria for selection were bone preservation and representation of adults in all age-classes. Elements from the left side were evaluated, unless missing from the collection in which case the right side was substituted. Use of the left side is justified by the reduced directional asymmetry of the lower limb (Auerbach and Ruff, 2004); and although there is some evidence to suggest asymmetry in the human sacrum (Plochocki, 2002), a recent study by Kurki (2017) has demonstrated pelvic asymmetry to be quite low, similar to lower limb patterns.

**Table 1**
Summary age and sex data for the test sample.

| Sex | Luís Lopes, University of Lisbon, Portugal | | | | Sassari, University of Bologna, Italy | | | | Combined sample | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n | Mean Age (years) | SD | Range (years) | n | Mean Age (years) | SD | Range (years) | n | Mean Age (years) | SD | Range (years) |
| Males | 29 | 56.2 | 20.5 | 20–88 | 34 | 54.1 | 18.9 | 21–82 | 63 | 55.1 | 19.5 | 20–88 |
| Females | 33 | 61.2 | 21.0 | 18–94 | 28 | 61.0 | 19.4 | 24–98 | 61 | 61.0 | 20.1 | 18–98 |
| Total | 62 | 58.7 | 20.8 | 18–94 | 62 | 57.6 | 19.2 | 21–98 | 124 | 58.1 | 20.0 | 18–98 |