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Research Article

Age-at-death estimation of pathological individuals: A complementary approach using teeth cementum annulations

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ABSTRACT

Bioarchaeologists rely on accurate estimations of age-at-death. Clearly, some pathological conditions are associated with gross morphological changes in the skeleton that could impact the effectiveness of age-at-death estimation (i.e. methods based on the pelvis, fourth rib, dental attrition, and cranial stenosis). The magnitude of this problem has not been widely studied due to a paucity of pathological skeletons of known age. We assessed age-at death for three individuals affected by bone dysplasias (achondroplasia, residual rickets, osteogenesis imperfecta) using cementum annulations and several osseous age indicators. We predicted osseous indicators that are based on gross morphological changes would yield age estimates discrepant from the cementochronology. Results demonstrated considerable differences in age estimates between morphological and histological techniques suggesting a need for additional research on the effects of pathology on the accuracy of morphological methods. Conversely, we addressed the proposition that cementum annulations will be inappropriate for age estimation in cases of chronic and severe rhino-maxillary infection and periodontitis. We assessed age-at-death for one individual with leprosy and found no indication the disease process affected cementum formation or preservation. The results of this research indicate the potential value of cementochronology in cases where skeletal pathological conditions constrain the usefulness of traditional age estimation approaches.

1. Introduction

Paleopathology is increasingly recognized as a critical tool for understanding the origin and evolution of human diseases, and it is the only means of reconstructing diversity in the etiology and epidemiological profiles of different diseases through time. Unfortunately, the field suffers from a lack of precision that limits its inferential power. At a very basic level, it is difficult to estimate age-at-death for adult skeletons, which has important impacts on demographic and epidemiological reconstructions (Séguy and Buchet, 2011) and constitutes an important part of the "osteological paradox" (Wood et al., 1992). Techniques for age estimation based on morphological changes are fundamentally limited by the typological approach to age-related changes, which assumes several morphological indicators will co-vary. An additional problem is that many of our techniques are based on Least Squares Regression, which is inappropriate for describing auto-correlated time-series data that vary in the longitudinal and cross-sectional dimension (Robbins Schug et al., 2013). Regression also suffers from a well-known centrist tendency and results in reference population mirroring (Bocquet-Appel and Masset, 1982).

These are all well-known difficulties and two important lines of research have arisen to address these issues. The Rostock Manifesto (Hoppa and Vaupel, 2002) argued for a Bayesian approach to the data. Many researchers have thus adopted probabilistic approaches to these data, including transition analysis (Boldsen et al., 2002), an increasingly popular and relatively easily employed approach that effectively alleviates some of the problems we have outlined (DeWitte, 2010; DeWitte et al., 2013). Taking a probabilistic approach to age-at-death smooths-over the lack of precision inherent to evaluating morphological changes and improves population-level comparisons (Milner and Boldsen,

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2012); however, it is a statistical fix and obviously does not address the imprecision of age related changes in the morphological indicators themselves.

Another approach to this 'crisis of paleodemography' (and paleoepidemiology) is to look for more accurate and precise indicators of age in adult human tissues. Cementum annulations are the closest attribute we have to a chronometric measure of age-at-death in the human dentition. When a count of cementum annulations is added to the age of eruption of a particular tooth, this method provides the most accurate and precise estimation on which to base biocultural research (Robbins Schug et al., 2012; Wittwer-Backofen et al., 2004). The major impediment for archaeological applications is the problem of taphonomic damage (Klevezal and Shishlina, 2001; Roksandic et al., 2009), which obviously affects all age indicators. However, even if cementum annulations are applied to age only a proportion of skeletons in a prehistoric population, their use has a significant impact on the age pyramid and on demographic statistics (Robbins Schug et al., 2012). The annulations are also helpful for identifying systematic biases in other age indicators (Robbins, 2004; Robbins Schug et al., 2012).

This paper addresses this fundamental issue in age estimation. The problem we have outlined is relevant to bioarchaeological research in general (Schmitt, 2002), but it is undeniably more relevant when we are considering pathological individuals. Morphological techniques assume that the pattern and rate of maturation for individuals does not significantly vary from a reference population. It is unclear whether this postulation is appropriate for specimens exhibiting pathological or morphologically abnormal features, particularly since the standards were developed from reference samples that (logically) excluded pathological cases.

This paper provides a comparison of age estimates from cementum annulations and other morphological indicators for four individuals to address two basic questions: (1) how is skeletal age estimation affected by skeletal dysplasias? (2) Are cementum annulations useful in cases of severe periodontal disease and oral infection? We hypothesized that skeletal dysplasia will impact the accuracy and precision of osseous age indicators. This was assessed through a comparison of estimates from the pelvis, fourth rib, and cementum annulations in four individuals with achondroplasia, osteogenesis imperfecta, and residual rickets. We predicted that in cases where pathological conditions have affected bone growth or altered skeletal morphology, age estimates based on skeletal morphology will be strongly discrepant from estimates based on cementum annulations. This research does not rely on an assumption about the accuracy of estimates from different techniques, but instead evaluates the level of difference among diverse techniques for the specific dysplasias considered here.

Conversely, our second research question involves the impact of infectious diseases that affect the periodontal ligament on the formation and preservation of cementum annulations. There is a commonly held assumption that individuals affected by oral pathological conditions, including severe periodontosis, will demonstrate disruptions in cementum annulation formation and erosion of the periodontal ligament that will lessen the utility of that approach. We tested that hypothesis through an assessment of age-at-death using osseous indicators and cementochronology in an individual with severe rhino-maxillary infection and periodontitis due to infection with *Mycobacterium leprae*. We predicted that age estimated from cementum annulations would be substantially different from that estimated from osseous indicators. To assess the presence and preservation of the cementum annulations, we performed a non-destructive evaluation of a tooth from an individual with Hansen's disease (leprosy) prior to undertaking histological analysis. We predicted the cementum annulations would not be intact, a result that would suggest that destructive analysis is unwarranted and morphological age indicators would be preferred in such cases. As micro_CT demonstrated intact tissues, histology was undertaken and we evaluated the evidence as to whether severe periodontal disease disrupted cementum formation. Specific predictions for each pathological condition and its potential impact on age estimation follow.

1.1. Achondroplasia

Achondroplasia is a congenital dysplasia resulting from a mutation in the FGFR3 gene, which produces fibroblast growth receptor, a protein involved in bone formation (http://www.genome.gov/19517823). Mutations in the FGFR3 gene alter the duration of endochondral bone growth (Deng et al., 1996) and have been linked to premature cranial stenosis (Doherty et al., 2007); however, it is unclear whether the mutation affects osteoblast activity in the intramembranous bones of the cranial vault (Opperman, 2000). What is clear is that in general, achondroplastic individuals have a combination of the following musculo-skeletal manifestations: disproportionately short limbs and resulting short stature, macrocephaly, midface hypoplasia, frontal bossing, flat and deformed thorax, short rib polydactyly syndrome, premature spinal stenosis, abnormal lumbar lordosis, muscle hypotonia, and brachydactyly (Auferheide and Rodriguez-Martin, 1998:360). Thus we hypothesized that achondroplasia will affect age-at-death estimated from the 4th rib. We predicted estimates from this indicator will depart significantly from estimates based on cementum annulations and dental attrition. While endochondral bone growth is affected, the intramembranous bones develop normally and thus timing of cranial suture closure should also not be affected.

1.2. Osteogenesis imperfecta and residual rickets

Osteogenesis imperfecta (OI) refers to pathologically frail bones resulting from a deficiency in the amount or composition of Type I collagen (Ortner, 2003:492).

There are four known types of OI and 90% of cases are caused by a single, dominant mutation in COL1A1 or COL1A2, genes responsible for making the protein constituents of Type I collagen (http://www.genome.gov/25521839). As 90% of osteoid is comprised of Type I collagen, this condition affects the entire skeleton and is often associated with dentinogenesis imperfecta as well (Ortner, 2003). The condition may or may not affect endochondral formation but always manifests in limited periosteal formation, thin cortices, persistent parallel lamellar bone at the periosteal surface, and other evidence of delayed osteonal remodeling in the compact bone (Ortner, 2003). Numerous fractures will affect the morphology and articulation of the skeletal elements.

Vitamin D deficiency contributes to the etiology of rickets in childhood and of osteomalacia in adults. Vitamin D is essential for mineralization of osteoid formed during bone growth and remodeling (Pitt, 1988). In growing children, significant effects due to vitamin D deficiency occur on the forming bone structure while adults only exhibit skeletal changes relating to incomplete mineralization of bone on pre-existing surfaces (Brickley and Ives, 2008). In adults, healed childhood conditions are detectable and classic manifestations of these residual condition include: residual bending of legs, lateral narrowing of pelvis, bulging at pubic symphysis, ventral projection of sacrum, curvature of ilia, anterior angulation of sacrum, kyphosis or scoliosis and vertebral body collapse, protrusion of sternum with rib angulation and with alteration in rib neck angle (Brickley and Ives, 2008).

Both conditions (OI and rickets) affect bone growth and mineralization. Thus on a crude level, we predicted that these two conditions would be associated with changes to articular surfaces that would negatively impact the precision of age estimates. We

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