11 Guidance on recording palaeopathology (abnormal variation)

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Due to the constraints imposed in providing a full revision, a much longer extended document, along with a full bibliography and more images, have been provided at: http://dro.dur.ac.uk/6160*, or can be sent as a pdf (contact c.a.roberts@durham.ac.uk).* I have taken key points from the longer document for this shorter document.

Please also note that this guidance is relevant to osteologists applying for Practitioner or Associate membership of the CIFA (Chartered Institute for Archaeologists); see: *https://www.archaeologists.net/sites/default/files/Osteology%20specialist%20competence%20matrix_final_0.pdf*

11.1 Introduction

Palaeopathology is the study of evidence of disease in the bones and teeth of archaeological skeletons and the soft tissues of preserved bodies, but disease can also be reflected in the discovery of parasite eggs found with bodies, in soils of graves containing skeletons, and also in archaeological contexts such as latrines and cesspits.

In general, the quality and quantity of data recorded still varies considerably across the sector (Larsen 2015, 2) and remains a complex, debated and developing issue in bioarchaeology.

11.2 Recording of pathological lesions

(See Ortner 2003)

The following lists the main points from the long version of this chapter and reflects the most recent advances in palaeopathology.

Four key methods are used: **macroscopic, radiological, histological and biomolecular**. Most people use the macroscopic method, and sometimes with radiology (in a commercial and an academic environment). Histological and biomolecular methods are used less frequently, because of costs and access to facilities. Useful references for these methods include Turner-Walker and Mays (2008), Mays (2008a), and Brown and Brown (2010).

It is recommended that preservation of the skeleton should be recorded first (this has implications for what pathological conditions may be recorded/whether distribution patterns can be documented).

Comparison of abnormal with normal bone and dental elements is a pre-requisite to recognising the abnormal, as is access to a disarticulated comparative skeleton and excellent knowledge of the normal appearance of the bones/teeth.

It is recommended that *definite* abnormalities that are not the result of what can be normal variation, pseudopathology, or postmortem damage should be recorded.

Use clinical data as a base to understand the bone changes, but remember that it may not always be appropriate (Mays 2012). For example, a commonly used text is Resnick's *Diagnosis of Bone and Joint Disorders* (latest edition: 2002).

Detailed clear and objective descriptions of pathological lesions are essential (and should be made available for future use, being archived electronically for download). Those descriptions should be used with clinical data to produce differential diagnoses. Consult the following website for terms: https://paleopathology-association.wildapricot.org/Nomenclature-in-Paleopathology. Pathological lesions should also be illustrated with photographs and illustrations, as appropriate.

Palaeopathological and clinical texts usually illustrate the most chronic/severe expressions of disease. However, chronic skeletal lesions do not develop 'overnight'; they may progress perhaps over several months or years. The timing and extent of development of lesions will also vary between individuals for a variety of reasons, such as immune system strength.

There have been recent developments for diagnosis, for example extracting microbial ancient DNA (eg, see Salo et al 1994; Müller et al 2014, Schuenemann et al 2013, Bos et al 2011), despite methodological problems (see Brown and Brown 2010); disease specific proteins, and other biomolecules (eg, mycolic acids) have also been used to diagnose disease. However, positive results for aDNA of a pathogen does not necessarily mean that that the disease caused the bone changes. Relatively recent developments include: looking at strains of pathogens, susceptibility and resistance genes, and diagnosis of disease that only affects the soft tissues.

Sampling for biomolecules for disease diagnosis should only be done when a full skeletal analysis has been done, and the questions being asked cannot be answered in any other way (see also Chapter 13, and https://historicengland.org.uk/images-books/publications/science-and-dead/).

Recording the 'severity' of dental or skeletal changes in disease needs reflection. What do the different grades mean? If recorded, then intra- and inter-observer error tests are needed, at least, to ensure recording consistency within and between observers. Greater 'severity' of bone changes does not necessary correlate with worse symptoms (eg, see Riddle et al 1988). Recording presence or absence is a safer route to follow.

The updates below refer to the sections used in the 2004 version of this chapter:

11.7.1 Infectious disease: *Non-specific infection*: infections potentially caused by a range of organisms that cannot usually be identified; *Specific infection* (where the causative organism is known; this might be a bacteria, fungus, virus, or parasite).

11.7.2 Trauma: see Bennike (2008) and Lovell (2008) for updated references on recording trauma.

11.7.3 Joint disease: Only diagnose osteoarthritis (OA) if eburnation exists or, if not, two other bone changes (eg, porosity and osteophytes). Osteophytes alone may indicate the ageing process and should not be used for an OA diagnosis.

The different joint disease lesions should not be 'lumped' together to indicate severity; an increase in the extent of one lesion may not necessarily be paralleled by an increase in extent of another.

11.7.3 Metabolic disease: Brickley and Ives (2008); Mays (2008b). *Cribra orbitalia* recording: see above regarding 'grades of severity'. O*steoporosis:* see Agarwal and Stout (2003).

11.7.6 Neoplastic disease: (Brothwell 2008, 2012).

11.7.7 Dental disease: (Hillson 2008). **(i) Caries:** for severity of grades (if recorded) use Hillson (2001). **(ii) Calculus:** see above regarding 'grades of severity'. Note recent advances in analysing dental calculus (Adler et al 2013; Warinner et al 2014). **(iv) Enamel hypoplasia:** see the FDI scoring system (Hillson 2005). **(v) Periapical lesions: (**Ogden 2008).

11.8 Presentation of data and interpretation: *It is recommended that the reader consults the longer version of this section*. Summary statistics are recommended (English Heritage 2004). Active (woven) new bone formation indicates the disease or trauma that caused the lesions was active at or around the time of death (perimortem). Usually it is not possible to suggest the cause of death from analysing skeletal remains, only what diseases or trauma the person experienced through their lives – bioarchaeologists record the skeleton of a person at the point of their death. The bones and teeth reflect an accumulation of disease processes throughout that person's life. Wood et al (1992) remains a very important reference for palaeopathology.

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(NB: a much more extensive bibliography can be accessed in the long version of this update)

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