

Published version of this paper:

Henderson, CY. 2013. Do diseases cause enthesal changes at fibrous entheses? International Journal of Paleopathology 3: 64-69. DOI: [10.1016/j.ijpp.2013.03.007](https://doi.org/10.1016/j.ijpp.2013.03.007).

Brief communication: Do diseases cause enthesal changes at fibrous entheses?

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Abstract

Fibrous entheses have been widely used to study activity-related stress to infer behavioural patterns in past populations. Unlike their fibrocartilaginous counterparts, the effect of disease processes has been less widely discussed in the osteoarchaeological literature. This study uses a literature review to ascertain whether specific pathological changes should be taken into account when recording fibrous entheses. Due to the anatomical structure of fibrous entheses it was hypothesised that diseases which affect the structure of bone or the periosteum might be a factor in causing enthesal changes. A search of over one hundred terms was performed in PubMed with no year range stipulated, but no papers were found which related to enthesal changes caused by any of the expected pathological processes. The paucity of literature might be due to lack of symptoms in living individuals, it is therefore suggested that a systematic study of skeletal remains is performed using recently developed recording methods to determine if pathological conditions need to be taken into account when recording enthesal changes to study activity-related stress. The search did highlight a link between calcific tendinitis and cortical erosion at fibrous entheses, and it is recommended that this is taken into account when interpreting enthesal changes in past populations.

Keywords

osteoporosis, periostitis, Paget's disease, enthesopathy, enthesitis, tendinitis

Abbreviations.

EC – enthesal changes

1. Introduction

Fibrous entheses (for a description see Benjamin et al., 2002; Henderson et al., 2013; Jurmain et al., 2012) have been widely used to record activity-related stress (Cardoso, 2008; Chapman, 1997; Eshed et al., 2004; Hawkey, 1998; Hawkey and Merbs, 1995; Lieverse et al., 2009; Lovell and Dublenko, 1999; Molnar, 2006; Molnar, 2008; Molnar et al., 2010; Niimäki, 2012; Pany, 2005; Papathanasiou, 2005; Peterson, 1998; Schrader, 2012; Steen and Lane, 1998; Toyne, 2008; Weiss, 2003; Weiss, 2007; Weiss et al., 2010; Wysocki and Whittle, 2000; Zaübecki, 2009). It has recently been acknowledged (Henderson et al., 2013; Jurmain et al., 2012; Villotte, 2006; Villotte and Knüsel, 2013) that the clinical literature provides insufficient data to interpret changes (previously called musculoskeletal stress markers) at fibrous entheses. Changes to fibrocartilaginous entheses are known to be associated with, among other factors, physical stress and specific diseases, e.g. the seronegative spondyloarthropathies and diffuse idiopathic skeletal hyperostosis (Henderson, 2008; Jurmain et al., 2012; Kacki and Villotte, 2006). In contrast, no diseases are regularly taken into account when recording fibrous entheses.

Many of the powerful muscles attach to the skeleton at these entheses, e.g. the deltoid insertion. It is therefore important to understand any potential factors not directly associated with movement, that cause EC at fibrous entheses. These entheses rely on large footprints to attach the tendon to the bone, sometimes via the periosteum (Benjamin et al., 2002). It is therefore hypothesised that diseases affecting the structure of the bone or involving inflammation of the periosteum will cause EC at these entheses. The aim of this brief communication is to use a literature search to determine if there is clinical or palaeopathological literature to support this hypothesis.

2. Materials and Methods

A set of search terms was predefined (Table 1) based on a previous literature search and macroscopic skeletal analysis (Henderson, 2009), alongside terms used in the biomedical literature (Villotte and Knüsel, 2013). The specific pathological changes chosen all affect the structure of the

bone and, it was hypothesised, may therefore affect the integrity of the enthesis. Clinical literature was searched using the search tool PubMed (<http://www.ncbi.nlm.nih.gov/pubmed>) on the 15th of February, 2013. No start date was set for the search, thus searching all of PubMed. The aim of these search terms was to be as broad as possible, while restricting the search to fibrous entheses. Web of Knowledge was used, on the following day, to search for papers in the *International Journal of Osteoarchaeology* and the *Journal of Archaeological Science*. The *International Journal of Palaeopathology* was searched using Science Direct.

3. Results

The results (Table 1) show that of 141 searches performed in PubMed only 16 yielded results totalling 35 papers. Of these, seven related to the periodontal ligament (Albair et al., 1982; Bragger et al., 1990; Fernyhough and Page, 1983; Isidor et al., 1985; Karring et al., 1985; Luder and Zappa, 1998; Nishimura et al., 1989), and four related to experiments in non-human animals (Harris et al., 2002; Hashimoto et al., 2007; Miyakoshi et al., 2010; Suliman et al., 1997). Of the remaining papers, seven focus on one specific condition: calcific tendinitis (Cahir and Saifuddin, 2005; Chadwick, 1989; Dürr et al., 1997; El-Essawy and Vanhoenacker, 2012; Fritz et al., 1994; Hayes et al., 1987a; Ikegawa, 1996). No PubMed citations concern EC in relation to periostitis, Paget's disease or osteoporosis. The same 141 searches were performed in Web of Knowledge and yielded no results. No papers were found in *The International Journal of Paleopathology* using the terms “enthesis” or “entheses” and given the lack of papers in other journals, it was decided not to pursue this search. Instead, all titles and highlights of published papers were considered, but no papers appearing to discuss the diseases and entheses listed in Table 1 were found.

4. Discussion and conclusions

Understanding the factors which cause EC is important for the development of recording methods for EC and interpreting them. Due to the availability of clinical studies describing normal and abnormal fibrocartilaginous entheses, along with causes of EC, recent osteological research has focussed on developing methods for recording these entheses (Henderson et al., 2013). However, many of the large muscles of the body attach via fibrous entheses, e.g. the deltoid, pectoralis major and gluteus maximus insertions, as well as the tibialis anterior and posterior origins (Villotte, 2006). The anatomical structure of these entheses involves the attachment of the tendon to bone on diaphyses and meaphyses of long bones, as well as cranial muscles and the periodontal ligament

(Benjamin et al., 2002; Benjamin and Ralphs, 1997; Beresford, 1981; Hems and Tillmann, 2000). These entheses are characterised by collagen fibres (Benjamin et al., 2002; Cooper and Misol, 1970), which anchor tendon to bone or tendon to periosteum (Benjamin et al., 2002; François et al., 2001; Hems and Tillmann, 2000). During growth all these attachments occur via the periosteum, but often become direct to bone with age (Benjamin and Ralphs, 1999). The collagen fibres are anchored within bone matrix which is highly cellular and crossed with fibres (Benjamin et al., 2002; Beresford, 1981; Hems and Tillmann, 2000). Consequently, fibrous entheses which attach directly to the bone have roughened, striated and raised footprints, while those attaching via the periosteum do not leave such distinct marks (Benjamin et al., 2002; François et al., 2001).

This structure of fibrous entheses should mean that they are vulnerable to changes affecting the structural integrity of the periosteum (particularly during growth) and bone. However, no clinical observations were found to support this hypothesis. Changes at fibrous entheses in the living may be asymptomatic and their relatively large footprint may protect against rupture. It is possible that this is the reason for these results, rather than a real lack of EC in these individuals.

The lack of palaeopathological literature may support the clinical finding that fibrous EC are not associated with periostitis or other diseases. However, a non-systematic palaeopathological study has previously found EC in conjunction with periostitis and osteitis, while none were found in a skeleton with Paget's disease (Henderson, 2009a). Therefore, it is recommended that a systematic study of EC, using recently developed anatomically appropriate recording methods, should be undertaken in skeletons with diseases which affect the structure of bone or which involve the periosteum to determine if diseases should be taken into account when interpreting EC at fibrous entheses. To assist this, entheses should be more regularly recorded and EC (or the lack of EC) reported when they occur in conjunction with other pathological changes. This study would also provide valuable data on the effect of these diseases on the musculoskeletal system for both clinicians and palaeopathologists.

The only notable condition associated with fibrous entheses was found in the seven papers which discussed calcific tendinitis in relation to cortical defects at fibrous entheses. This has only rarely been discussed in the palaeopathological literature (Henderson, 2009b; Villotte et al., 2010; Villotte, 2008). In the clinical setting, women have a higher frequency than men but the mean age of presentation is similar, at approximately 50 years old (Flemming et al., 2003; Harvie et al., 2007), although it also occurs in subadults (Flemming et al., 2003; Oliva et al., 2012). It is important to distinguish between calcific tendinitis and, what has been termed, "insertional calcific tendinitis"

(Oliva et al., 2012), which is endochondral ossification causing enthesophytes, i.e. bone spurs, at fibrocartilaginous entheses (Benjamin et al., 2000). In calcific tendinitis calcium salts are deposited at the point of communication between the blood vessels of the soft and hard tissues (Resnick, 1995), but the aetiopathogenesis is not fully understood (Oliva et al., 2012). It is likely to involve trauma, either acute or repetitive, or chemical changes associated with endocrine diseases (particularly diabetes and thyroid and oestrogen metabolism disorders), but there may also be a genetic predisposition (Harvie et al., 2007; Oliva et al., 2012). The current hypothesis is that cellular disturbance occurs during healing leading to disruption of the normal cell differentiation process, in turn leading to chondrocytes or osteoblasts replacing the tenocytes (Oliva et al., 2012). Skeletal changes are rare or are rarely identified clinically (less than 10% of a PubMed search found papers associated with bony changes), but they do occur. These changes include cortical erosions (Cahir and Saifuddin, 2005; Chadwick, 1989; Flemming et al., 2003; Fritz et al., 1994; Hayes et al., 1987b), periosteal reactions, bone marrow extensions (Dürr et al., 1997; Flemming et al., 2003) and new bone formation (Chadwick, 1989), particularly woven bone (Hayes et al., 1987). Similar changes have been identified at fibrocartilaginous entheses (Chan et al., 2004), particularly (but not exclusively) in the rotator cuff (Diehl et al., 2011; Oliva et al., 2012; Porcellini et al., 2009; Uthoff and Loehr, 1997). Therefore, this should be considered as a possible differential diagnosis for cortical erosions, periosteal reaction or new bone formation at or adjacent to either fibrous or fibrocartilaginous entheses, especially if these changes affect only one enthesis or an endocrine disease is suspected.

In conclusion, further research involving more systematic recording of entheses is required to study if diseases cause EC at fibrous entheses. It is also important to consider calcific tendinitis as a differential diagnosis for EC at both fibrous and fibrocartilaginous entheses.

Acknowledgements

This research was funded by the Fundação para a Ciência e a Tecnologia, reference: SFRH/BPD82559/2011.

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Tables

Table 1. Set of defined searches with results. Input into PubMed took the form: “fibrous enthesis” AND rupture.

First part of search phrase	Conjunction	Second part of search phrase	PubMed	IJO and J. Arch Sci
fibrous enthesis		pathology		0
		periositis		0
		spur		0
		lytic		0
		enthesitis	The phrase "fibrous enthesis" w as not found in PubMed	0
		tendinopathy		0
		rupture		0
		enthesopathy		0
		hypertrophic osteoarthropathy		0
		Paget's disease		0
fibrous attachment		enthesiopathy		0
		enthesitis		0
		enthesopathy		0
		insertiopathy	The term "insertiopathy" w as not found in PubMed	0
		insertitis	The term "insertitis" w as not found in PubMed	0
		lytic		0
		osteoporosis		0
		pathology	Albair et al., 1982; Bragger et al., 1990; Fernyhough and Page, 1983; Harris et al., 2002; Hashimoto et al., 2007; Isidor et al., 1985; Karring et al., 1985; Luder and Zappa, 1998; Nishimura et al., 1989	0
		periositis		0
		rupture	Hashimoto et al., 2007	0
fibrous insertion		spur		0
		tendinitis		0
		tendinopathy		0
		tendonitis		0
		hypertrophic osteoarthropathy		0
		Paget's disease		0
		enthesiopathy		0
		enthesitis		0
		enthesopathy		0
		insertiopathy	The term "insertiopathy" w as not found in PubMed	0
deltoid insertion		insertitis	The term "insertitis" w as not found in PubMed	0
		lytic		0
		osteoporosis		0
		pathology	Dupont and Brown, 2012	0
		periositis		0
		rupture		0
		spur		0
		tendinitis	Dupont and Brown, 2012	0
		tendinopathy	Dupont and Brown, 2012	0
		tendonitis	Dupont and Brown, 2012	0
deltoid attachment		rupture	Warner and Parsons, 2001	0
		periositis		0
		spur		0
		lytic	Nakagawa et al., 2002	0
		enthesitis		0
		tendinopathy	Fritz et al., 1994	0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
deltoid tuberosity		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy	Lee et al., 2000	0
pectoralis major insertion		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
		lytic		0
pectoralis major attachment	AND	enthesitis	Cahir and Saifuddin, 2005; Chadwick, 1989; Durr et al., 1997; Essawy and Vanhoenacker, 2012; Hayes et al., 1987; Ikegawa, 1996	0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy	The phrase "pectoralis major attachment" w as not found in PubMed	0
		enthesopathy		0
teres major insertion		rupture		0
		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
teres major attachment		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy	The phrase "teres major attachment" not found in PubMed	0
gluteus maximus insertion		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
gluteus maximus attachment		spur		0
		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
		lytic		0
		enthesitis		0
tibialis anterior insertion		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
tibialis anterior attachment		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
		lytic		0
tibialis posterior insertion		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
tibialis posterior attachment		rupture		0
		periositis		0
		spur		0
		lytic		0
		enthesitis		0
		tendinopathy		0
		enthesopathy		0
		rupture		0
		periositis		0
		spur		0
deltoid		Page's disease		0
		osteoporosis	Anon., 1993; Briggs et al., 2007; Le Bellec et al., 2002; Revel et al., 1993; Roderer et al., 2011	0
pectoralis major		Page's disease		0
		osteoporosis	Braun et al., 2012; Kau et al., 2010	0
gluteus maximus		Page's disease	Dufourmentel and Peiron, 1984; Murakami et al., 1996; Sasaki et al., 1999; Tjalna et al., 2001	0
		osteoporosis		0
tibialis anterior		Page's disease		0
		osteoporosis	Chang et al., 2011; Malaviya, 2009; Miyakoshi et al., 2010; Sulman et al., 1997	0
tibialis posterior		Page's disease		0
		osteoporosis	Malaviya, 2009	0

