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Research Article

Analysis of nutritional disease in prehistory: The search for scurvy in antiquity and today

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ABSTRACT

In this paper, we discuss the issues surrounding the study of scurvy, or vitamin C deficiency, in paleopathology, and highlight the work of Donald Ortner in advancing this area of research. This micronutrient deficiency impacts collagen formation and results in damage to a variety of bodily tissues. While clinical manifestations are observed routinely, the lack of specific signatures on bone makes paleopathological diagnosis difficult. Rapid growth in infants, children, and subadults provides abundant remodeled tissue and an increase in vascularization that makes identification possible in younger segments of the population. However, diagnosis of scurvy in adults remains problematic, given that diagnostic lesions are strikingly similar to those associated with rickets, osteomalacia, and other conditions. We argue that this confounding factor underscores the need for a broader anthropological approach to scurvy research that expands beyond differential diagnosis to include more accurate reconstruction of diets and available resources, greater consideration of the possibility – even likelihood – of multiple nutrient deficiencies simultaneously affecting an individual, and the patterning of these deficiencies along lines of status, sex, and age.

1. Introduction

> "Circumstantial evidence is a very tricky thing," answered Holmes thoughtfully. "It may seem to point very straight to one thing, but if you shift your own point of view a little, you may find it pointing in an equally uncompromising manner to something entirely different." (Doyle, 1891, p. 402)

We have three objectives in this essay: to describe the history of the search for markers of scurvy and similar diet-related pathologies, to celebrate Donald J. Ortner's[1] role in unraveling the role of nutritional deficiencies in the adaptation and well-being of human populations, and to suggest broader anthropological perspectives useful in the study of these deficiencies. Over the course of his long and productive career, Ortner investigated tooth mutilation (Ortner, 1966), osteon remodeling (Ortner, 1975), hypothyroidism (Ortner and Hotz, 2005), carcinomas (Ortner et al., 1991), and nutritional diseases such as scurvy, rickets (Ortner and Mays, 1998), and iron deficiency anemia (Von Endt and Ortner, 1982). While we note limitations in the descriptive and diagnostic tradition of paleopathology that might limit more meaningful study of an elusive skeletal condition like scurvy, we also review the progress that has been made in scurvy diagnosis and highlight encouraging directions in broader anthropological research on this condition in antiquity.

2. Contextual background

Donald Ortner was a major force in the development of methodological and theoretical issues in paleopathology (Ortner, 1991, 2002, 2009; Ortner and Aufderheide, 1991). Ortner and Arthur C. Aufderheide organized a symposium held at the International Congress of Anthropological and Ethnological Sciences in Zagreb, Yugoslavia in July, 1988 that reviewed the then-current state of paleopathology with an eye to future developments. The proceedings of the conference, *Human Paleopathology: Current Syntheses and Future Options* (Ortner and Aufderheide, 1991) offers insights into a critical period in method and theory in paleopathology.

Ortner (1991) framed the theoretical and methodological perspective that defines his contribution to the discipline of

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[1] Donald J. Ortner, in addition to be a major force in paleopathology, was the epitome of a scientist and gentleman. While Don and I (GJA) were frequently at opposite ends of controversial issues in paleopathology, these were always dealt with as scientific matters and were never taken personally. No matter how heated the debate, we always greeted each other in a genuinely friendly manner. Don also played a significant role in mentoring three of my former students: Michael Blakey and Mark Mack were mentored by Don as undergraduates before they enrolled at the University of Massachusetts, and Molly Zuckerman before she enrolled as a graduate student at Emory. I will miss him and our scientific and social interactions.

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paleopathology. According to Ortner, the paleopathologist must answer two deceptively simple questions that require complex answers: "What is it?" and "What does it mean?" The answer to each question is fundamental. Given the difficulties of diagnosis in contemporary biomedicine despite its advanced technology, it is not surprising that paleopathologists would find diagnosis essential but also incredibly. This problem is compounded by the fact that paleopathologists have, until recently, frequently diagnosed pathological conditions without describing them in detail or discussing their distribution among skeletal elements. Naming a lesion without completing a differential diagnosis makes it difficult for other researchers to evaluate the validity of the original pathological determination and to standardize the diagnostic criteria for conditions with similar manifestations. An important aspect of paleopathology research, and one often glossed over by more descriptive, case-oriented approaches, is the second question: What does it mean? This question should extend beyond mechanisms and a list of associated symptoms to include a broader discussion of causal factors.

One research area that would benefit from this more inclusive approach is the study of scurvy. Ortner had an abiding interest in scurvy (Ortner et al., 2001; Ortner and Ericksen, 1997; Ortner et al., 1999) that served as a stimulus for renewed interest in the disease among paleopathologists. Unfortunately, there are only a few new techniques for diagnosing scurvy in skeletal remains. While the use of collagen extraction and analysis shows promise (Koon, 2010; Travis, 2008b), the application of standard macroscopic and microscopic techniques remain the current tools of choice (Brickley and Ives, 2006, 2008; Murray and Kodicek, 1949; Ortner and Ericksen, 1997). Even so, a review of health and disease in Britain (Roberts and Cox, 2003), with indisputable documentary evidence of morbidity and mortality from scurvy from the post-Medieval period (1550–1850 CE), found no skeletal evidence for vitamin C deficiency (Brickley and Ives, 2006). This creates a riddle for the paleopathologist, and the search for scurvy in earlier time periods has been just as elusive. Wells (1975, p. 756) describes the difficulties in diagnosing scurvy:

> It is not an easy disease to recognize in skeletal remains because its chief features are swollen, spongy, infected gums, and multiple subcutaneous and perifollicular hemorrhages giving rise to extensive purpuric and ecchymotic areas, anemia, lassitude and loss of muscle tone, and a tendency to sudden death on slight exertion. Since these are all manifestations which affect soft tissues rather than bones, it is understandable that its paleopathological recognition is very rare.

3. The scurvy enigma

Almost all animals, except for *Homo sapiens*, higher primates, fruit bats, bulbul birds, guinea pigs, and fish metabolically synthesize the enzyme L-gulonolactone oxidase that is required to convert glucose to vitamin C. Without the enzyme, vitamin C must be consumed from food sources. Vitamin C, a simple water-soluble molecule, is found in many plants, with especially high concentrations in citrus, tomatoes, potatoes, cabbages, and green peppers (García-Closas et al., 2004). This vitamin is essential for its capacity as an antioxidant and facilitator of many metabolic pathways. Healthy humans have stores of vitamin C that allow them to endure total dietary deprivation for 160–200 days (Hodges et al., 1971), after which symptoms of scurvy will manifest. Scholars have suggested that scurvy has caused more suffering among humans throughout history than any other disease of nutritional deficiency, aside from generalized famine (Carpenter, 1988). In fact, Stone (1966, p. 345) argued, "In the long period of human prehistory and history, scurvy has caused more deaths, created more human misery and has altered the course of history more than any other single cause."

Scurvy manifests as a generalized condition with a variety of symptoms that progress over time and can vary between individuals (Table 1). Evidence from clinical settings is measured primarily in behavioral features and changes in soft tissue. Behavioral features include fatigue (Hodges et al., 1971), loss of strength (asthenia) (Fain, 2005) and changes in emotion (Hodges et al., 1971). There are changes in soft tissue such as inflammatory hypertrophy of the gingiva (Fain, 2005) that leads to swollen gums (Hodges et al., 1971) and periodontal bleeding (Hirschmann and Raugi, 1999). Weakness in the vascular system leads to pinpoint bleeding of the skin (petechiae bleeding) (Hirschmann and Raugi, 1999) that causes dermal bleeding appearing as bruises or vascular purpura (Fain, 2005). Edema occurs as fluid accumulates in tissue (Hirschmann and Raugi, 1999; Hodges et al., 1971) with significant bleeding into the joint spaces as hemarthroses (Fain, 2005; Hirschmann and Raugi, 1999; Maat, 2004) and muscle as hematomas (Fain, 2005), leading to joint pain (arthralgia) and muscle pain (myalgia). Changes in hair with follicular hyperkeratosis (Hirschmann and Raugi, 1999) and congested follicles (Hodges et al., 1971) round out the soft tissue changes (Table 1).

Deficiency of vitamin C impacts the formation of collagen, the building block of connective tissue and the majority of the protein phase of bone. The body's global response to vitamin C deficiency is not surprising given its importance in collagen synthesis and the ubiquitous presence of collagen in human tissue. Collagen is created by fibroblasts and comprises over 25% of the body's total proteins; it is also the primary component of connective tissues. Collagen can form elongated fibrils that are the basic structure of fibrous tissues such as tendons, ligaments, and skin. Significant amounts of collagen provide structure to the cornea, cartilage, bone, blood vessels, intestines, and intervertebral discs. Collagen also forms the endomysium of muscle tissue. Impaired collagen structure results in defective formation of bone osteoid and blood vessel walls, resulting in weakened arteries and veins. Initially, there is a state of fatigue with malaise and lethargy, followed by hemorrhaging of small blood vessels that leave petechial spots on the skin, swollen joints with muscular aches and pains, and bleeding gums. Advanced stages of the condition are characterized by suppurating pus-producing wounds, neuropathy, and death (Hodges et al., 1971). Untreated scurvy is invariably fatal, but, death from scurvy is rare in modern times.

While the symptoms of vitamin C deficiency can be widespread and dramatic, the treatment is remarkably straightforward. All that is required for a full recovery is the resumption of normal vitamin C intake; as little as 6.5 mg of vitamin C will result in a slow but steady decline in symptoms of scurvy (Hodges et al., 1971). Given that the most diagnostic features (see below) of scurvy are found in soft tissue, the impact on bone is difficult to decipher. However, skeletal signs (Table 1) include antemortem tooth loss, abnormal dentin production, altered bone formation in subadults due to the inability of osteoblasts to produce the osteoid seam, and possibly elevated iron levels from increased absorption (Fain, 2005).

Though humans and human ancestors have long been susceptible to scurvy, attempts to determine the sources of scurvy in human populations came surprisingly late. Though most commonly attributed to European maritime explorers seeking out new lands through oceanic travel, scurvy was common in sailors long before the Age of Discovery (Wells, 1975). It appears to have been described initially during the fifth and fourth centuries BCE by Hippocrates (Stone, 1966), who documented the physical symptoms of pain in the lower extremities and gangrene of the gums (Hess, 1920; Hirsch, 1885). In the three centuries between 1500 and 1800 CE, a period characterized by an explosive increase in long duration maritime travel, scurvy is thought to have caused the death of at least

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