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Biocultural perspectives of vitamin D deficiency in the past

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ABSTRACT

Recently, significant attention has been paid to aspects of health associated with vitamin D deficiency in the current population; this interest has coincided with identification of paleopathological features of deficiency. Vitamin D is synthesised in the skin on exposure to sunlight, and is also obtained through a small number of dietary sources. There are a number of causes of deficiency, but those linked to sunlight and diet are most common. Latitude is important, but factors such as skin pigmentation, clothing, housing style, environmental conditions, work practices, and diet – including breastfeeding and weaning – also contribute. These factors have been the subject of a number of recent epidemiological studies illustrating diverse causes, many of which are directly linked to social and cultural factors. In this paper we review current clinical, epidemiological and bioarchaeological studies of vitamin D deficiency, highlighting the biocultural and life course dimensions that can be brought to studies of this condition in the past. We demonstrate that within the context of past communities vitamin D status represents a very significant source of information that can contribute to the analysis of the organisation, operation, and evolution of past human societies.

1. Introduction

There has recently been significant attention paid to aspects of health associated with vitamin D and the wide range of health factors linked to deficiency. The growth in interest has been widespread with extensive coverage in popular media sources and academic literature alike (e.g., Parker-Pope, 2010; Peterlik, 2012). The most extensive coverage of vitamin D deficiency has been in the biomedical and epidemiological literature; however, anthropologists have begun to explore vitamin D deficiency in an evolutionary framework (Chaplin and Jablonski, 2013; Jablonski and Chaplin, 2012). There have also been significant advances in identifying and understanding features associated with vitamin D deficiency in archaeological skeletal remains (reviewed by Brickley and Ives, 2008). The convergence of the availability of this information makes now an ideal time to use information on vitamin D deficiency in consideration of a wide range of issues operating in past societies.

Vitamin D deficiency is a product of biophysical variables – geographical latitude, skin pigmentation, and bioavailability of vitamin D in food sources – and cultural factors that determine individual and population risk such as infant and child feeding practices and behaviours that affect exposure to sunlight. Within populations these determinants are mediated by an individual's life course stage, social status, and inequalities that influence cultural behaviour and access to material resources such as food, clothing and time spent outside. Biocultural approaches, in which biological information is considered as interactional with the cultural context (Dufour, 2006; Goodman and Leatherman, 1998; Zuckerman and Armelagos, 2011), offer ideal means to investigate the paleopathology of diseases linked to vitamin D deficiency.

In addition to a biocultural framework, recent epidemiological studies of vitamin D deficiencies indicate that a life course approach (see Ben-Shlomo and Kuh, 2002) is also necessary in order to understand the changing manifestations of vitamin D deficiencies throughout an individual's lifetime. Although it does not fit the definition of a chronic disease, vitamin D deficiency can occur at any stage of life and may be recurrent. Deficiencies in infancy and childhood can not only result in rickets but may have implications for diseases throughout an individual's life course, including those that typically manifest later in life such as osteoporosis (Docio et al., 1998; Holick, 2004a). Recent developments in theoretical approaches therefore have considerable potential for use in studies of past vitamin D deficiency. As defined by Ben-Shlomo and Kuh (2002: 285) the life course approach to chronic disease epidemiology considers "...physical and social exposures during gestation, childhood, adolescence, young adulthood and later adult life. It includes studies of the biological, behavioural and psychosocial pathways that operate across an individual's life

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course, as well as across generations, to influence the development of chronic diseases." Research in contemporary groups demonstrates that vitamin D status is directly linked to a range of important biocultural factors (e.g., Henderson, 2005; Lim et al., 2012; Meltzer, 2007), and in past populations a life course approach can provide a direct window on the lived experiences of frequently under-represented groups at various stages of the life course (e.g., Prowse, 2010).

In this paper we review current clinical, epidemiological and bioarchaeological studies of vitamin D deficiency with a view to highlighting the biocultural and life course dimensions that can be brought to studies of these conditions in the past. Within the context of past communities vitamin D status represents a very significant source of information on a wide range of important social and cultural factors, many of which are not directly dealt with by written sources. Even when texts are available, groups such as the poor, women and children are often invisible; their life histories can only be understood through analysis of their remains. This paper puts forward a theoretical framework from which hypotheses can be tested using data on vitamin D deficiency. The objective of this paper is to illustrate how biocultural and life course frameworks are crucial in interpreting vitamin D deficiency in past populations, and in turn how investigations of vitamin D deficiency and associated metabolic bone diseases can contribute to analysis of the organisation, operation, and evolution of past human societies.

2. How do we obtain and use vitamin D?

Vitamin D is a hormone of the steroid family, whose main function is to maintain calcium homeostasis in the body (Holick, 2006). In humans, vitamin D may be synthesised from sunlight-induced – ultraviolet B (UVB) radiation conversion of sterol compounds that exist in the skin (Holick, 2005, 2008). This means vitamin D is synthesised in the skin when it is exposed to natural light. The exposure time required is generally low; Holick (2004b) estimates 5–10 min, two to three times per week with limited skin exposure. Alternatively vitamin D may be ingested through a small number of dietary sources (see Table 1). Either route, sunlight exposure or diet, requires that vitamin D enter the systemic circulation as a pro-hormone precursor, which must undergo two conversion steps, in the liver and kidney respectively, to become active and affect target tissues (Holick, 2006). Ingested or UVB synthesised pre-hormone vitamin D_3 is converted in the liver to 25-hydroxyvitamin D 25(OH)D, which is in turn taken up by the kidney and converted to the active circulating form of vitamin D, $1,25(OH)_2D$. Target tissues, such as bone, intestine, and kidney, express vitamin D receptors (VDR) that bind and take up the hormone allowing it to affect changes in cellular processes and regulation (Pike and Shevde, 2005). Failure at any step of the pre-hormone through target tissue pathway can lead to vitamin D deficiency.

Calcium homeostasis, the maintenance of circulating calcium levels within a narrow physiological range, is the main function of vitamin D, and deficiency in vitamin D limits the body's ability to absorb calcium and phosphorous from the intestines. It has been discovered that diets lacking in phosphorous and, particularly, calcium could lead to the development of metabolic bone diseases (Holick, 2006). Severe calcium deficiency can also lead to the development of secondary hyperparathyroidism and loss of phosphates, which itself has effects on the structure and overall health of bone. Parathyroid hormone (PTH) acts in several ways in response to low blood calcium levels. It acts on bone directly to increase resorption of calcium into the blood (Potts and Jüppner, 1998). In the kidney, PTH decreases the excretion of calcium to the urine and increases the reabsorption of calcium from the urine back into the blood (Potts and Jüppner, 1998). Finally, PTH acts indirectly to increase intestinal absorption of calcium by increasing the conversion of 25(OH)D to the active hormone in the kidney (Guyton and Hall, 2006). Such changes produce pathological changes in bone, particularly at the growth plates, resulting in various clinical manifestations of vitamin D deficiency.

Several causes of vitamin D deficiency have been identified which correspond to failures at various stages of the vitamin D pathway. These can be classified a number of ways, but the main categories can be considered: input insufficiencies of either sunlight (Holick, 1996) or dietary acquired vitamin D (Dent and Smith, 1969); inherited disorders for example Types I, II and III hereditary vitamin D dependent rickets (Glorieux and St-Arnaud, 2005; Malloy and Feldman, 2003; Holick, 2006); autosomal dominant hypophosphatemic rickets (Econs and McEnery, 1997) and X-linked hypophosphatemic rickets (HYP Consortium, 1995); malabsorption disorders, those that affect uptake of vitamin D or calcium from the intestine, for example cystic fibrosis (Hanly et al., 1985) or celiac disease (Melvin et al., 1970); renal or metabolic disorders such as chronic kidney disease (CKD) (Dusso et al., 2005) or liver failure (Dibble et al., 1981), which result in the loss of conversion enzymes necessary for the production of active vitamin D. Though clinically organised, each cause of vitamin D deficiency is subject to influence by culturally determined behaviours and processes. Input insufficiencies, like "nutritional" rickets, are the most common form of vitamin D related disease (Mithal et al., 2009) and are the most sensitive to socio-cultural influence. The inherited disorders are relatively rare (ADHR Consortium, 2000), but malabsorption disorders are slightly more common (Rewers, 2005; Anderson and Smith, 2003). Individuals with these conditions and those that could be linked to metabolic problems would not have survived for extended periods prior to the availability of modern medical treatments. It is possible that those with renal problems can live with their condition sufficiently long to develop clinically detectable bone changes without medical treatment (Coen et al., 1996); however, these would not be common in the past.

Basit (2013) provide a comprehensive review of genetic polymorphisms regulating vitamin D production, and potential links to a multitude of health conditions. Beyond what is already known about vitamin D's role in maintaining bone health, vitamin D is thought to have an important role in other biological systems such as the immune system, potentially affecting susceptibility to and ability to combat infectious diseases such as tuberculosis (Talat et al., 2010; Peterlik, 2012; Basit, 2013). Deficiencies and insufficiencies of vitamin D[1] have also been linked to a plethora of chronic illnesses including: type 1 diabetes, autoimmune diseases (e.g., multiple sclerosis), cardiovascular disease, schizophrenia, and various cancers (Basit, 2013; Grant, 2006; Holick, 2010; McGrath et al., 2010). Despite an upsurge of research on vitamin D and its links to these health conditions, additional evidence is still required. The trend to link almost every disease imaginable to vitamin D deficiency has recently been discussed by Peterlik (2012). Indeed, the IOM (2011) recently concluded that currently there is insufficient evidence for links between vitamin D and other health conditions beyond bone health on which to base changes to Dietary Reference Intakes (DRIs) for vitamin D. Of course that does not mean that more concrete evidence will not appear in the future, or that we should not be concerned with currently identified population deficiencies

[1] Vitamin D insufficiency is differentiated from deficiency in that vitamin D deficiency usually results in impaired bone mineralisation whereas insufficiency is defined as suboptimal vitamin D blood levels that may be associated with other disease outcomes. Defining suboptimal vitamin D serum levels is still debated; however, sometimes a cutoff value of 30 ng/mL of serum 25 (OH) D is used to define insufficiency, above which is considered to be optimal (Thacher and Clarke, 2011).