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Using modern human cortical bone distribution to test the systemic robusticity hypothesis

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ABSTRACT

The systemic robusticity hypothesis links the thickness of cortical bone in both the cranium and limb bones. This hypothesis posits that thick cortical bone is in part a systemic response to circulating hormones, such as growth hormone and thyroid hormone, possibly related to physical activity or cold climates. Although this hypothesis has gained popular traction, only rarely has robusticity of the cranium and postcranial skeleton been considered jointly. We acquired computed tomographic scans from associated crania, femora and humeri from single individuals representing 11 populations in Africa and North America ($n = 228$). Cortical thickness in the parietal, frontal and occipital bones and cortical bone area in limb bone diaphyses were analyzed using correlation, multiple regression and general linear models to test the hypothesis. Absolute thickness values from the crania were not correlated with cortical bone area of the femur or humerus, which is at odds with the systemic robusticity hypothesis. However, measures of cortical bone scaled by total vault thickness and limb cross-sectional area were positively correlated between the cranium and postcranium. When accounting for a range of potential confounding variables, including sex, age and body mass, variation in relative postcranial cortical bone area explained ~20% of variation in the proportion of cortical cranial bone thickness. While these findings provide limited support for the systemic robusticity hypothesis, cranial cortical thickness did not track climate or physical activity across populations. Thus, some of the variation in cranial cortical bone thickness in modern humans is attributable to systemic effects, but the driving force behind this effect remains obscure. Moreover, neither absolute nor proportional measures of cranial cortical bone thickness are positively correlated with total cranial bone thickness, complicating the extrapolation of these findings to extinct species where only cranial vault thickness has been measured.

1. Introduction

Cranial and postcranial robusticity play important but distinct roles in our understanding of hominin evolution and modern human variation. Cranial robusticity encompasses two facets, elevated cranial vault thickness (CVT) and well-developed (strongly expressed) cranial superstructures (CS). Postcranial robusticity primarily refers to increased strength of limb bones relative to size, but can include larger joint surface areas and musculoskeletal stress markers (Ruff et al., 1993; Churchill, 1998). Cranial robusticity figures prominently in Plio-Pleistocene hominin systematics (e.g., Kimbel and Rak, 1993), while postcranial robusticity has traditionally contributed to our understanding of locomotor and subsistence activity, particularly among Neanderthals and early modern humans (e.g., Trinkaus and Ruff, 1999a). The underlying causes of cranial robusticity remain speculative and poorly studied, such that the relative influence of genetics and environmental effects are not well known. More extensive investigation of postcranial robusticity reveals a complex interplay among genetics, behavior, climate, body size and ontogeny in the development of long bone robusticity (Pearson, 2000; Pearson and Lieberman, 2004; Wallace et al., 2010; Osipov et al., 2016). Though cranial and postcranial robusticity are generally treated as independent

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entities, they may intersect operationally in the expression of increased cortical bone development. This observation is the cornerstone of hypotheses that link the expression of this particular aspect of robusticity in the cranial and the appendicular skeletons through endocrine or allometric effects. Using computed tomographic (CT) data, this study evaluates the main prediction of any hypothesis postulating that enhanced cranial and postcranial cortical bone development are part of a systemic occurrence: measures of cortical bone are positively correlated in the cranial vault and limb bones within individuals.

1.1. Systemic endocrine mechanism for increased cortical bone deposition

Numerous hormones have been implicated in systemic control of bone modeling (reshaping of bone through modeling drifts that occurs primarily during growth), and remodeling (replacement of old or damaged bone tissue through a process of coupled resorption and formation) (Raggatt and Partridge, 2010; Kini and Nandeesh, 2012). The main hormonal influence on bone modeling is the growth hormone -insulin-like growth factor-1 (GH-IGF-1) axis, but additional hormones are also involved, including the thyroid hormone T_3 (triiodothyronine), parathyroid hormone, glucocorticosteroids, and sex hormones (Butler and Le Roith, 2001; Murphy and Williams, 2004; Rauch, 2005; Wit and Camacho-Hübner, 2011; Xu et al., 2011). T_3, GH and sex hormones are also involved in bone remodeling, as are calcitonin (also produced by the thyroid gland), parathyroid (PTH) and calcitriol hormones (Sommerfeldt and Rubin, 2001; Hadjidakis and Androulakis, 2006). The latter three hormones impact bone remodeling via their roles as mineral homeostasis regulators (Sommerfeldt and Rubin, 2001). T_3 promotes bone remodeling directly by stimulating osteoblasts and indirectly by inducing IGF-1, and possibly by influencing osteoclast activity (Yen, 2001; Bassett and Williams, 2003; Williams, 2013). The role of GH is to directly stimulate osteoblasts, to directly and indirectly stimulate osteoclasts, and to regulate IGF-1.

Disorders affecting the GH-IGF-I axis, as well as hyperthyroidism and hypothyroidism all have detrimental effects on bone growth and/or remodeling. Defects in the GH-IGF-I system, including GH deficiency, GH insensitivity and IGF-1 genetic mutation, lead to growth failure and midfacial hypoplasia (Högler and Shaw, 2010; David et al., 2011). Adult onset GH deficiency leads to reduced bone mineral density and osteoporosis (Holmes et al., 1994; Carroll et al., 1998). A critical evaluation of the data by Högler and Shaw (2010) raised concerns about the proper scaling of measures of bone structure, but still concluded that GH deficiency impairs formation of periosteal (cortical) bone in humans and that GH therapy reverses this effect by producing periosteal expansion via stimulation of IGF-1. Childhood hypothyroidism leads to growth retardation and delayed bone maturation whereas hyperthyroidism leads to growth acceleration and advanced bone age but premature fusion of epiphyseal growth plates and, in some instances, craniosynostosis (Waung et al., 2012). Hypothyroidism in adulthood results in decreased bone turnover and longer duration of the bone remodeling cycle leading to increased secondary mineralization. Hyperthyroidism has the opposite effect — high bone turnover leading to osteoporosis and reduced bone mineral density (Waung et al., 2012). The underlying etiology of GH-IGH-I-related short stature is multifactorial, but includes mutations affecting GH1, GHR, GHSR, GHRH-R, IFG-1, PROP1, POU1F1 and PTPN11 genes and may be sporadic or follow an autosomal recessive, autosomal dominant or x-linked pattern of inheritance, but can also be due to structural brain anomalies, trauma, and infection (Phillips and Cogan, 1994; Rosenfeld et al., 1994; Wajnrajch et al., 1996; Baumann and Maheshwari, 1997; Procter et al., 1998; Kofoed et al., 2003; Dattani and Preece, 2004; Pantel et al., 2006; Serra-Nédélec et al., 2012). These types of disorders are interesting because they can be viewed as extreme systemic effects whereas the systemic effects implicated in cold-climate adaptation and increased physical activity would be of a less dramatic nature.

1.2. Systemic robusticity hypotheses

Increased robusticity in cold climates was proposed for Late Pleistocene/Holocene Australians (Bulbeck, 2001) and for southern South Americans (Bernal et al., 2006; Perez et al., 2007). There was not a correlation between climatic variables and robusticity traits worldwide (Baab et al., 2010), but Bernal et al. (2006) specifically implicated extreme cold climates in the expression of robusticity, rather than identifying a robusticity cline related to temperature. In other words, there may be a climatic threshold beyond which a robust phenotype emerges. The arctic populations from Point Hope, Alaska do not, however, show similarly high levels of cranial robusticity despite their extreme arctic climate (Baab et al., 2010). Bernal et al. (2006) outlined a potential endocrine mechanism related to cold-adapted physiology. Indigenous cold climate-adapted groups exhibit elevated basal metabolic rates (Galloway et al., 2000; Leonard et al., 2002; Snodgrass et al., 2005), probably due to high levels of thyroid hormones, particularly T_4 (thyroxine), which can be converted to T_3 elsewhere in the body (Leonard et al., 1999, 2002). The role of T_3 in bone modeling/remodeling was reviewed above. Brothwell (1975) also suggested that an earlier onset or increased amount of hormone production during the pubertal growth spurt in Neanderthals could explain their cold-adapted, robust postcranial skeleton, as well as certain aspects of their facial skeleton (e.g., nasal and sinus morphology). Likewise, Churchill (1998) argued that numerous aspects of Neanderthal anatomy, including long bone shape and strength could be byproducts of selection for a hyperpolar body form related to endocrine shifts in this species.

Regarding physical activity, Lieberman (1996) found that pigs and armadillos that were exercised regularly had significantly higher cranial and postcranial dimensions than the non-exercising controls. Lieberman (1996) attributed the larger cross-sectional dimensions of the tibia to mechanical loading (a localized response), but this mechanism does not apply to the cranium. Rather, Lieberman (1996) attributed the exercise-induced increase in non-weight bearing postcranial bones and the cranium to an increase in circulating growth hormone (GH) that accompanies exercise (Schalch, 1967; Wallace et al., 2001). There has been little work looking directly at GH effects in the skull, but rats treated with recombinant human GH exhibited greater bone volume and improved mechanical strength of the bone than controls in healed artificial defects of the parietal bone (Cacciafesta et al., 2001). However, Copes et al. (2011) was unable to replicate Lieberman's (1996) result in mice, and Brown (1987) found no relationship between measures of CVT and two measures of femoral robusticity (transverse midshaft breadth and bicondylar breadth) in aboriginal Australians. Furthermore, changes in bone structural properties attributable to elevated GH are highly variable across the postcranial skeleton (Mosekilde et al., 1999). For example, while swimming leads to elevated GH in humans (Botoula et al., 2004), total cross-sectional area and cortical bone thickness of the tibia were only slightly higher in swimmers than controls and considerably less than in athletes training in higher impact sports (Nikander et al., 2006), probably because the former does not involve strain to the skeleton to the same degree as the latter. It is therefore uncertain how great the effect of elevated GH would be on cranial cortical bone in the absence of mechanical loading from joint moments and/or ground reaction forces.