Growing Pains: Opportunities to Adjust Phenotypic Trajectories in Childhood and Adolescence Complicate Studies of Developmental Plasticity in Late *Homo*

CAIT B. MCPHERSON*
Department of Pathology, University of New Mexico School of Medicine, Reginald Heber Fitz Hall, R. 335, University of New Mexico, Albuquerque, NM 87131, USA; ORCID: 0000-0001-8515-6159; cmcpherson@health.unm.edu

*corresponding author: Cait B. McPherson; *cmcpherson@health.unm.edu*

submitted: 27 May 2022; revised: 3 December 2022; revised: 14 December 2023; accepted: 26 January 2024

Guest Editors: Robert Acio Benitez (School of Anthropology, University of Arizona), John K. Murray (Institute of Human Origins, School of Human Evolution and Social Change, Arizona State University, and Susan C. Antón (Department of Anthropology, New York University)

Handling Editor in Chief: Katerina Harvati

ABSTRACT

Developmental plasticity, the regulation of ontogenesis in response to environmental cues, is hypothesized to evolve in spatially and temporally heterogeneous environments and may facilitate dispersal, novel habitat occupation, and niche construction. In contemporary human populations, exposure to environmental adversity informs a variety of developmental processes, including the pace and tempo of somatic growth and reproductive maturation. The ability to regulate these aspects of development in response to available resources may have been advantageous in the novel and marginal environments encountered by our ancestors, outweighing potential costs associated with elevated morbidity and mortality risks in adulthood. Yet only recently have biological anthropologists begun to systematically explore the relationship between developmental plasticity and patterns of growth, morbidity, and mortality in the human skeletal and hominin fossil records. What we currently know about hominin evolution suggests that, after the emergence of *Homo erectus*, climate change and repeated episodes of dispersal promoted the accumulation and maintenance of plastic traits in our genus. At the same time, the evolution of prolonged childhood and adolescence phases extended the hominin developmental lifecycle, creating additional opportunities for environmental signals to inform phenotypic trajectories. Experiences in childhood and adolescence may alter processes of skeletal growth, development, and maturation, potentially confounding efforts to associate episodes of early life stress with downstream phenotypic effects. Studies of developmental plasticity in late *Homo*, particularly tests of the Developmental Origins of Health and Disease (DOHaD) hypothesis, should carefully consider how opportunities for plasticity late in the developmental lifecycle contribute to patterns of phenotypic variation. Accounting for sources of bias amplified by our derived developmental pattern, including catch-up growth, swamping, and equifinality, will aid researchers in investigating the potential costs and benefits of plastic processes initiated during development and their impact on skeletal phenotype.

INTRODUCTION

In the context of human evolution, development is a biocultural process in which genetic and extragenetic (e.g., cultural, behavioral, epigenetic) systems of inheritance collectively shape phenotypic trajectories (Bogin et al. 2017; Nowell 2016; Rosenberg 2021; Tomasello 1999; West-Eberhard 2003). The Extended Evolutionary Synthesis (EES) provides a theoretical framework for examining the complex interplay between these systems while offering a diverse toolkit for interpreting patterns of phenotypic variation observed in the archaeological and paleoanthropological records (Antón and Kuzawa 2017; Murray et al. 2020; Prentiss 2021). Within this framework, developmental plasticity—the regulation of ontogenesis in response to

environmental cues—represents an important source of extragenetic variation that has historically been underexplored in human evolutionary biology (Antón and Kuzawa 2017; Kuzawa and Bragg 2012).

Studying hominin developmental plasticity in diverse ecological and temporal contexts may foster a more nuanced understanding of our species' evolutionary history, while enabling us to better address persistent questions about the nature of plasticity informed by environmental adversity. Human biologists are well aware of the importance of early life environments in setting long-term phenotypic trajectories (Gluckman and Hanson 2006; Gluckman et al. 2010; Kuzawa 2007; Kuzawa and Quinn 2009; Worthman and Kuzara 2005). Only recently have childhood and adolescent experiences received similar consideration in research related to contemporary and past patterns of human growth, behavior, and physiology (Avery et al. 2022; Gettler et al. 2022; Lewis 2022; Sisk and Gee 2022; Urlacher 2023). Studies of childhood and adolescent growth and development suggest that both maturational tempo and the pace and magnitude of somatic growth are sensitive to environmental cues received during these life history phases (Gawlik and Hochberg 2012; Gettler et al. 2022; Ellis, 2013, 2019; Kuzawa and Bragg 2012; Pantsiotou et al. 2008; Svefors 2019). Alterations to these processes may influence aspects of hard tissue growth, development, and maturation, contributing to patterns of phenotypic variation in skeletal and fossil samples associated with modern humans and hominins with similar life history characteristics. If unaccounted for in methodological design, plasticity late in the developmental lifecycle may confound efforts to associate episodes of early life adversity with aspects of adult phenotype (McPherson 2021), interpret evidence of frailty or resilience in response to developmental stress (McFadden and Oxenham 2020), or assess the costs of critical life history trade-offs (Temple 2019) using skeletal evidence. Examining developmental plasticity in the context of species-level life history patterns, which structure opportunities for environmental signals to influence phenotypic trajectories, is the first step in addressing this interpretive challenge.

The same environmental conditions that selected for derived characteristics of the hominin life history pattern (e.g., prolonged development, increased juvenile dependency) likely favored plastic capacities (Antón and Snodgrass 2012). Hominin populations experienced significant spatial and temporal variation throughout the Pleistocene as a result of climate change and dispersal, and phenotypic plasticity may have facilitated range expansion (Antón 2007; Antón et al. 2016; Grove 2014; Grove et al. 2015; Potts 2012; Scheiner and Holt 2012; Wells and Stock 2007). In theory, climatic instability acts as a selective force favoring the development of plastic capacities, which simultaneously buffer populations against emergent environmental challenges and equip them for successful dispersal into novel habitats during subsequent periods of stability (Grove 2014; Potts 2012; Wells and Stock 2007). It is possible that the development of greater behavioral and physiological plasticity in hominins both promoted and shaped the patterning of subsequent dispersal events, enhancing and maintaining plastic capacities in a lineage that likely already exhibited significant behavioral, dietary, and physiological flexibility (Grove 2011, 2014; Grove et al. 2015; Wells and Stock 2007). For these reasons, *Homo erectus*, the first member of our genus to disperse widely out of Africa, has been suggested as the potential origin of the enhanced developmental plasticity observed in modern human populations (Aiello and Antón 2012; Antón and Snodgrass 2012; Kuzawa and Bragg 2012; Wells and Stock 2007).

Traits associated with physiological tolerances, immune function, dietary and behavioral flexibility, and the pace and tempo of life history are often subject to stronger selection under conditions of dispersal and/or climate change (Bradshaw and Holzapfel 2008; Reed et al. 2010). Modern humans exhibit significant developmental plasticity within these trait categories, which may aid individuals in overcoming environmental adversity by regulating energetic trade-offs between core physiological functions (Kuzawa 2007; Kuzawa and Bragg 2012; Worthman and Kuzara 2005). In contemporary populations, developmental stress has been linked to phenotypic variation in patterns of somatic growth (Lampl and Schoen 2017; Wells 2016), neuroendocrine function (McEwen 2008; Raymond et al. 2021a; Thayer and Kuzawa 2014), immune function (Measelle and Ablow 2018), individual variation in disease susceptibility (Danese and McEwen 2012; Gluckman et al. 2009), brown adipose tissue metabolism (Levy et al. 2021), and the timing of key life history transitions (Chua et al. 2017; Gettler et al. 2015). Whether alternative phenotypes associated with early life adversity enhance fitness, represent accommodations to local environmental conditions, or are products of constrained or dysfunctional developmental processes remains a subject of intense debate within biological anthropology (see Snyder-Mackler et al. 2020). Research on developmental plasticity in contemporary populations is subject to significant methodological limitations, and traits that appear detrimental in Western social and ecological contexts may be advantageous to populations living in harsh and marginal environments (Ellis and del Giudice 2019; Ellis et al. 2017). Regardless, the impact of developmental plasticity on epidemiological and demographic trends in contemporary human populations has major implications for studies of phenotypic variation and health in both archaeological skeletal samples (Agarwal 2016; Gowland 2015) and hominin fossil samples (Antón and Kuzawa 2017).

Since hard tissue embodies phenotypic-environmental interactions experienced at both individual and generational timescales (Agarwal 2016; Gowland 2015), skeletal and fossil samples represent a record of hominin environmental histories extending into deep time. In modern humans, a variety of environmental factors influence development (e.g., activity patterns, nutrition, temperature, maternal condition, mortality risks), and plastic aspects of the skeletal system shaped by these factors (e.g., robusticity, diaphyseal growth, limb proportions) may be used to elucidate patterns of phenotypic variation in the hominin

fossil record historically attributed to genetic selection (Antón 2016; Antón and Kuzawa, 2017; Migliano and Guillon 2012). Variation in plastic traits within and between populations may indicate how, and under what circumstances, extragenetic factors contribute to processes of local adaptation and environmental accommodation (Antón and Kuzawa, 2017; Kuzawa and Bragg 2012). Additionally, the presence and patterning of skeletal biomarkers can provide information about the relationship between developmental stress and life history trade-offs related to growth, morbidity, and mortality (Amoroso and Garcia 2018; Davis et al. 2019; McFadden and Oxenham 2020; McPherson 2021; Smith 2006; Temple 2019). Yet interpreting phenotypic variation in an incomplete fossil record, let alone variation attributable to developmental plasticity, presents a variety of methodological challenges related to sample size, preservation, and spatial and temporal distribution (see Wood and Smith 2022). Studies of developmental plasticity in archaeological and paleoanthropological contexts are also subject to biases associated with mortuary samples (Woods et al. 1992) and processes of hard tissue development and maintenance (Clark et al. 1986; Hedges et al. 2007; Seifert and Watkins 1997).

Even in relatively well-preserved archaeological samples, derived features of the modern human life history pattern and aspects of hard tissue biology may interactively obscure associations between environmental cues and downstream phenotypic effects in the skeletal system (McPherson 2021). This interpretive problem may broadly apply to studies of developmental plasticity in late *Homo*. Distinct childhood and adolescent life history phases emerged during the Pleistocene as hominin populations encountered novel environmental challenges and developed increasingly complex social behaviors (Bogin 2003; Nowell and White 2010). The introduction of these phases to the hominin life history pattern widened the gap between infancy and adulthood (Bogin 1999), increasing the odds of disjuncture between developmental and adult environments (Botero et al. 2015). A growing body of research suggests that sensitive windows within these life history phases represent critical opportunities for environmental conditions to inform patterns of behavior, somatic growth, and reproductive maturation prior to biological and social transition into adulthood (Avery et al. 2022; Decrausaz and Cameron 2022; Gettler et al. 2022; Lewis 2022; Sisk and Gee 2022; Urlacher 2023). These sensitive windows may allow slowly-developing juveniles to respond to environmental change late in the developmental lifecycle, shaping patterns of skeletal variation in the process. Consequently, this underexplored source of variation has the potential to impact studies of hominin developmental plasticity that utilize skeletal biomarkers to evaluate associations between episodes developmental stress and aspects of adult phenotype. Tests of the Developmental Origins of Health and Disease (DOHaD) hypothesis (Barker 1995; Gluckman and Hanson 2006), a growing research focus in several branches of skeletal biology (Agarwal 2016; Antón et al. 2016; McFadden and Oxenham 2020; McKerracher et al. 2019), may be particularly vulnerable.

Specifically, plasticity late in the developmental lifecycle may amplify sources of bias related to equifinality, catch-up growth, and swamping, impeding efforts to identify the costs and benefits of physiological trade-offs occurring within and across life history phases. Trade-offs between somatic growth and reproductive investment can accelerate skeletal maturation, altering patterns of epiphyseal fusion and inhibiting diaphyseal growth (Onat and İşeri 1995; Onat and Ertem 1995), while trade-offs between growth and survival due to nutritional or immunological stress often delay skeletal maturation and slow or inhibit growth (Cavallo et al. 2021). The period of development in which these trade-offs occur, and their impact on the pace of growth and maturation, may influence costs associated with elevated morbidity and mortality risks (Beltrand et al. 2009; Nobili et al. 2008; Martin et al. 2017; Thompson et al. 2020; Visuthranukul et al. 2019). As different developmental trajectories are capable of producing similar outcomes (equifinality), late alterations to processes of growth and maturation can bias interpretation if not taken into consideration. Catch-up growth in childhood and adolescence may effectively overwrite evidence of constrained early growth in highly plastic skeletal elements like the long bone diaphyses, provided that sufficient resources are available (Christian and Smith 2018; Depauw and Oxley 2018; Llop-Vinolas et al. 2004; Pantsiotou et al. 2008; Svefors 2019). Alternatively, evidence of phenotypic responses to early life environments may be "swamped" by responses to cumulative, proximate signals of stress experienced downstream in development (Clark et al. 1986; Weibel et al. 2020). Compared to adults, subadults exhibit elevated bone turnover rates, which peak in infancy and again in adolescence (Hedges et al. 2007; Seifert and Watkins 1997). During the adolescent growth spurt, high rates of cortical bone remodeling may further contribute to loss of information about phenotypic-environmental interactions in prior life history phases.

The primary goal of this paper is to provide a framework for identifying potential sources of bias related to plasticity late in the developmental lifecycle, facilitating more robust analyses of the costs and benefits of developmental plasticity in different environmental contexts. To this end, it first provides a brief discussion of the relationship between life history pattern and plasticity. Then, it considers how models of developmental plasticity that inform anthropological studies typically emphasize the influence of early life environments on phenotypic trajectories, while associations between childhood and adolescent experiences and adult phenotype remain relatively understudied. Next, it examines characteristics of hominin life histories that impact how phenotypic-environmental interactions are recorded in hard tissue throughout the developmental lifecycle. Finally, it discusses current approaches to studying developmental plasticity designed to capture evidence of the presence, timing, and frequency of stress exposures. These approaches may allow skeletal biologists to work around biases related to variations in hard tissue

development and maintenance, so that plastic responses to adversity and their associated costs can be assessed in skeletal, and ideally, fossil samples.

EXAMINING DEVELOPMENTAL PLASTICITY IN EARLY LIFE AND BEYOND

All organisms retain some degree of phenotypic plasticity throughout the lifespan but are most sensitive to environmental influence during development (West-Eberhard 2003). The ability to regulate aspects of development in response to environmental cues may benefit species likely to experience temporal and spatial heterogeneity in their environment, but developmental constraints and informational deficits may limit the ability of plastic processes to produce contextually optimal traits (Hereford 2009; Scheiner and Holt 2011). Not all environmental signals are reliable indicators of future conditions, and even potentially informative signals may contribute to physiological dysfunction if there is significant disjuncture between environmental variables encountered in early life and later life history phases (Botero et al. 2015; Oates 2011). Such disjuncture may be more likely to impact species with slow life histories, and in primates, it is often the case that key features of developmental environments poorly resemble those experienced in adulthood (Botero et al. 2015). The interaction between aspects of environmental heterogeneity and life history characteristics may therefore influence whether plasticity or fixity is favored in the evolution of a particular trait (Hereford 2009; Scheiner and Holt 2011), and if plasticity is favored, how phenotypes might incorporate environmental information (see Del Giudice et al. 2011, Kuzawa 2005; Nettle and Bateson 2015). Indeed, the ways in which developing organisms experience spatial and temporal heterogeneity can impact how different biological mechanisms (e.g., neuroendocrine stress pathways, DNA methylation) operating at different timescales contribute to the expression of plastic traits (Burggren and Mueller 2019; West-Eberhard 2003).

Just as rates of growth and development vary within and across life history phases, phenotypic sensitivity to environmental signals varies throughout the developmental lifecycle (Bogin 2006; Stearns 1992; West-Eberhard 2003). Although models of developmental plasticity remain speculative, it has been theorized that variations in phenotypic sensitivity may help organisms cope with changes in the "informational properties" of their developmental environments, to include changes in the frequency and reliability of environmental signals throughout ontogeny (Fawcett and Frankenhuis 2015; Walasek et al. 2021). Since not all biological systems, or components of a system, are equally receptive to environmental influence at a particular point in time, infrequent, acute stressors may be less likely to produce dysfunction (West-Eberhard 2003). Conversely, heightened receptivity during periods of accelerated growth and life history transitions may aid organisms in regulating energetic expenditures (Ellis 2013; West-Eberhard 2003; Worthman and Kuzara 2005). Thus, the informational properties of environmental signals (e.g., intensity, duration, frequency), relative to a species' life history pattern, may influence the extent to which they inform phenotypic trajectories.

Plasticity within biological systems generally tends to decrease as organisms age due to energetic and physiological constraints (Murren et al. 2015; Stearns 1992; West-Eberhard, 2003). Environmental signals received in early life are, consequently, thought to exert the strongest influence over phenotypic trajectories (Gluckman et al. 2010; Murren et al. 2015; Wells 2019). This concept is fundamental to the DOHaD hypothesis (Barker 1995; Gluckman and Hanson 2006), which posits that stress experienced within the first 1,000 days after conception is a major contributor to patterns of morbidity and mortality in living human populations. It also informs current models of developmental plasticity (e.g., adaptive calibration, developmental constraints, maternal capital, phenotypic inertia, predictive adaptive response) that otherwise offer different explanatory frameworks for how the phenotype responds to environmental heterogeneity experienced at different timescales (Del Giudice et al. 2011; Kuzawa 2005; Nettle and Bateson 2015; Smith 1985; Wells 2019). Accordingly, the majority of recent studies dedicated to examining developmental plasticity in human and non-human primates have focused on the relationship between measures of adversity in early life and patterns of phenotypic variation in physiology and behavior (see Snyder-Mackler et al. 2020). These studies have collectively generated significant empirical evidence to support the theory that early life environments strongly influence adult phenotype, and that phenotypic-environmental interactions during this critical period may drive a substantial proportion of variation attributable to plasticity (Danese and McEwen 2012; Gluckman et al. 2009, 2010; Kuzawa and Quinn 2009; Thayer and Kuzawa 2014; Wells 2016).

However, plastic systems that do not completely canalize in early life remain open to the influence of environmental conditions experienced during subsequent periods of growth and development. Developmental plasticity in later life history phases is comparatively understudied in human biology, and this represents a fundamental gap in our understanding of the relationship between developmental environments and adult phenotypic outcomes. Research involving human and non-human animal populations suggests that sensitive developmental windows (SDWs)—periods of heightened receptivity to environmental feedback—exist for various systems and tissues throughout the life course (Amoroso and Garcia 2018; Burggren and Mueller 2019; Davis et al. 2019; Haapasalo et al. 2000, 2009; MacKelvie et al. 2002; Measelle and Ablow 2018). SDWs facilitate phenotypic-environmental interactions at multiple scales (e.g., tissues, systems, organisms), providing additional opportunities for environmental signals to influence different aspects of phenotype at different stages of development (Burggren and Mueller 2019; West-Eberhard 2003). Much attention has been focused on identifying SDWs in gestation and infancy, but there is growing evidence to suggest that additional SDWs for a variety of plastic traits may exist beyond early life. In human popu-

lations, adversity experienced within SDWs in childhood and adolescence may influence numerous aspects of adult phenotype, including bone density (Haapasalo et al. 2000, 2009; MacKelvie et al. 2002), stature (Bhutta et al. 2013; Christian and Smith 2018; Depauw and Oxley 2018; Pantsiotou et al. 2008; Svefors et al. 2019), brain structure and function (Fuhrmann et al. 2015), hypothalamic-pituitary-adrenal (HPA) axis activity (Raymond et al. 2021a, b), reproductive life history strategy (Brumbach et al. 2009), and endocrine function (Gettler et al. 2022). Adolescence and childhood may therefore provide opportunities for environmental conditions to adjust phenotypic trajectories in response to novel challenges, relaxed constraints, or variations in resource availability experienced late in the developmental lifecycle (Depauw and Oxley 2018; Fuhrmann et al. 2015; Gawlik and Hochberg 2012).

If this is the case, the addition of extended childhood and adolescent phases to the hominin life history pattern had major implications for the expression of developmental plasticity in hominins, and potentially, the production of phenotypic variation in the skeletal and fossil records. What we currently know about the evolution of the hominin life history pattern suggests that changes in the length, pace, and tempo of juvenile development may have created additional opportunities for environmental conditions to influence physiological trade-offs and the timing of key life history transitions. In particular, the relatively recent appearance of the adolescent growth spurt may have introduced opportunities for late adjustments to processes of somatic growth and reproductive maturation.

LIFE HISTORY PATTERN AND PLASTICITY

Infants and juveniles are underrepresented in hominin fossil assemblages, and the relative scarcity of subadult fossils places significant limitations on efforts to estimate changes in growth velocity throughout ontogeny and the timing of key developmental milestones. As a result, the evolutionary origins of the modern human developmental pattern remain uncertain. Modern humans have a unique life history pattern in which prolonged childhood and adolescent phases separate infancy and adulthood, approximately doubling the length of the subadult growth period relative to extant apes (Bogin 1999). Our greatly extended childhood may be further divided into two phases—an evolutionarily novel early childhood phase and a middle childhood phase that roughly corresponds with the juvenile phase of extant great apes (Bogin 1997, 1999; Thompson and Nelson 2011). The completion of weaning marks the beginning of the early childhood phase, which is characterized by extraordinarily rapid brain development and continuing parental dependency (Bogin 1997; Thompson and Nelson 2011). During the subsequent middle childhood phase, intensive social and cognitive development coincides with a period of moderate to slow somatic growth prior to the onset of puberty (Bogin 2003, 2006; Hochberg 2008).

There is disagreement over the timing of the transition between childhood phases in the anthropological literature, with the start of middle childhood variably defined using different dental (e.g., permanent M1 emergence) and brain growth (e.g., cessation of growth, 90% of adult growth) milestones, which typically occur between five and seven years post-birth (Thompson and Nelson 2011). Given the goals of the present paper, it is important to note that human life history phases do not perfectly correlate with extant ape and extinct hominin life history phases. Additionally, different approaches to defining subadult life history phases substantially complicate efforts to compare life history patterns within and across different hominin genera (see Thompson and Nelson 2011 for an extended discussion).

Human evolutionary biologists have generated a variety of theories to explain the emergence of our unique childhood phase, with the energetics of brain development and rapid pace of human reproduction frequently cited as potential selective factors (see Urlacher 2023). When total life span is controlled for, modern humans experience extended periods of synaptic development compared to other juvenile primates (e.g., chimpanzees, macaques), with intensive development occurring in the first five years of life (Liu et al. 2012). The protracted and metabolically expensive process of brain development, which continues into adulthood, is counterbalanced by reduced somatic growth rates, alloparenting behaviors, and technoeconomic systems that buffer against environmental perturbations (Bogin 1997; Leonard and Robertson 1994; Neubauer and Hublin 2012; Rosenberg 2021; Tomasello 1999). An additional benefit of a prolonged childhood is that it extends the window in which individuals may acquire essential social skills and cultural information through instruction, observation, and imaginative play—an essential process in a species characterized by social complexity and cultural diversity (Nowell 2016; Tomasello 1999). Similarly, adolescence may delay the onset of energetic costs associated with reproductive effort and maintenance of a larger body, while providing opportunities to practice potentially risky adult social and sexual behaviors without incurring the associated costs (Antón and Leigh 2003; Bogin 1997; Leigh 1996).

Comparative studies of hominin skeletal and dental development suggest it is unlikely that derived characteristics of the modern human developmental pattern, including an extended period of moderate growth preceding puberty followed by an adolescent growth spurt, evolved in our genus prior to 1.5 Ma (Bogin 2020; Schwartz 2012; Thompson and Nelson 2011). Indeed, distinct early childhood and adolescent life history phases may have evolved relatively recently within our lineage, with adolescence emerging only in the last 100,000 years (Bogin 1999, 2020; Gawlik and Hochberg 2012; Hochberg 2012; Smith 1992; Smith and Tompkins 1995; Thompson and Nelson 2011). Patterns of skeletal and dental development exhibited by australopithecines and early *Homo* indicate that they likely experienced relatively brief periods of juvenile dependency and accelerated maturation in comparison to late *Homo* (Dean et al. 2001; Hemmer 2014; Schwartz 2012). Gracile australopithecines matured at rates similar to extant great apes, potentially reaching sexual maturity around eight to nine

years of age, in comparison to 9.8 years for chimpanzees (Hemmer 2014). Similarly, *H. habilis* potentially reached reproductive maturity as early as ten years of age (Hemmer 2014). It has been theorized that *H. habilis*, whose fossil record ends around 1.9 mya, may have been the first hominin to experience a distinct childhood phase involving rapid early growth followed by a period of decelerated, stable somatic growth (Bogin 1999, 2006). However, Thompson and Nelson (2011) argue that this is unlikely, as estimated rates of dental development in *H. habilis* are similar to those of australopithecines.

The early life of *H. erectus,* the first known member of our genus to migrate out of Africa, is poorly understood due to limited subadult remains. Studies of these suggest that it may have been the first hominin species to experience a brief childhood phase (Bogin 2006; Thompson and Nelson 2011) and a growth spurt preceding adulthood (Antón and Leigh 2003; Gawlik and Hochberg 2012; Leigh 2004; Tardieu 1998). In comparison to *H. habilis*, *H. erectus* may have experienced an even shorter period of infancy relative to total lifespan, followed by brief childhood phase lasting approximately a year (Bogin 2006; Gawlik and Hochberg 2012). Studies of juvenile cranial capacity suggest a rate of relative brain growth slower than chimpanzees and faster than modern humans (Bogin 2006; Gawlik and Hochberg 2012; Thompson and Nelson 2011). Similarly, *H. erectus* may have exhibited an intermediate pattern of somatic growth, with development occurring over a period similar to (Thompson and Nelson 2011) or slightly longer than (Dean and Smith 2009) that exhibited by modern great apes. Using estimated body mass and brain mass to predict life history characteristics, Hemmer (2014) hypothesized that *H. erectus* females likely reached sexual maturity and first gave birth around 13 and 16 years of age, respectively. These estimates are later than those for early *Homo* (9.5 and 12.5 years for *H. habilis*), but significantly earlier than estimates for Middle Pleistocene *Homo* (14.5, 18 years) and observed values derived from modern human populations (16.5, 19.3 years) (Hemmer 2014). The rate of growth experienced by juvenile *H. erectus* in their early teens was likely quite high and may have involved spurts in growth similar to those observed in other primate species (Leigh 2004; Tardieu 1998). Although limited by sample size, a study of facial and mandibular growth velocity in *H. erectus* by Antón and Leigh (2003) suggests that the presence of an adolescent growth spurt should not be ruled out in this species. However, based on available lines of dental, cranial, and postcranial evidence, there is currently limited empirical support for a delayed adolescent growth spurt in *H. erectus* characteristic of the modern human life history pattern (Bogin 2020; Schwartz 2012).

When this trait emerged in our genus is unknown, and it is possible that it is unique to *H. sapiens*. The body of existing literature suggests that both Neanderthals and Middle Pleistocene *Homo* experienced prolonged childhood and adolescent phases in comparison to early *Homo*, although these phases were likely compressed in comparison to modern *H. sapiens* (Bermúdez de Castro et al. 2003; Bogin 1999, 2003; Ramirez-Rozzi and Bermúdez de Castro 2004; Tardieu 1998; Thompson and Nelson 2011). Rates of dental development may have been faster in Middle Pleistocene *Homo*, potentially indicative of an accelerated developmental lifecycle (Ramirez-Rozzi and Bermúdez de Castro 2004). An analysis of internal molar microstructure by Macchiarelli and colleagues (2006) suggests that Neanderthals may have developed at a pace similar to modern humans, with a distinct early childhood phase. This theory is further supported by a study of Neanderthal endocranial development conducted by Ponce de León and colleagues (2016) which found that trajectories of Neanderthal brain growth likely resembled those of modern humans from the Late Pleistocene. However, lines of dental evidence provide increasing support for a relatively accelerated pace of growth and development in Neanderthals (see Schwartz 2012). Smith and colleagues' (2010) analysis of dental crown development in Neanderthals and *H. sapiens* indicates that Neanderthal dental development was likely faster than modern human dental development. Meanwhile, dental development in Middle Paleolithic *H. sapiens* from Qafzeh, Israel (90–100 kya) and Jebel Irhoud, Morocco (160 kya) may have proceeded at a pace more similar to contemporary *H. sapiens* populations. Additionally, skeletal evidence indicates that completion of somatic growth in Neanderthals likely occurred in the mid-teens, from which it can be inferred that they experienced higher rates of adolescent growth than modern humans (Thompson and Nelson 2011). Based on what we currently know about the pace and tempo of adolescent development in early modern humans and Neanderthals, they both may have experienced adolescent growth spurts (Thompson and Nelson 2011), but the timing and velocity of these growth spurts remains uncertain.

In general, this evidence suggests a trend toward slower life histories and larger generational gaps in the hominin lineage, with proportionately longer childhoods and adolescence relative to total potential lifespan (Figure 1). Significant differences in the relative lengths of these phases between *H. erectus* and modern humans further suggest that our derived life history pattern is likely a relatively recent development, potentially emerging in the middle to late Pleistocene (Schwartz 2012). Although the widening gap between infancy and adulthood was potentially a byproduct of prolonged brain development, it increased opportunities for environmental cues to cumulatively adjust developmental trajectories in a variety of biological systems (Gawlik and Hochberg 2012; Leonard and Robertson 1994; Neubauer and Hublin 2012). Of equal importance, the late addition of childhood and adolescence to the hominin life history pattern may have also introduced sensitive windows at key stages in the developmental lifecycle.

Sisk and Gee (2022) suggest that it may be helpful to conceptualize adolescence itself as a sensitive window in the context of modern human life history, as plasticity is enhanced across multiple systems during this phase of development. As previously discussed, coping with the informational properties of developmental environments can be

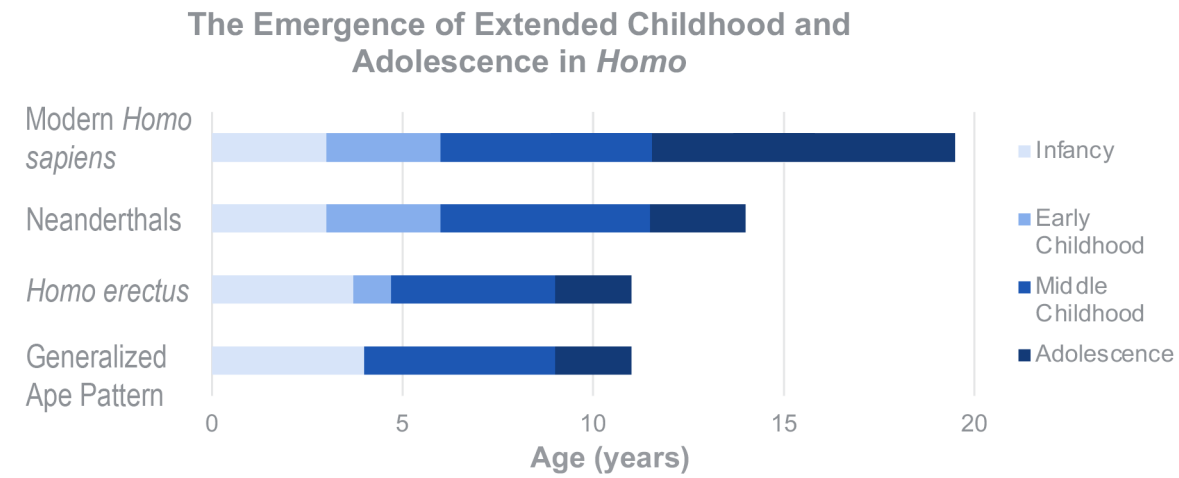

Figure 1. Length of childhood and adolescence relative to total potential lifespan in hominins.

a significant challenge for organisms that experience spatial and temporal heterogeneity throughout development (Fawcett and Frankenhuis 2015). The addition of sensitive windows later in the developmental lifecycle of hominins may have been necessary to overcome problems associated with signal reliability, especially in systems that experience extended development across multiple life history stages.

During the adolescent growth spurt, defined by a peak in growth velocity, individuals may experience significant increases in bone turnover rates (Hedges et al. 2007; Seifert and Watkins 1997) and the linear dimensions of some skeletal elements, to include the long bone diaphyses and mandibular ramus (Antón and Leigh 2003; Wells 2016). Unsurprisingly, a significant percentage of adult stature may be attained during adolescence, as much as 15%–25% in girls (Christian and Smith 2018). The pace and tempo of somatic growth and reproduction are closely linked to nutritional status, but in modern human populations, signals of energetic constraint in early life may not be as informative as proximate signals of resource availability in late childhood and adolescence (Botero et al. 2015; Ellis et al. 2009; Kuzawa and Bragg 2012). For this reason, it has been theorized that environmental cues received in childhood may have a particularly strong effect on growth trajectories, while sensitive windows in adolescence may influence trade-offs associated with reproductive life history transitions and longevity (Gawlik and Hochberg 2012).

Since the adolescent growth spurt represents a major energetic expenditure, signals of environmental conditions during late growth phases may allow individuals to adjust the pace and magnitude of somatic growth in response to available resources and extrinsic mortality risks. Life history trade-offs between growth, survival, and reproductive investment shape patterns of variation in stature and body size observed in both present and past populations (Kuzawa and Bragg 2012; Migliano and Guillon 2012; Schwartz 2012; Stearns 1992; Wells 2016), and late adjustments to growth trajectories may contribute to phenotypic variation in skeletal and fossil samples. Thompson and colleagues (2020) argue that, until very recently in our species' history, human populations typically exhibited growth trajectories shaped by exposure to chronic nutritional and immunological stressors throughout the developmental lifecycle. Slow rates of somatic growth occurring over an extended duration were common, but if conditions allowed, episodes of accelerated growth could make up for stature lost as a consequence of prior stress events (Thompson et al. 2020). Alternatively, an accelerated pace of development resulting in reduced stature and earlier age at reproductive maturity may have characterized growth trajectories in environments associated with limited resources and high extrinsic mortality risks, (Kuzawa and Bragg 2012; Migliano and Guillon 2012). In environments with adequate resources but high extrinsic mortality risks, accelerated development might also have occurred without associated reductions in stature (Schwartz 2012). Studies of maturational tempo in modern human populations indicate that there is significant flexibility in the tempo of reproductive maturation as it relates to somatic growth (Ellis 2013, 2019; Kuzawa and Bragg 2012). Collectively, they suggest the existence of plastic mechanisms that compensate for trade-offs in somatic growth and reproduction through modifications to developmental tempo.

Under the right environmental circumstances, these mechanisms may also provide individuals with opportunities to overcome the effect of early adversity. Studies of pubertal development indicate that early onset of puberty in girls may result in a relative delay in peak height velocity (PHV), but not compromised adult height (Llop-Vinolas et al. 2004; Pantsiotou et al. 2008). In addition, a recent longitudinal study of a rural Bangladeshi cohort found that children who exhibited recovery from early life stunting exhibited pubertal development similar to non-stunted children (Svefors 2019). However, recovery may only be possible in environments where there are sufficient resources available. For a more comprehensive discussion of factors con-

tributing to patterns of somatic growth in contemporary and past human populations, refer to the following articles: Migliano and Guillon (2012), Steckel (2009), Thompson et al. (2020), and Wells (2016).

The ability of humans—and potentially, other hominins—to fine-tune growth trajectories across multiple life history stages may significantly complicate efforts to relate the effects of developmental environments to adult phenotypic outcomes. In order to understand the relationship between developmental plasticity and skeletal variation, we must therefore develop methodological approaches that allow us to better interpret patterns of phenotypic-environmental interactions occurring across the life course.

THE SKELETAL SYSTEM AS A RECORD OF PHENOTYPIC-ENVIRONMENTAL INTERACTIONS

As Temple (2019) cautions, evidence of plasticity in the skeletal system may not be adaptive in and of itself, but instead may indicate the presence of adaptive plastic processes in systems that do not preserve in the archaeological record. Plasticity in hard tissue may largely reflect life history trade-offs that enhance survival and reproduction by delaying or inhibiting growth, often at the cost of enhanced increased morbidity and mortality risk in adulthood (Temple 2019). Processes of compensatory growth and phenotypic adjustment late in development may obscure hard tissue evidence of trade-offs initiated in early life, while also incurring delayed costs of their own that may not be apparent until adulthood (Martin et al. 2017; Nobili et al. 2008). Since skeletal elements vary in the length of their developmental life cycles and receptivity to environmental cues, they may differentially embody the effects of these trade-offs (McPherson 2021).

Despite these challenges, archaeological and paleoanthropological studies have much to contribute to ongoing debates about the potential costs and benefits of phenotypic plasticity. The skeletal system can simultaneously record evidence of plastic responses to stress at different stages in development, relevant phenotypic outcomes, and proxy measures of health and fitness. Suites of carefully selected biomarkers can be used to identify links between the timing of stress exposures in an individual's developmental life cycle and patterns of growth, morbidity, and mortality at the population level. In turn, these patterns may indicate periods of development in which important life history trade-offs are initiated (Amoroso and Garcia 2018; Moes et al. 2022; Newman and Gowland 2015; Temple 2019; Watts 2013, 2015; Weisensee 2013), and whether these trade-offs contributed to frailty or resilience in a particular environmental context (McFadden and Oxenham 2020). Studies of developmental plasticity are enhanced by the use of multi-marker models that incorporate biomarkers indicative of stress experienced at different life history phases, to include measures of acute stress (e.g., enamel defects, Harris lines) and chronic stress experienced within relatively discrete periods of development (e.g., vertebral neural canal dimensions, measures of dental and cranial fluctuating asymmetry) (McPherson 2021). By capturing evidence of stress experienced within developmental periods of interest, associations between the timing and patterning of stress events, phenotypic outcomes of interest (e.g., estimated height) and indicators of fitness (e.g., estimated age at death, cause of death, evidence of disease stress) can be more rigorously tested.

Examining how stress is embodied in elements that are differentially responsive to environmental cues at different periods in development may help skeletal biologists account for potential information loss due to compensatory plasticity, catch-up growth, or swamping. This approach is briefly outlined in the following sections, which summarize how current multi-marker models of skeletal stress may be used to develop more nuanced tests of hypotheses related to adaptive developmental plasticity and DOHaD. These approaches to interpreting developmental stress, which prioritize signal timing, frequency, and patterning, may aid researchers in more clearly relating episodes of early life stress to adult phenotypic outcomes of interest.

INDICATORS OF LIFE HISTORY TRADE-OFFS IN THE SKELETAL SYSTEM

While a variety of regulatory mechanisms may synergistically contribute to plastic developmental processes, not all of them are equally visible in the skeletal record. Although this problematizes efforts to reconstruct developmental processes in historical and archaeological contexts, one potential regulatory mechanism—the stress response system—is directly involved in the production of a diverse array of hard tissue biomarkers (Donatti et al. 2011; Lorentz et al. 2019; Mazziotti and Giustina 2013; Temple and Edes 2022). Neuroendocrine stress pathways involving the hypothalamo-pituitary-adrenal (HPA) axis are hypothesized to regulate processes of growth and development in a variety of biological systems (Crespi and Denver 2005; Ponzi et al. 2020; Worthman and Kuzara 2005). Activation of the stress response system as a reaction to a perceived threat downregulates the production of growth hormone, in effect driving trade-offs between growth and survival that may influence the timing of life history transitions (Ellis 2013; Worthman and Kuzara 2005). Within this framework, stress hormones (e.g., corticosteroids) act as intermediary signals of environmental adversity, providing developing systems with information about potential challenges in order to optimize the allocation of energetic resources (Ellis 2013; Lu et al. 2018). Thus, stress provides organisms with essential environmental context that may inform key life history trade-offs while enacting costs and applying constraints to developmental processes.

A key advantage of studying developmental plasticity in the skeletal system is its modularity—the division of the system into individual elements and functional units. Just as organisms exhibit different degrees of sensitivity to environmental signals throughout their lifespans, so too do skeletal elements within their own developmental lifecycles. In the skeletal system, interactions between corticosteroids and growth hormone (GH) impact hard tissue metab-

olism and development at varying time scales, producing artifacts that can be used to estimate the timing of these interactions relative to an individual's chronological age (Donatti et al. 2011; Lorentz et al. 2019; Mazziotti and Giustina 2013). Using biomarkers to link stress exposures to downstream phenotypic outcomes is a particular strength of skeletal biology, exemplified by traditional paleopathological approaches. In the case of biomarkers associated with acute stress episodes (e.g., enamel defects), this can often be accomplished with a relatively high degree of precision (Davis et al. 2019; Smith 2006; Smith et al. 2006). Other biomarkers reflect cumulative stress exposures experienced during relatively narrow windows in early life (e.g., fluctuating asymmetry in deciduous teeth and portions of the basicranium), or periods encompassing early life and early childhood (e.g., vertebral neural canal diameters, measures of fluctuating asymmetry in permanent teeth), while others reflect stress experienced over extended developmental timelines (e.g., long bone diaphyseal measures, vertebral body height).

Since a variety of environmental signals associated with adversity (e.g., resource, disease, and psychosocial stress) use corticosteroids as intermediary signals, it is not always possible to work backwards from a stress biomarker to identify its precise cause. However, by assessing the patterning of stress embodied in skeletal elements, and the estimated timing and frequency of stress episodes relative to an individual's chronological age, it may be possible to partly reconstruct aspects of their developmental environment. Skeletal development in modern humans is well documented in the biological literature at the level of both elements and functional units, including known periods of accelerated growth, sequences of epiphyseal fusion, and rates of remodeling (Cunningham et al. 2017; Lewis 2017). Although features of some hominin life histories are incompletely understood, sufficient data is available to estimate patterns of skeletal and dental development in several other species of late *Homo* (Hemmer 2014; Macchiarelli 2006; Nava 2020; Thompson and Nelson 2011). Although necessarily speculative in the case of extinct hominins, knowledge of hard tissue development makes it possible to identify elements most likely to record evidence of phenotypic-environmental interactions within the developmental periods most relevant to the aims of a study.

RELATING ENVIRONMENTAL SIGNALS TO PHENOTYPIC OUTCOMES

In contexts where other factors contributing to phenotypic variation within a trait (e.g., cultural, behavioral, genetic) are incompletely known, observed variation cannot necessarily be attributed to plastic developmental processes without a way of identifying the presence and timing of environmental signals involved in phenotypic-environmental interactions. For this reason, studies of developmental plasticity that utilize skeletal samples should ideally follow the framework proposed by Doughty and Resnik (2004) and include proxy measures of three categories of information—the timing of stress exposures, phenotypic products related to those stress exposures, and when applicable, measures of fitness. In studies designed to not only identify the presence of plastic variation, but assess its potential adaptive value, the same biomarker should never be used as both evidence of a plastic response and a proxy measure of fitness. This is especially relevant to studies of plasticity in somatic growth, as recent research has challenged associations between measures of constrained growth and indicators of health and fitness (Scheffler and Hermanussen 2021). Additionally, in the absence of information about signal timing, it may not be possible to determine whether stress experienced within a particular developmental episode inhibited growth or acted as an informational signal influencing future development (McPherson 2021). Since these processes are not necessarily mutually exclusive, multi-marker models involving elements that differentially embody stress may be needed to disentangle their effects.

In the context of life history theory, biomarkers associated with acute stress episodes (e.g., enamel defects, Harris lines) signal the dynamic reallocation of energetic resources involved in short-term trade-offs between growth and other expensive physiological functions (Temple 2019; Worthman and Kuzara 2005). If experienced within relevant sensitive windows, these acute stress episodes may also trigger additional life history trade-offs associated with phenotypic products that are only observable downstream, potentially in later life history stages (e.g., associations between early life stress and accelerated life histories) (McPherson 2021; Temple 2019). Meanwhile, measures associated with long-term growth trajectories (e.g., patterns of stunting, estimated body mass, sexual dimorphism) represent phenotypic end products informed by a variety of genetic and extragenetic processes, with plastic processes contributing to observed patterns of variation (Agarwal 2016). Early differentiating, highly conservative elements with limited potential for remodeling may act as "time capsules" of early life environments, retaining information about phenotypic-environmental interactions into adulthood (McPherson 2021). Conversely, elements with extended developmental lifecycles may remain responsive to environmental cues beyond early life, with the potential for catch-up growth in response to improved conditions. For instance, long bone diaphyses and vertebral bodies exhibit multiple periods of intensive growth occurring across life history phases and capture information about phenotypic-environmental interactions over an extended period (Cunningham et al. 2017; Dimeglio and Canavese 2012; Svefors et al. 2019; Wells 2016).

Loss of data due to taphonomic processes is a major limiting factor in studies of developmental plasticity involving skeletal and fossil samples, and bone mineral density is thought to be a key factor in the survival and relative preservation of skeletal elements (Biehler-Gomez et al. 2022; Willey et al. 1997). Elements with a high proportion of trabecular bone (e.g., vertebrae, ribs, sternum, appendicular elements) are typically less likely to survive than those with greater cortical density (e.g., long bone diaphyses) or the dentition (Biehler-Gomez et al. 2022; Stojanowski et al.

2002). Knowing that researchers working in archaeological and paleoanthropological contexts may be severely limited in the number and quality of skeletal elements available, what follows is a brief (and non-comprehensive) discussion of recent studies of developmental plasticity in skeletal biology that may serve as useful models (see McPherson 2021 for an extended discussion). Several of these involve explicit tests of the DOHaD hypothesis, while others focus on identifying periods of development in which potential life history trade-offs involving growth and survival are initiated.

A variety of recent studies in skeletal biology have utilized enamel defects to evaluate associations between the timing and frequency of developmental stress episodes and adult phenotypic outcomes, to include age at death (Armelagos et al. 2009; Garland 2020), estimated stature and age at death (Temple 2014, 2019), and aspects of behavioral phenotype (Davis et al. 2019). Most recently, Moes and colleagues (2022) used enamel defects to assess relationships between episodes of early life stress and cranial fluctuating asymmetry (FA), a measure of developmental instability, in a sample of 48 individuals from Colonial-era cemeteries in Mexico City. After dividing permanent canine crowns into three segments representing different periods in development (~1–2.5, 2.5–4, and 4–5.5 years), they assessed the presence and frequency of enamel defects within these periods and their association with measures of cranial FA (Moes et al. 2022). Contrary to their expectations, environmental stress experienced between 4 and 5.5 years had a unique and potentially durable impact on developmental instability, which they hypothesized may relate to physiological trade-offs in early childhood associated with the high energetic demands of brain development (Kuzawa et al. 2014; Moes et al. 2022).

The results of Moes and colleagues' (2022) analysis suggest that the informational properties of environmental signals should be carefully considered by investigators and reaffirm the importance of timing and frequency as key factors influencing plastic phenotypic responses (Garland 2020; McPherson 2021; Temple 2019). Furthermore, their results support the theory that stressors experienced after early life may significantly impact phenotypic trajectories. Although relatively underutilized, measures of cranial FA may have considerable utility as proxies of cumulative developmental stress and have been previously used by skeletal biologists to evaluate associations between early life environments, age at death, and cause of death (Chovalopoulou 2017; Weisensee 2013). The development of the cranium is complex, with functional units undergoing different rates of growth and exhibiting varying degrees of plasticity in response to environmental signals (Cunningham et al. 2017). Conservative elements of the cranium that exhibit limited capacity for remodeling, including more durable portions of the basicranium, may be particularly suitable for assessing early life stress in future studies (Chovaloupoulou et al. 2017).

Vertebrae are less likely to survive intact in skeletal and fossil assemblages, but vertebral biomarkers should also be considered for inclusion in studies of plasticity whenever possible due to their ease of measurement and ability to capture information about phenotypic-environmental interactions at different scales. Several vertebral measures, but most notably the anterior-posterior (AP) and transverse (TR) vertebral neural canal diameters, have been successfully used to assess relationships between episodes of developmental stress and measures of growth, morbidity, and mortality in archaeological contexts (Amoroso and Garcia 2018; Newman and Gowland 2015; Watts 2013, 2015). In modern humans, the AP diameter of the vertebral neural canal likely experiences limited growth beyond five years of age, and Newman and Gowland (2015) have hypothesized that most of this growth occurs in early life. While the TR diameter similarly completes most of its growth in the first five years of life, it may achieve limited gains in size between early childhood and late adolescence (Newman and Gowland 2015; Watts 2013). Thus, AP and TR diameters of the vertebral neural canal may embody stress experienced in two overlapping periods of development, and differences in patterns of associations between constrained growth in these measures and phenotypic outcomes of interest may yield valuable information to investigators (Watts 2015).

Watts' (2015) nuanced analysis of developmental stress in late Medieval and post-Medieval London samples (n=462, 480), utilized enamel defects in conjunction with AP and TR VNC measures to estimate the timing of stress events and their association with trade-offs involving growth and survival. The results of her study found that episodes of constrained early growth, indicated by the presence of linear enamel hypoplasia (LEH) and constrained AP VNC diameters, were not strongly associated with estimated age at death in either sample. Equally intriguing was the finding that constrained early growth was not associated with constrained later growth in childhood and adolescence, represented by TR VNC diameters. However, measures of constrained growth in these later life history phases were significantly associated with estimated age at death, suggesting that trade-offs between growth and longevity initiated in childhood and adolescence may drive patterns of mortality in some populations (Watts 2015).

Although the models utilized in these studies may not be applicable to all research contexts, use of dental and skeletal biomarkers to assess the timing and frequency of stress events may enrich traditional approaches to studying the phenotypic effects of developmental stress in archaeological and paleoanthropological contexts. They may also provide a potential starting point for assessing the potential costs and benefits of developmental plasticity and its involvement in evolutionary processes.

THE PROBLEM OF EQUIFINALITY IN STUDIES OF DEVELOPMENTAL PLASTICITY

Existing approaches to studying developmental plasticity in anthropology and public health often use estimated height and body mass as proxy measures of plastic responses to developmental stress (Antón et al. 2016; Pant-

siotou et al. 2008; Svefors et al. 2019). Estimated stature and body mass are highly useful measures in archaeological and paleoanthropological studies, as they may be used to assess life history characteristics (Helmut 2014) and patterns of growth (Svefors et al. 2019; Wells 2016). Furthermore, body mass can be estimated using a variety of dental, cranial, and post-cranial measures, while stature estimates typically utilize dimensions of long bone diaphyses that preserve well in archaeological and paleoanthropological contexts. These measures reflect complex interactions between genetic and extragenetic systems of inheritance, and without additional contextual information, it is not always possible to assess the contribution of plastic developmental processes to phenotypic variation.

Referring to the previous section, long bone dimensions (e.g., diaphyseal length) in particular are shaped by extended episodes of phenotypic-environmental interactions occurring across life history phases, with the potential for catch-up growth and exercise-induced increases in bone density in childhood and adolescence (Haapasalo et al. 2000, 2009; Svefors et al. 2019). Ultimately, in the absence of contextual data, variation in measures that embody long-term developmental trajectories may indicate the presence of plasticity but provide only limited information about the phenotypic-environmental interactions that informed development. In the further absence of information about the timing of phenotypic-environmental interactions, specifically, it may not be possible to identify periods of heightened susceptibility to environmental influence or to evaluate costs of plasticity related to life history trade-offs (McPherson 2021; Temple 2019). This is not a problem if the primary goal of a study is to identify the presence of plasticity in response to a stressor but represents a significant obstacle to interpretation in studies intended to evaluate aspects of the DOHaD hypothesis or the adaptive benefits of plasticity.

Accounting for the timing and frequency of stressors at different stages in development refines processes of hypothesis testing by facilitating detection of more subtle patterns of trade-offs between growth and longevity in archaeological samples. Evidence of inhibited diaphyseal growth in the presence of acute stress biomarkers occurring over an extended developmental timeline may be indicative of a developmental environment characterized by constraint. In this context, constrained or stunted growth may indicate that plastic processes were insufficient to overcome chronic energetic deprivation. However, evidence of inhibited growth in early life alongside indicators of accelerated maturation and elevated mortality risk in adulthood may be more suggestive of strategic adjustments to the pace and tempo of life history (Del Giudice et al. 2011). "Normal" growth occurring alongside evidence of significant early life stress may suggest the presence of trade-offs that enhanced resilience, but this pattern may coincide with elevated mortality risk in adulthood (Ellis et al. 2017; Temple 2019). Alternatively, if there is limited evidence of stress in late childhood and early adolescence, this pattern could also indicate the presence of compensatory plasticity facilitating catch-up growth.

As these hypothetical examples illustrate, there are multiple pathways to each phenotypic outcome (constrained vs. normal growth), and these involve different sets of plastic responses and associated life history trade-offs. Constrained growth is associated with a variety of negative measures of health and wellbeing (Chua et al. 2017; Danese and McEwen 2012; Kuzawa and Quinn 2009; Watts 2013), but catch-up growth is not cost-free and may also modify individual susceptibility and mortality risk across the lifespan (Martin et al. 2017). Studies of the costs of catch-up growth are limited, and more evidence is needed to define its relationship to measures of adult health. The costs of catch-up growth may depend on when it occurs in the developmental life cycle, and several studies of catch-up growth in early life suggest that it may not produce significant metabolic consequences in adulthood if it occurs prior to two years of age (Beltrand et al. 2009; Visuthranukul et al. 2019). Others suggest that individuals who overcome constrained growth in early life through compensatory plastic processes may face costs in adulthood in the form of increased susceptibility to cardiovascular disease and metabolic disorders (Nobili et al. 2008; Martin et al. 2017). Populations with access to sufficient resources may not have to make significant trade-offs between the pace of growth and pace of maturation. In contemporary Western populations, there is an increasing trend toward accelerated patterns of skeletal growth and maturation with no associated losses in linear growth (Boeyer et al. 2018; Thompson et al. 2020). Yet even though greater height is often associated with measures of fitness (e.g., longer lifespans, reduced mortality risks), individuals who experience accelerated growth trajectories may still face downstream costs in the form of elevated mortality risks in adulthood (Thompson et al. 2020).

In studies of plasticity, growth trajectories should be given the same consideration as their end products. Costs associated with accelerated or inhibited growth—and when they apply—may largely determine whether plasticity is adaptive within a particular environmental context. These considerations are especially crucial in studies involving skeletal and fossil samples, as apparently maladaptive artifacts of plasticity in the skeletal system may signal the presence of underlying plastic processes that enhance resilience and promote survival, while measures of health may disguise costly trade-offs (McFadden and Oxenham 2020; Temple 2019; Temple and Edes 2022).

DISCUSSION

Studying developmental plasticity in the skeletal and fossil record is methodologically challenging, as hard tissue is limited in its ability to capture evidence of phenotypic-environmental interactions and there are significant gaps in the hominin fossil record. This situation complicates efforts to study plasticity in the deep past, as incomplete preservation may impede our ability to apply relevant techniques to fossil material or to gather sample sizes large or cohesive enough to permit comparative studies within

or across populations. At present, there are few comparative studies of developmental plasticity in the anthropological literature, and even fewer in paleoanthropology despite increased interest in the role played by developmental plasticity in hominin evolution (Antón and Kuzawa 2017; Antón et al. 2016; McKerracher et al. 2019).

However, a recent study by Antón and colleagues (2016) that employed body size and degree of sexual dimorphism as proxy measures of plastic stress suggests that modern humans and species of widely dispersed non-human primates exhibit greater plasticity in somatic growth than extinct species of hominins. This study included 35 human and non-human primate (*Macaca* and *Chlorocebus*) populations from known environmental contexts, and 14 time and space restricted paleodemes of fossil *Homo*, to include *H. erectus* and Neanderthals. Antón and colleagues (2016) found that the human and non-human primate populations exhibited the greatest similarity in pattern, while both Neanderthals and *H. erectus* exhibited reduced plasticity in comparison to modern humans. However, Neanderthals exhibited less plasticity than *H. erectus.* Although small sample sizes significantly limited their examination of plastic variation in extinct hominins, they suggest that there are indications of "unusual" variability in *H. erectus* worth exploring in further analyses when additional fossil evidence becomes available (Antón et al. 2016: 15).

The results of Antón and colleagues' (2016) initial analysis are intriguing, raising lines of further inquiry and inviting additional comparative studies. The high degree of plasticity observed in modern human populations may be partially attributable to the buffering effect of social and technoeconomic systems, but this does not explain evidence of enhanced developmental plasticity in extant non-human primates relative to extinct hominins. Studies of non-human primate species suggest that we have likely underestimated the behavioral and physiological plasticity of our living relatives, especially in widely dispersed lineages occupying more variable ecological niches (Snyder-Mackler et al. 2020). It could be the case that the components required to evolve enhanced developmental plasticity were present in the primate lineage prior to the emergence of *Homo*, and recent environmental histories rather than phylogeny may be better predictors of plastic capabilities. The enhanced plasticity of modern humans may have rapidly accumulated in response to environmental heterogeneity experienced through repeated episodes of dispersal and occupation of extremely diverse ecological niches (Grove 2014). Similarly, differences in the relative plasticity of Neanderthals and *H. erectus* may also reflect differences in the rate and geographic extent of their dispersal.

Still, it remains unclear whether plasticity in growth and maturation is adaptive or a product of inhibited development (Campos et al. 2021; Lea et al. 2015, 2018; Snyder-Mackler et al. 2020). Studies of developmental plasticity in more diverse social, ecological, and temporal contexts may provide us with essential information about the environmental factors that promote or constrain plasticity. Tests of the DOHaD hypothesis in skeletal and fossil samples may further indicate how phenotypic-environmental interactions experienced at different phases in the lifecycle contribute to frailty or resilience (Agarwal 2016; Antón et al. 2016; McFadden and Oxenham 2020). Given the evolutionary history of our genus, and the presence of enhanced plastic capabilities in widely dispersed extant primates, it may indeed be the case that developmental plasticity is most beneficial in environments characterized by novelty and adversity. If so, developmental plasticity research focusing on wealthy, contemporary Western populations may be best positioned to assess costs associated with predictive limitations and environmental "mismatches," rather than plasticity's potential benefits. Exploring developmental plasticity in deep time may help us better understand how plastic processes shape morbidity and mortality trends observed in living populations, the phenotypic-environmental interactions that shaped the history of our lineage, and the selective environments in which our plastic capacities evolved.

ACKNOWLEDGEMENTS

Work completed in support of this manuscript was made possible by the National Science Foundation, Directorate for Social and Behavioral Sciences, Division of Behavioral and Cognitive Sciences and the University of Arizona School of Anthropology. The author would like to thank Drs. Ivy Pike, Mary Stiner, and James Watson for their insightful comments on early versions of this manuscript and their continued encouragement throughout the writing process. The author is also grateful for the thoughtful feedback provided by the guest editors of this issue, Chris Kuzawa, and three anonymous reviewers, whose suggestions significantly improved the final version of this paper.

REFERENCES

Agarwal, S.C., 2016. Bone morphologies and histories, life course approaches in bioarchaeology. Yearb. Phys. Anthropol. 159, 130–149.

Agrawal, A., 2001. Phenotypic plasticity in the interactions and evolution of species. Science 294, 321–326.

Aiello, L.C., Antón, S.C., 2012. Human biology and the origins of *Homo*. Curr. Anthropol. 53, S269–S277.

Aldermann, H., Behrman, J.R., Glewwe, P., Fernald, L., Walker, S., Bundy, D.A.P., de Silva, N., Horton, S., Jamison, D.T., Patton, G.C., 2017. Evidence of impact of interventions on growth and development during early and middle childhood. In: Aldermann, H., Behrman, J.R., Glewwe, P., Fernald, L., Walker, S., Bundy, D.A.P., de Silva, N., Horton, S., Jamison, D.T., Patton, G.C. (Eds.), Child and Adolescent Health and Development. The International Bank for Reconstruction and Development, The World Bank, Washington, D.C. https://doi.org/10.1596/978-1-4648-0423-6_ch7

Alpert, P., Simms, E., 2002. The relative advantages of plas-

ticity and fixity in different environments: When is it good for a plant to adjust? Evol. Ecol. 16, 285–297.
Amoroso, A., Garcia, S.J., 2018. Can early-life growth disruptions predict longevity? Testing the association between vertebral neural canal (VNC) size and age-at-death. Int. J. Paleopathol. 22, 8–17.
Antón, S.C., 2007. Climatic influences on the evolution of early *Homo*? Folia. Primatol. 78, 365–388.
Antón, S.C., 2012. Early *Homo*, who, when and where? Curr. Anthropol. 53, S278–S298.
Antón, S.C., Kuzawa, C.W., 2017. Early *Homo*, plasticity and the extended evolutionary synthesis. Interface Focus *7*, 20170004–20170004. https://doi.org/10.1098/rsfs.2017.0004
Antón, S.C., Leigh, S.R., 2003. Growth and life history in *Homo erectus*. In: Thompson, J.L., Krovitz, G.E., Nelson, A.J. (Eds.), Patterns of Growth and Development in the Genus *Homo*. Cambridge University Press, Cambridge, pp. 219–245.
Antón, S.C., Snodgrass, J.J., 2012. Origins and evolution of the genus *Homo*. Curr. Anthropol. 53, 5479–5496.
Antón, S.C., Taboada, H.G., Middleton, E.R., Rainwater, C.W., Taylor, A.B., Turner, T.R., Turnquist, J.E., Weinstein, K.J., Williams, S.A., 2016. Morphological variation in *Homo erectus* and the origins of developmental plasticity. Philos. Trans. R. Soc. B 371, 1–18.
Armelagos, G.J., Goodman, A.H., Harper, K.N., Blakey, M.L., 2009. Enamel hypoplasia and early mortality: bioarcheological support for the Barker hypothesis. Evol. Anthropol. 18, 261–271.
Avery, L.C., Prowse, T.L., Findlay, S., Brickley, M.B., 2022. Bioarchaeological approaches to the study of adolescence. Child. Past. 15, 3–14.
Barker, D.J.P., 1995. Intrauterine programming of adult disease. Trends Mol. Med. 1, 418–423.
Beltrand, J., Nicolescu, R., Kaguelidou, F., Verkauskiene, R., Sibony, O., Chevenne, D., Claris, O., Lévy-Marchal, C., 2009. Catch-up growth following fetal growth restriction promotes rapid restoration of fat mass but without metabolic consequences at one year of age. PLOS One 4, e5343.
Bermúdez de Castro, J.M., Ramírez Rozzi, F., Martinón-Torres, M., Sarmiento Pérez, S., Rosas, A., 2003. Patterns of dental development in Lower and Middle Pleistocene hominins from Atapuerca, (Spain). In: Thompson, J. L., Krovitz, G. E., Nelson, A.J. (Eds.), Patterns of Growth and Development in the Genus *Homo*. Cambridge University Press, Cambridge, pp. 246–270.
Bhutta, Z.A., Das, J.K., Rizvi, A., Gaffey, M.F., Walker, N., Horton, S., Webb, P., Lartey, A., Black, R.E., 2013. Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? The Lancet 382, 452–477.
Biehler-Gomez, L. Mattia, M., Mondellini, M., Palazzolo, L., Cattaneo, C., 2022. Differential skeletal preservation between sexes: a diachronic study in Milan over 2000 years. Archaeol. Anthropol. Sci. 14, 147. https://doi.org/10.1007/s12520-022-01616-0
Boeyer, M.E., Sherwood, R.J., Deroche, C.B., Duren, D.L., 2020. Early maturity as the new normal: a century-long study of bone age. Clin. Orthop. Relat. Res. 476, 2122.
Bogin, B., 1997. Evolutionary hypotheses for human childhood. Am. J. Phys. Anthropol. 104, 63–89.
Bogin, B., 1999. Evolutionary perspective on human growth. Annu. Rev. Anthropol. 28, 109–53.
Bogin, B., 2003. The human pattern of growth and development in paleontological perspective. In: Thompson, J.L., Krovitz, G.E., Nelson, A.J. (Eds.), Patterns of Growth and Development in the Genus *Homo*. Cambridge University Press, Cambridge, pp. 15–44.
Bogin, B., 2006. The evolution of human childhood and fertility. In: Hawkes, K., Paine, R.R., (Eds.), The Evolution of Human Life History. School of American Research Press, Santa Fe, pp. 197–230.
Bogin, B., 2020. Evolution of the human life cycle. In: Thompson, J.L., Krovitz, G.E., Nelson, A.J., (Eds.), Patterns of Human Growth. Cambridge University Press, Cambridge, pp. 187–272.
Bogin, B., Varea, C., Hermanussen, M., Scheffler, C., 2017. Human life course biology, a centennial perspective of scholarship on the human pattern of physical growth and its place in human biocultural evolution. Am. J. Phys. Anthropol. 165, 834–854.
Botero, C.A., Weissing, F.J., Wright, J., Rubenstein, D.R., 2015. Evolutionary tipping points in the capacity to adapt to environmental change. Proc. Nat. Acad. Sci. U.S.A. 112, 184–189.
Bradshaw, W., Holzapfel, C., 2008. Genetic response to rapid climate change: it's seasonal timing that matters. Mol. Ecol. 17, 157–166.
Brumbach, B.H., Figueredo, A.J., Ellis, B.J., 2009. Effects of harsh and unpredictable environments in adolescence on the development of life history strategies. Hum. Nat. 20, 25–51.
Burggren, W.W., Mueller, C.A., 2019. Developmental critical windows and sensitive periods as three-dimensional constructs in time and space. Physiol. Biochem. Zool. 88, 91102.
Campos, F.A., Archie, E.A., Gesquiere, L.R., Tung, J., Altmann, J., Alberts, S.C., 2021. Glucocorticoid exposure predicts survival in female baboons. Sci. Adv. 7, eabf6759.
Cavallo, F., Mohn, A., Chiarelli, F., Giannini, C., 2021. Evaluation of bone age in children: a mini-review. Front. Pediatr. 9. https://doi.org/10.3389/fped.2021.580314
Champagne, F.A., 2013. Epigenetics and developmental plasticity across species. Dev. Psychob. 55, 33–41.
Chovalopoulou, M., Papageorgopoulou, C., Bertsatos, A., 2017. Cranium asymmetry in a modern Greek population sample of known age and sex. Int. J. Leg. Med. 131, 803–812.
Christian, P., Smith, E.R., 2018. Adolescent undernutrition: global burden, physiology, and nutritional risks. Ann. Nutr. Metab. 72, 316–328.
Chua, K.J., Lukaszewski, A.W., Grant, D.M., Song, O., 2017. Human life history strategies, calibrated to ex-

ternal or internal cues? Evol. Psychol. 15. https://doi.org/10.1177/1474704916677342

Clark, G.A., Hall, N.R., Armelagos, G.J., Borkan, G.A., Panjabi, M.M., Wetzel, F.T., 1986. Poor growth prior to early childhood: decreased health and lifespan in the adult. Am. J. Phys. Anthropol. 70, 145–160.

Crespi, E.J., Denver, R.J., 2005. Ancient origins of human developmental plasticity. Am. J. Hum. Biol. 17, 44–54.

Cunningham, C., Scheuer, L., Black, S., 2017. Developmental Juvenile Osteology. Elsevier Science, St. Louis.

Danese, A., McEwen, B.S., 2012. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. Physiol. Behav. 106, 29–39.

David, L.A., Maurice, C.F., Carmody, R.N., Gootenberg, D.B., Button, J.E., Wolfe, B.E., Ling, A.V., Devllin, S., Varma, L., Fischbach, M.A., Biddinger, S.B., Dutton, R.J., Turnbaugh, P.J., 2013. Diet rapidly and reproducibly alters the human gut microbiome. Nature 505, 559–563.

Davis, K.A., Mountain, R.V., Pickett, O.R., Den Besten, P.K., Bidlack, F.B., Dunn, E.C., 2019. Teeth as potential new tools to measure early-life adversity and subsequent mental health risk, an interdisciplinary review and conceptual model. Biol. Psychiat. 87, P502–513.

Dean, M.C., 2006. Tooth microstructure tracks the pace of human life-history evolution. Philos. Trans. R. Soc. B 273, 2799–2808.

Dean, M.C., Leakey, M.G., Reid, D.J., Schrenk, F., Schwartz, G.T., Stringer, C., Walker, A., 2001. Growth processes in teeth distinguish modern humans from *Homo erectus* and earlier hominins. Nature 414, 628—631.

Dean, M.C., Smith, H. B., 2009. Growth and development of the Nariokotome youth, KNMWT 15000. In: Grine, F.E., Fleagle, J.G., Leakey, R.E. (Eds.), The First Humans: Origin of the Genus *Homo*. Springer, New York, pp. 101–120.

Decrausaz, S., Cameron, M.E., 2022. A growth area: a review of the value of clinical studies of child growth for palaeopathology. *Evol. Med. Public Health*. 10, 108–122.

De Filippo, C., Cavalieri, D., Di Paola, M., Ramazzotti, M., Poullet, J.B., Massart, S., Collini, S., Pieraccini, G., Lionetti, P., 2010. Impact of diet in shaping gut microbiota revealed by a comparative study in children from Europe and rural Africa, Proc. Nat. Acad. Sci. U.S.A. 107, 14691–14696.

Del Giudice, M., 2015. Attachment in middle childhood: an evolutionary–developmental perspective. In: G. Bosmans, K. A. Kerns (Eds.), Attachment in Middle Childhood: Theoretical Advances and New Directions in an Emerging Field. New Dir. Child and Adolesc. Dev. 148, 15–30.

Del Giudice, M., Ellis, B.J., Shirtcliff, E.A., 2011. The adaptive calibration model of stress responsivity. Neurosci. Biobehav. Rev. 35, 1562–1592.

Depauw, E., Oxley, D., 2018. Toddlers, teenagers, and terminal heights: the importance of puberty for adult male stature, Flanders, 1800–16. Econ. Hist. Rev. 72, 925–952.

Diméglio, A., Canavese, F., 2012. The growing spine: how spinal deformities influence normal spine and thoracic cage growth. Eur. Spine. J. 21, 64–70.

Donatti, L.T., Koch, V.H.K., Takayama, L., Pereira, R.M.R., 2011. Effects of glucocorticoids on growth and bone mineralization. J. Pediat. 87, 4–12.

Doughty, P., Reznick, D.N., 2004. Patterns and analysis of adaptive phenotypic plasticity in animals. In: DeWitt, T.J., Scheiner, S.M. (Eds.), Phenotypic Plasticity, Functional and Conceptual Approaches. Oxford University Press, New York, pp. 126–150.

Eli, I., Sarnat, H., Talmi, E., 1989. Effect of the birth process on the neonatal line in primary tooth enamel. J. Clin. Pediatr. Dent. 11, 220–223.

Ellis, B.J., 2013. The hypothalamic–pituitary–gonadal axis: a switch-controlled, condition sensitive system in the regulation of life history strategies. Horm. Behav. 64, 215–225.

Ellis, B.J., Bianchi, J., Griskevicius, V., Frankenhuis, W.E., 2017. Beyond risk and protective factors, an adaptation-based approach to resilience. Perspect. Psychol. Sci. 12, 561–587.

Ellis, B.J., Del Giudice, M., 2019. Developmental adaptation to stress, an evolutionary perspective. Ann. Rev. Psychol. 70, 111–139.

Ellis, B.J., Figueredo, A.J., Brumbach, B.H., Schlomer, G.L., 2009. Fundamental dimensions of environmental risk: the impact of harsh versus unpredictable environments on the evolution and development of life history strategies. Hum. Nature 20, 204–268.

Fawcett, T.W., Frankenhuis W.E., 2015. Adaptive explanations for sensitive windows in development. Front. Zool. 12. https://doi.org/10.1186/1742-9994-12-S1-S3

Forman, M.R., Mangini, L.D., Thelus-Jean, R., Hayward, M.D., 2013. Life-course origins of the ages at menarche and menopause. Adolescent Health. Med. Ther. 4, 1–21.

Frederick, M.J., Gallup, G.G., 2007. Fluctuating dental asymmetry in great apes, fossil hominins, and other humans: implications for changing stressors during human evolution. Acta Psychol. Sin. 39, 489–494.

Fuhrmann, D., Knoll, L.J., Blakemore, S., 2015. Adolescence as a sensitive period of brain development. Trends Cog. Sci. 19, 558–566.

Garland, C.J., 2020. Implications of accumulative stress burdens during critical periods of early postnatal life for mortality risk among Guale interred in a colonial era cemetery in Spanish Florida (ca. AD 1605–1680). Am. J. Phys. Anthropol. 172, 621–637.

Gawlik, A., Hochberg, Z., 2012. Lessons from the life history of natural fertility societies on child growth and maturation. Swiss Med. Wkly. 142, w13600.

Gettler, L.T., McDade, T.W., Bragg, J.M., Feranil, A.B., Kuzawa, C.W., 2015. Developmental energetics, sibling death, and parental instability as predictors of maturational tempo and life history scheduling in males from Cebu, Philippines. Am. J. Phys. Anthropol. 158, 175–184.

Gettler, L.T., Rosenbaum, S., Kuo, P.X., Bechayda, S.A., McDade, T.W., 2022. Evidence for an adolescent sensitive

period to family experiences influencing adult male testosterone production. Proc. Nat. Acad. Sci. U.S.A. 119, e2202874119.

Gluckman, P.D., Hanson, M.A., 2006. The developmental origins of health and disease: the breadth and importance of the concept. In: Wintour, E.M., Owens, J.A. (Eds.), Early Life Origins of Health and Disease. Springer Science and Business Media, New York, pp. 1–7.

Gluckman, P., Hanson, M., Buklijas, T., Low, F.M., Beedle, A.S., 2009. Epigenetic mechanisms that underpin metabolic and cardiovascular diseases. Nat. Rev. Endocrinol. 5, 401–408.

Gluckman, P., Hanson, M., Buklijas, T., 2010. A conceptual framework for the developmental origins of health and disease. *J. Dev. Orig. Health Dis. 1*, 6–18.

Goodman, A.H., Martin, D.L., Armelagos, G.J., 1984. Indications of stress from bone and teeth. In: Cohen, M.N., Armelagos, G.J. (Eds.), Paleopathology at the Origins of Agriculture. Academic Press, Cambridge, pp. 13–44.

Goodman, A.H., Thomas, R.B., Swedlund, A.C., Armelagos, G.J., 1988. Biocultural perspectives on stress in prehistoric, historical, and contemporary population research. Am. J. Phys. Anthropol. 31, 169–202.

Gowland, R.L., 2015. Entangled lives, implications of the developmental origins of health and disease hypothesis for bioarchaeology and the life course. Am. J. Phys. Anthropol. 158, 530–540.

Grove, M., 2011. Change and variability in Plio-Pleistocene climates: modelling the hominin response. J. Archaeol. Sci. 38, 3038e3047.

Grove, M., 2014. Evolution and dispersal under climatic instability: a simple evolutionary algorithm. Adapt. Behav. 22, 235e254.

Grove, M., 2015. Palaeoclimates, plasticity, and the early dispersal of *Homo sapiens*. Quatern. Int. 369, 17–37.

Grove, M., Lamb, H., Roberts, H., Davies, S., Marshall, M., Bates, R., Huws, D., 2015. Climatic variability, plasticity, and dispersal, a case study from Lake Tana, Ethiopia. J. Hum. Evol. 87, 32–47.

Guatelli-Steinberg, D., Reid, D.J., 2008. What molars contribute to an emerging understanding of lateral enamel formation in Neandertals vs. modern humans. J. Hum. Evol. 54, 236–250.

Haapasalo, H., Kontulainen, S., Sievänen, S., Kannus, P., Järvinen, M., Vuori, I., 2000. Exercise-induced bone gain is due to enlargement in bone size without a change in volumetric bone density, a peripheral quantitative computed tomography study of the upper arms of male tennis players. Bone 27, 351–357.

Haapasalo, H., Kannus, P., Sievänen, H., Pasanen, M., Uusi-rasi, K., Heinonen, A., Oja, P., Vuori, I., 2009. Effect of long-term unilateral activity on bone mineral density of female junior tennis players. J. Bone. Miner. Res. 13, 310–319.

Hartman, S., Belsky, J., 2018. Prenatal programming of postnatal plasticity revisited—and extended. Dev. Psychopathol. 30, 825–842.

Hedges, R.E.M., Clement, J.G., Thomas, C.D.L., O'Connell, T., 2007. Collagen turnover in the adult femoral midshaft: modeled from anthropogenic radiocarbon tracer measurements. Am. J. Phys. Anthropol. 133, 808–816.

Hemmer, H., 2014. Estimation of basic life history data of fossil hominoids. In: Winfried, H., Tattersall, I (Eds.), Handbook of Paleoanthropology. Springer, Heidelberg, pp. 703–743.

Henrich, J., Heine, S.J., Norenzayan, A., 2010. Most people are not WEIRD. Nature 466, 7302.

Hereford, J., 2009. A quantitative survey of local adaptation and fitness trade-offs. Am. Nat. 173, 579–588.

Hillson, S.W., 1992. Dental enamel growth, perikymata and hypoplasia in ancient tooth crowns. J. R. Soc. Med. 85, 460–466.

Hochberg, Z., 2008. Juvenility in the context of life history theory. Arch. Dis. Child. 93, 534–539.

Hochberg, Z., 2012. Evo-Devo of Child Growth, Treatise on Child Growth and Human Evolution. Wiley-Blackwell, New York.

Kuzawa, C.W., 2005. Fetal origins of developmental plasticity, are fetal cues reliable predictors of future nutritional environments? Am. J. Hum. Biol. 17, 5–21.

Kuzawa, C.W., 2007. Developmental origins of life history, growth, productivity, and reproduction. Am. J. Hum. Biol. 19, 654–661.

Kuzawa, C.W., Quinn, E., 2009. Developmental origins of adult function and health, evolutionary hypotheses. Ann. Rev. Anthropol. 38, 131–47.

Kuzawa, C.W., Thayer, Z.M., 2011. Timescales of human adaptation, the role of epigenetic processes. Epigenomics 3, 221–234.

Kuzawa, C.W., 2013. You are what your mother ate? Am. J. Clin. Nut. 97, 1157–1158.

Kuzawa, C.W., Bragg, J.M., 2012. Plasticity in human life history strategy, implications for contemporary human variation and the evolution of genus *Homo*. Curr. Anthropol. 53(S6), 5369–5382.

Kuzawa, C.W., Chugani, H.T., Grossman, L.I., Lipovich, L., Muzik, O., Hof, P.R., Wildman, D.E., Sherwood, C.C., Leonard, W.R., Lange, N., 2014. Metabolic costs and evolutionary implications of human brain development. Proc. Nat. Acad. Sci. U.S.A. 111, 13010–13015.

Lampl, M., Schoen, M., 2017. How long bones grow children, mechanistic paths to variation in human height growth. Am. J. Hum. Biol. 29, e22983.

Lea, A.J., Altmann, J., Alberts, S.C., Tung, J., 2015. Developmental constraints in a wild primate. Am. Nat. 185, 809–821.

Lea, A.J., Tung, J., Archie, E.A., Alberts, S.C., 2018. Developmental plasticity research in evolution and human health. Evol. Med. Public Health 2017, 201–205. https://doi.org/10.1093/emph/eoy007

Leigh, S.R., 2004. Brain growth, life history and cognition in primate and human evolution. Am. J. Primatol. 62, 139–164.

Leonard, W.R., Robertson, M.L., 1994. Evolutionary perspectives on human nutrition, the influence of brain

and body size on diet and metabolism. Am. J. Hum. Biol. 6, 77–88.

Levy, S.B., Klimova, T.M., Zakharova, R.N., Fedorov, A.I., Fedorova, V.I., Baltakhinova, M.E., 2021. Evidence for a sensitive period of plasticity in brown adipose tissue during early childhood among indigenous Siberians. Am. J. Phys. Anthropol. 175, 1–13.

Lewis, M.E., 2017. Paleopathology of Children, Identification of Pathological Conditions in the Human Skeletal Remains of Children. Academic Press, London.

Lewis, M.E., 2022. Exploring adolescence as a key life history stage in bioarchaeology. Am. J. Bio. Anthropol. 179, 1–16. https://doi.org/10.1002/ajpa.24615

Liu, X., Somel, M., Tang, L., Yan, Z., Jiang, X., Guo, S., Yuan, Y., He, L., Oleksiak, A., Zhang, Y., Li, N., Hu, Y., Chen, W., Qiu, Z., Pääbo, S., Khaitovich, P., 2012. Extension of cortical synaptic development distinguishes humans from chimpanzees and macaques. Genome Res. 22, 611–622.

Livshits, G., Kobyliansky, E., 1991. Fluctuating asymmetry as a possible measure of developmental homeostasis in humans: a review. Hum. Bio. 63, 441–466.

Llop-Viñolas, D., Vizmanos, B., Monosterolo, R.C., Subías, J.E., Fernández-Ballart, J.D., Martí-Henneberg, C., 2004. Onset of puberty at eight years of age in girls determines a specific tempo of puberty but does not affect adult height. Acta Paediatr. 93, 874–879.

Lorentz, K.O., Lemmers, S.A.M., Chrysostomou, C., Dirks, W., Zaruri, M.R., Foruzanfar, F., Sajjadi, S.M.S., 2019. Use of dental microstructure to investigate the role of prenatal and early life physiological stress in age at death. J. Archaeol. Sci. 104, 85–96.

Low, F.M., Gluckman, P.D., Hanson, M.A., 2012. Developmental plasticity, epigenetics and human health. Evol. Biol. 39, 650–665.

Lu, A., Petrullo, L., Carrera, S., Feder, J., Schneider-Crease, I., Snyder-Mackler, N., 2019. Developmental responses to early-life adversity, evolutionary and mechanistic perspectives. Evol. Anthropol. 28, 249–266.

MacKelvie, K.J., Khan, K.M., McKay, H.A., 2002. Is there a critical period for bone response to weight-bearing exercise in children and adolescents? A systematic review. Br. J. Sports. Med. 36, 250–257.

Macchiarelli, R., Bondioli, L., Debenath, A., Mazurier, A., Tournepiche, J., Birch, W., Dean, C., 2006. How Neanderthal teeth grew. Nature 444, 748–751.

Marshall, W.A., Tanner, J.M., 1986. Puberty. In: Falkner, F., Tanner, J.M. (Eds.), Postnatal Growth Neurobiology. Springer, Boston, pp. 171–209.

Martin, A., Connelly, A., Bland, R., Reilly, J., 2017. Health impact of catch-up growth in low-birth weight infants, systematic review, evidence appraisal, and meta-analysis. Matern. Child. Nutr. 13, e12297.

Mazziotti, G., Giustina, A., 2013. Glucocorticoids and the regulation of growth hormone secretion. Nat. Rev. Endocrinol. 9, 265–276.

McEwen, B.S., 2008. Understanding the potency of stressful early life experiences on brain and body function. Metabolism 57, S11-S15.

McDermott, C.L., Hilton, K., Park, A.T., Tooley, U.A., Boroshok, A.L., Mupparapu, M., Scott, J.M., Bumann, E.E., Mackey, A.P., 2021. Early life stress is associated with earlier emergence of permanent molars. Proc. Nat. Acad. Sci. U.S.A. 118, e2105304118.

McFadden, C., Oxenham, M.F., 2020. A paleoepidemiological approach to the osteological paradox: investigating stress, frailty and resilience through cribra orbitalia. Am. J. Phys. Anthropol. 173, 205–217.

McKerracher, L., Fried, R., Kim, A. W., Moffat, T., Sloboda, D.M., Galloway, T., 2020. Synergies between the Developmental Origins of Health and Disease framework and multiple branches of evolutionary anthropology. Evol. Anthropol. 29, 214–219.

McPherson, C.B., 2021. Examining developmental plasticity in the skeletal system through a sensitive developmental windows framework. Am. J. Phys. Anthropol. 176, 163–178.

Measelle, J.R., Ablow, J.C., 2018. Contributions of early adversity to pro-inflammatory phenotype in infancy: the buffer provided by attachment security. Attach. Hum. Dev. 20, 1–23.

Migliano, A.B., Guillon, M. 2012. The effects of mortality, subsistence, and ecology on human adult height and implications for *Homo* evolution. Curr. Anthropol. 53, S359–S368. https://doi.org/10.1086/667694

Moes, E., Willermet, C., Hunley, K., Ragsdale, C., Edgar, H.J.H., 2022. Childhood stress and developmental instability: comparing microscopic enamel defects and cranial fluctuating asymmetry in a colonial Mexican sample. Am. J. Bio. Anthropol. 179, 134–147.

Moller, A.P., 1999. Asymmetry as a predictor of growth, fecundity, and survival. Ecol. Letters 2, 149–156.

Murray, J.K., Benitez, R.A., O'Brien, M.J. 2020. The extended evolutionary synthesis and human origins: archaeological perspectives. Evol. Anthropol. 30, 4–7.

Murren, C.J., Auld, J.R., Callahan, H., Ghalambor, C.K., Handelsman, C.A., Heskel, M.A., Kingsolver, J.G., Maclean, H.J., Masel, J., Maughan, H., Pfennig, D.W., Relyea, R.A., Seiter, S., Snell-Rood, E., Steiner, U.K., Schlichting, C.D., 2015. Constraints on the evolution of phenotypic plasticity, limits and costs of phenotype and plasticity. Heredity 115, 293–301.

Nava, A., Lugli, F., Romandini, M., Badino, F., Evans, D., Helbing, A.H., Oxilia, G., Arrighi, S., Bortolini, E., Delpiano, D., Duches, R., Figus, C., Livraghi, A., Marciani, G., Silvestrini, S., Cipriani, A., Giovanardi, T., Pini, R., Tuniz, C., Bernardini, F., Dori, I., Coppa, A., Cristiani, E., Dean, C., Bondioli, L., Peresani, M., Muller, W., Banazzi, S., 2020. Early life of Neanderthals. Proc. Nat. Acad. Sci. U.S.A. 117, 28719–28726.

Nettle, D., Bateson, M., 2015. Adaptive developmental plasticity: what is it, how can we recognize it and when can it evolve? Proc. R. Soc. B 282, 20151005.

Newman, S.L., Gowland, R.L., 2015. Brief communication, the use of non-adult vertebral dimensions as indicators of growth disruption and non-specific indicators of

health stress in skeletal populations. Am. J. Phys. Anthropol. 158, 155–164.
Nettle, D., Bateson, M., 2015. Adaptive developmental plasticity, what is it, how can we recognize it and when can it evolve? Philos. Trans. R. Soc. B 282, 20151005.
Nettle, D., Frankenhuis, W.E., Rickard, I.J., 2013. The evolution of predictive adaptive responses in human life history. Philos. Trans. R. Soc. B 280, e20131343.
Neubauer, S., Hublin, J., 2012. The evolution of human brain development. Evol. Biol. 39, 568–586.
Nobili V., Alisi A., Panera N., Agostoni C., 2008. Low birth weight and catch-up-growth associated with metabolic syndrome, a ten year systematic review. Pediatr. Endocrinol. Rev. 6, 241–247.
Nowell, A., 2016. Childhood, play, and the evolution of cultural capacity in Neanderthals and modern humans. In: Haidle, M.N., Conrad, N.J., Bolus, M. (Eds.), The Nature of Culture, based on an Interdisciplinary Symposium 'The Nature of Culture,' Tübingen, Germany. Yale University Press, New Haven, pp. 87–98.
Nowell, A., White, M., 2010. Growing up in the Middle Pleistocene: life history strategies and their relationship to Acheulian industries. In: Nowell, A., Davidson, I. (Eds.), Stone Tools and the Evolution of Human Cognition. University Press of Colorado, Denver, pp. 67–81.
Onat, T., Ertem, B., 1995. Age at menarche: relationships to socioeconomic status, growth rate in stature and weight, and skeletal and sexual maturation. Am. J. Hum. Biol., 7, 741–750.
Onat, T., İşeri, H., 1995. Rate of skeletal maturation in relation to secondary sexual development during female adolescence. Am. J. Hum. Biol. 7, 751–755.
Palkovich, A.M., 1984. Agriculture, marginal environments, and nutritional stress in the prehistoric southwest. In: Nathan, M., Armelagos, G.J., (Eds.), Paleopathology at the Origins of Agriculture. University Press of Florida, Gainesville, pp. 425–438.
Pantsiotou, S., Papadimitriou, A., Dourous, K., Priftis, K., Nicolaidou, P., Fretzayas, A., 2008. Maturational tempo differences in relation to the timing of the onset of puberty. Acta Paediatr. 97, 217–220.
Ponce de León, M.S., Bienvenu, T., Akazawa, T., Zollikofer, C.P.E., 2016. Brain development is similar in Neanderthals and modern humans. Curr. Biol. 26, PR665–PR666.
Ponzi, D., Flinn, M.V., Muehlenbein, M.P., Nepomnaschy, P.A., 2020. Hormones and human developmental plasticity. Mol. Cell. Endocrinol. 505, e110721.
Potts, R., 2012. Environmental and behavioral evidence pertaining to the evolution of early *Homo*. Curr. Anthropol. 53, S299–S317.
Prentiss, A.M., 2021. Theoretical plurality, the extended evolutionary synthesis, and archaeology. Proc. Nat. Acad. Sci. U.S.A. 118, e2006564118. https://doi.org/10.1073/pnas.2006564118
Prentice, A.M., Ward, K.A., Goldberg, G.R., Jarjou, L.M., Moore, S.E., Fulford, A.J., Prentice, A., 2013. Critical windows for nutritional interventions against stunting. Am. J. Clin. Nutr. 97, 911–918.
Ramirez-Rozzi, F.V., Bermúdez de Castro, J.M., 2004. Surprisingly rapid growth in Neanderthals. Nature 428, 936–939.
Randhawa, M., Seo, I., Liebel, F., Southall, M.D., Kollias, N., Ruvolo, E., 2015. Visible light induces melanogenesis in human skin through a photoadaptive response. PLOS One 10, e0130949.
Raymond, C., Marin, M., Wolosianski, V., Journalt, A., Longpre, C., Leclaire, S., Cernik, R., Juster, R., Lupien, S.J., 2021a. Early childhood adversity and HPA axis activity in adulthood: the importance of considering minimal age at exposure. Psychoneuroendocrinology 124, e105042.
Raymond, C., Marin, M., Wolosianski, V., Journalt, A., Longpre, C., Leclaire, S., Cernik, R., Juster, R., Lupien, S.J., 2021b. Adult women exposed to early adversity after eight years old show attentional bias to threat. Front. Behav. Neurosci. 15. https://doi.org/10.3389/fnbeh.2021.628099
Reed, T.E., Waples, R.S., Schindler, D.E., Hard, J.J., Kinnison, M.T., 2010. Phenotypic plasticity and population viability, the importance of environmental predictability. Philos. Trans. R. Soc. B 277, 3391–3400.
Reed, T.E., Schindler, D.E., Waples, R.S., 2010. Interacting effects of phenotypic plasticity and evolution on population persistence in a changing climate. Conserv. Biol. 25, 56–63.
Rosenberg, K.R., 2021. The evolution of human infancy, why it helps to be helpless. Ann. Rev. Anthropol. 50, 423–440.
Scheffler, C., Hermanussen, M., 2021. Stunting is the natural condition of human height. Am. J. Hum. Biol. 34, e23693. https://doi.org/10.1002/ajhb.23693
Scheiner, S.M., Holt, R.D., 2011. The genetics of phenotypic plasticity. X. Variation versus uncertainty. Ecol. Evol. 2, 751–767.
Schwartz, G.T., 2012. Growth, development, and life history throughout the evolution of *Homo*. Curr. Anthropol. 53, S395–S408.
Seifert, M.F., Watkins, B.A., 1997. Role of dietary lipid and antioxidants in bone metabolism. Nutr. Res. 17, 1209–1228.
Sisk, L.M., Gee, D.G., 2022. Stress and adolescence: vulnerability and opportunity during a sensitive period of development. Curr. Opin. Psychol. 44, 286–292.
Smith, J.M., Burian, R., Kauffman, S., Alberch, P., Campbell, J., Goodwin, B., Wolpert, L., 1985. Developmental constraints and evolution: a perspective from the Mountain Lake conference on development and evolution. Q. Rev. Biol. 60, 265–287.
Smith, B., 1992. Life history and the evolution of human maturation. Evol. Anthropol. 1, 134–42.
Smith, B., Tompkins, R., 1995. Toward a life history of the hominidae. Ann. Rev. Anthropol. 24, 257–279.
Smith, T.M., 2006. Experimental determination of the periodicity of incremental features in enamel. J. Anat. 208, 99–113.

Smith, T.M., Reid, D.J., Sirianni, J.E., 2006. The accuracy of histological assessments of dental development and age at death. J. Anat. 208, 125–138.

Smith, T.M., Tafforeau, P., Reid, D.J., 2010. Dental evidence for ontogenetic differences between modern human and Neanderthals. Proc. Nat. Acad. Sci. U.S.A. 107, 20923–20928.

Snyder-Mackler, N., Burger, J. R., Gaydosh, L., Belsky, D.W., Noppert, G.A., Campos, F.A., Bartolomucci, A., Yang, Y.C., Aiello, A.E., O'Rand, A., Harris, K.M., Shively, C.A., Alberts, S.C., Tung, J., 2020. Social determinants of health and survival in humans and other animals. Science 368, eaax9553. https://doi.org/10.1126/science.aax9553

Stearns, S.C., 1992. The Evolution of Life Histories. Oxford University Press, New York.

Steckel, R.H., 2009. Heights and human welfare: recent developments and new directions. Explor. Econ. Hist. 46, 1–23.

Stojanowski, C.M., Seidemann, R.M., Doran, G.H., 2002. Differential skeletal preservation at Windover Pond: causes and consequences. Am. J. Phys. Anthropol. 119,15–26.

Stuart-Macadam, P., 1985. Porotic hyperostosis, representative of a childhood condition. Am. J. Phys. Anthropol. 66, 391–398.

Stuart-Macadam, P., 1989. Porotic hyperostosis, relationship between orbital and vault lesions. Am. J. Phys. Anthropol. 80, 187–193.

Svefors, P., Pervin, J., Khan., A.I., Rahman, A., Ekstrom, E., El Arifeen, S., Selling, K.E., Persson, L., 2019. Stunting, recovery from stunting and puberty development in the MINIMat cohort, Bangladesh. Acta Paediatrica. 109, 122–133.

Tardieu, C. 1998. Short adolescence in early hominids, infantile and adolescent growth of the human femur. Am. J. Phys. Anthropol. 107, 163–178.

Temple, D.H., 2014. Plasticity and constraint in response to early-life stressors among late/final Jomon period foragers from Japan, evidence for life history trade-offs from incremental microstructures of enamel. Am. J. Phys. Anthropol. 155, 537–545.

Temple, D.H., 2019. Bioarchaeological evidence for adaptive plasticity and constraint, exploring life-history trade-offs in the human past. Evol. Anthropol. 28, 34–46.

Temple, D.H., Edes, A.N., 2022. Stress in bioarchaeology, epidemiology, and evolutionary medicine: an integrated conceptual model of shared history from the descriptive to the developmental. In: Plomp, K.A., Roberts, C.A., Elton, S., Bentley, G.R. (Eds.), Paleopathology and Evolutionary Medicine. Oxford University Press, Oxford. pp. 261–283.

Thayer, Z.M., Kuzawa, C.W., 2011. Biological memories of past environments, epigenetic pathways to health disparities. Epigenetics 6, 98–803.

Thayer, Z.M., Kuzawa, C.W., 2014. Early origins of health disparities, material deprivation predicts maternal evening cortisol in pregnancy and offspring cortisol reactivity in the first few weeks of life. Am. J. Hum. Biol. 26, 723–730.

Thompson, J.L., Nelson, A.J., 2011. Middle childhood and modern human origins. Hum. Nat. 22, 249–280.

Thompson, K., Quanjer, B., Murkens, M., 2020. Grow fast, die young? The causes and consequences of adult height and prolonged growth in nineteenth century Maastricht. Soc. Sci. Med. 266, 113430.

Tomasello, M., 1999. The human adaptation for culture. Ann. Rev. Anthropol. 28, 509–529.

Urlacher, S.S., 2023. The energetics of childhood: current knowledge and insights into human variation, evolution, and health. Yearb. Biol. Anthropol. 181, 94–117.

Visuthranukul, C., Abrams, S., Hawthorne, K., Hagan, J., Hair, A., 2019. Premature small for gestational age infants fed an exclusive human milk-based diet achieve catch-up growth without metabolic consequences at 2 years of age. Arch. Dis. Child. Fetal and Neonatal Ed. 104, F242–F247.

Waddington, C.H., 1957. The Strategy of the Genes. George Allen & Unwin, London.

Walasek, N., Frankenhuis, W.E., Panchanathan, K., 2021. An evolutionary model of sensitive periods when the reliability of cues varies across ontogeny. Behavior. Ecol. 33, 101–114.

Walker R.S., Gurven, M., Burger, O., Hamilton M.J., 2008. The trade-off between number and size of offspring in humans and other primates. Philos. Trans. R. Soc. B 275, 827–833.

Walker R., Gurven, M., Hill, K., Migliano, A., Chagnon, N., De Souza, R., 2006. Growth rates and life histories in twenty-two small-scale societies. Am. J. Hum. Biol. 18, 295–311.

Watts, R., 2013. Childhood development and adult longevity in an archaeological population from Barton-upon-Humber, Lincolnshire, England. Int. J. Paleopathol. 3, 95–104.

Watts, R., 2015. The long-term impact of developmental stress. Evidence from later medieval and post-medieval London (AD 1117 – 1853). Am. J. Phys. Anthropol. 158, 569–580.

Weibel, C.J., Tung, J., Alberts, S.C., Archie, E.A., 2020. Accelerated reproduction is not an adaptive response to early-life adversity in wild baboons. Proc. Nat. Acad. Sci. U.S.A. 117, 24909–24919.

Weisensee, K.E., 2013. Assessing the relationship between fluctuating asymmetry and cause of death in skeletal remains, a test of the developmental origins of health and disease hypothesis. Am. J. Hum. Biol. 25, 411–417.

Wells, J.C.K., 2016. Worldwide variability in growth and its association with health, incorporating body composition, developmental plasticity, and intergenerational effects. Am. J. Hum. Biol. 29, e22954.

Wells, J.C.K., 2019. Developmental plasticity as adaptation: adjusting to the external environment under the imprint of maternal capital. Philos. Trans. R. Soc. Lond. B 374, 20180122.

Wells, J., Stock, J., 2007. The biology of the colonizing ape. Am. J. Phys. Anthropol. 134, 191–222.

West-Eberhard, M.J., 2003. Developmental Plasticity and Evolution. Oxford University Press, Oxford.

Willey, P., Galloway, A., Snyder, L., 1997. Bone mineral density and survival of elements and element portions in the bones of the Crow Creek massacre victims. Am. J. Anthropol. 104, 513–528.

Wood, B., Smith, R. J., 2022. Towards a more realistic interpretation of the human fossil record.Quat. Sci. Rev. 295, 107722.

Woods, J.W., Milner, G.R., Harpending, H.C., Weiss, K.M., Cohen, M.N., Eisenberg, L.E., Hutchinson, D.L., Jankauskas, R., Cesnys, G., Katzenberg, M.A., Lukacs, J.R., McGrath, J.W., Roth, E.A., Ubelaker, D.H., Wilkinson, R.G., 1992. The osteological paradox, problems of inferring prehistoric health from skeletal samples. Curr. Anthropol. 33, 343–370.

Worthman, C.M., Kuzara, J., 2005. Life history and the early origins of health differentials. Am. J. Hum. Biol. 17, 95–112.