Hard Tissue Correlates of Growth Rate Variation in Primates

by

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ABSTRACT

This dissertation project explores the links between the ultimate drivers of variation in primate growth rates and their proximate (i.e., hormonal) underpinnings via a hard-tissue structure, the sella turcica. In doing so, it proposes a novel, non-destructive method for estimating individual somatic growth rates, which are presently difficult to infer in the hominin and primate fossil records. It also investigates the inter- and intraspecific effects of ecology and environment on extant primates' growth rates.

The ultimate causes, or selective pressures, shaping growth rate have long been the subject of anthropological research, but the proximate mechanisms that underpin variation in growth rate are less well studied. At the proximate level, somatic growth is the direct result of hormones produced by endocrine glands such as the pituitary. This project builds upon the relationship between the size of the pituitary, which is positively correlated with growth rate across mammalian taxa, and the sella turcica, the bony structure within which the pituitary gland is housed. By pairing 3D cranial morphology data with growth data from a well-studied primate population, this research tests whether the size of the nonhuman primate sella turcica reflects somatic growth rate. It also assesses how aspects of ecology and demography (i.e., ultimate causes such as resource availability, food quality, extrinsic mortality) relate to extant primates' somatic growth rates both within the study population and across a comparative sample of 51 species. It further explores whether these ecological variables also explain variation in relative sella turcica size; together, the complementary components of this dissertation contribute to a better understanding of primate growth.

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INTRODUCTION

Modern humans are characterized by a unique pattern of growth and development compared to our closest primate relatives. We wean earlier than would be expected given our brain and body size but reach reproductive maturity late, have relatively long juvenile and adolescent periods but short interbirth intervals relative to other living primates, and have long lifespans but a substantial post-reproductive period compared to both other primates and many mammals (as reviewed in Bogin, 1999b; Jones, 2011). Together, these variables describe how an organism allocates energy towards growth, maintenance, and reproduction throughout life. The schedule of energy allocation towards these competing demands is its life history (Jones, 2011; Stearns, 1992). These unique aspects of human life history, growth, and development have long been of interest to biologists and paleoanthropologists, in part because the trajectory of human growth and development is hypothesized to be linked to other unique human features such as our large brains, extensive cooperation, and complex, cumulative culture (see Dunbar & Shultz, 2007; Koster et al., 2020; Street et al., 2017). The evolution of modern human life history, however, can be difficult to trace in the hominin fossil record since many life-history traits leave little direct evidence in the fossil record. Understanding both the proximate and ultimate causes of variation in growth and developmental events is essential to test hypotheses about the evolution of modern human life history and the anatomical, social, and behavioral traits proposed to be linked to it.

1.1. Growth in the context of life-history theory

The timing and duration of various growth, developmental, and reproductive events define a species' or individual's life history.[1] Positioned "at the heart of any understanding of adaptation in evolutionary biology" (Jones, 2011: R708), life-history theory provides explanations for why and how organisms differentially allocate their energetic resources towards growth, maintenance, and reproduction (and related tasks such as raising offspring and survival) and how this relates maximizing reproductive fitness under different ecological conditions (Bogin, 1999b, 2003; Charnov, 1991; Charnov & Berrigan, 1993; Charnov & Schaffer, 1973; Kappeler et al., 2003; Stearns, 1989, 1992). Successful growth and reproduction are essential for the survival of individual organisms, of entire populations, and of species, so variation in life history traits should be understood at both inter- and intraspecific levels.

Both inter- and intra-specific variation in life history can be adaptive. Although natural selection acts on individual phenotypes generated by individual genetic differences to shape the "species-typical" life-history traits that emerge at interspecific levels of comparison, these species-typical traits are simply the means around which the individuals in a population vary (Ellis et al., 2009). To flip perspectives, intraspecific variation in life history, which is the product of both underlying genetic differences and phenotypic plasticity, thus exists within the range of values that have been shaped by

[1] Though frequently used interchangeably outside the scientific literature, *growth* and *development* are distinct processes. "Growth" refers to quantitative increases in size or mass, while "development" describes qualitative or quantitative changes from a state that is relatively undifferentiated or immature to one that is more highly organized or specialized (Bogin, 2012). In the context of development, "maturation" is the process of reaching biological, behavioral, and cognitive functional capacity, as well as the state once full functional capacity has been achieved (Bogin, 1999b).

natural selection over evolutionary time. Within-population differences in life history are important sources of variation, as they are tied to differential survival in response to local conditions. Selection favors traits that result in increased reproductive fitness, which is a product of survival and fecundity across an individual's lifespan (Stearns, 1986). In theory, the ideal solution is to maximize both survival (i.e., growth and maintenance) and reproduction. In practice, organisms have finite metabolic budgets; energy used for one purpose cannot be used for another. Simultaneously maximizing both survival and reproduction is impossible, and as a result, trade-offs must be made such that resources and energy are allocated differentially throughout life. Metabolic investments are made in different expenditures at different points in an individual's life relative to fitness payoffs (Kramer et al., 2009). Evolutionary theory stipulates that the parameter to be optimized is reproductive success, but achieving reproductive success requires that organisms first survive to reproductive age and then ensure that their offspring survive to reproduce as well. The specific trade-offs in resource and energy allocation that individuals make depend on what is most efficient for a particular ecology and environment, while working within the constraints of an individual's or species' intrinsic anatomical and physiological characteristics (Bogin, 1999a, 2012; Sibly & Brown, 2007). There are various ways to balance competing growth, maintenance, and reproductive demands, costs, and benefits, and different taxa, even closely related ones, have evolved different solutions. While variation exists within the group, Primates typically invest early in growth and delay reproduction, which carries the high cost of potentially not surviving to reach reproductive age (Gadgil & Bossert, 1970).

Because life-history patterns vary within species in response to local conditions, as well as between species in response to selective pressures that operate on evolutionary time scales, they represent both individual and species-level optima in the allocation of energetic resources. Across populations, differences in life-history patterns can reflect adaptations to local ecological conditions (Ellis et al., 2009), giving life-history theory the ability to inform evolutionary hypotheses about why and how different species meet challenges related to growth and reproduction. These challenges may be short-term ones faced by an individual (e.g., whether a parent should invest in its own health or the growth of its offspring during times of energetic or environmental stress), or may play out at the species level (e.g., whether it behooves mothers to have a greater number of offspring per litter or larger offspring per reproductive event) (Stearns, 1992). Life-history theory is particularly relevant for studies of human evolution, as the combination of life-history traits in modern humans is unmatched in other primate taxa and is one of the unique aspects of human biology (Schultz, 1960). Employing life-history theory to understand the non-human primate origins of human growth and development can help to contextualize and explain a range of phenomena related to the pattern and pace of growth and reproduction in modern *Homo sapiens*, from our overall slow pace of life (Bogin, 1999b, 2012; Hawkes, 2006; Kappeler et al., 2003; Robson et al., 2006) to our relatively early weaning (Galdikas & Wood, 1990; Humphrey, 2010; Kennedy, 2005; Robson et al., 2006; Van Noordwijk et al., 2013; J. W. Wood, 1990) to the evolution of long post-reproductive lifespans (Bogin, 1999a; Hawkes et al., 1998; Hill & Hurtado, 1991; Kaplan et al., 2000; Madrigal & Meléndez-Obando, 2008).

A key aspect of human life history is the pattern and pace of somatic growth. Growth is the background metronome against which other developmental events unfold and aspects of growth directly impact juvenile survival, age at first reproduction, and lifetime reproductive output (Altmann & Alberts, 2005; Charnov & Schaffer, 1973; Dmitriew, 2011; Walker et al., 2006). As a result, the trajectory of growth influences the evolutionary success of individuals, and ultimately, populations. Because organisms typically do not grow and reproduce simultaneously, many life-history models (e.g., Charnov, 1991) focus primarily on explaining the variation in age at maturity—i.e., the point at which organisms experience a dramatic shift in energy allocation away from individual growth towards reproduction—rather than the duration or rate of growth.

Simply focusing on the end point of growth, however, ignores important variation since a key feature of primate (and particularly human) growth is that growth rates are not static but instead vary throughout ontogeny (Bogin, 2012; Leigh, 2001). Models that gloss over differences in the duration of pre-maturity periods or fluctuations in rates of growth during these periods can be especially problematic for studies of modern humans since our late maturation age is the result of changes during infancy and/or juvenile periods, as well as the addition of novel childhood and adolescent periods (Bogin, 1999b, 2012). Different phases of life can have vastly different repercussions for the amount of parental investment required (Garber & Leigh, 1997), the behavior and skills of individuals (Boinski & Fragaszy, 1989; Ferrari et al., 2000; Fragaszy, 1990; Mayr, 1963), mortality (Bronikowski et al., 2016; Jones, 2011; Larson et al., 2016), and social structure (Dittus, 1979; Leigh, 2001; Machanda & Rosati, 2020), so models that fail to account for

aspects of pre-maturity growth cannot fully explain variation in age at maturation or provide a framework to explore which components may be the target of selection.

Dynamic life-history models (e.g., Charnov, 1993; Migliano et al., 2007; Jones, 2009) that incorporate variation in pre-maturity growth have allowed the development and testing of a number of evolutionary hypotheses about the selective pressures acting on primate life histories. The rate of somatic growth and the timing of changes in growth rate are adaptations to a particular ecological setting and are determined by both ultimate and proximate causes. At the ultimate level, growth rates are shaped by selective pressures related to ecology and environment, such as resource abundance and/or quality (Altmann & Alberts, 2005; Janson & van Schaik, 1993) and mortality risk (Charnov & Berrigan, 1993; Sibly & Brown, 2007). These ultimate causes have long been the subject of anthropological research (e.g., Charnov and Schaffer, 1973). In contrast, how variation in the proximate, hormonal mechanisms that underpin growth reflects exposure to specific selective pressures and respond to variation in resource abundance, resource quality, and mortality risk is not well studied across the order Primates. In this dissertation, I therefore seek to link the ultimate drivers of variation in primate growth rates to their proximate (i.e., hormonal) mechanisms. Given the central role of hormonal mechanisms in regulating growth, this work may be able to contribute to the discussion about how proximate mechanisms are linked to broader, evolutionary trends and the unique aspects of growth and development that characterize different primate species.

1.2. Ultimate causes of growth rate variation

At the ultimate level, interspecific differences in growth rates, like all life-history traits, are shaped by selective pressures related to ecological, environmental, and demographic variables. Life-history theory has traditionally considered extrinsic mortality risk as paramount in driving interspecific variation in life history—Williams (1957) hypothesized that decreases in mortality due to changes in extrinsic factors such as reduced predation or starvation would lead to a slower rate of senescence and longer lifespans. In general, species subject to high adult mortality rates and thus a greater risk of dying during their reproductive lifespan tend to exhibit aspects of a "faster" life history than species subject to lower adult mortality rates (Harvey et al., 1987). Empirical evidence reinforces this finding, as many mammalian species that can mitigate predation threats by flying, gliding, or living in predator-free environments have longer lifespans for a given body size than those species that cannot (Austad, 1993; Austad & Fischer, 1992; Bronikowski et al., 2011; Holmes & Austad, 1994).

Compared to many other mammals, primates tend to have slow life histories (Jones, 2011), which suggests an evolutionary history characterized by comparatively low mortality. Within the primate order, however, arboreal primate species (which face lower predation risks and thus lower mortality rates than terrestrial primates) do not have longer lifespans than terrestrial ones (Shattuck & Williams, 2010). This is likely a product of the long evolutionary history of arboreality in the primate lineage, which carries a low extrinsic mortality risk (Martin, 1990; Shattuck & Williams, 2010). Although a large body of work (e.g., Charnov, 1991; Charnov & Schaffer, 1973; Charnov, 1993a; Kozlowski & Weiner, 1997) corroborates the major role predation-related extrinsic mortality plays in shaping primate life history, the relationship between

growth rate—particularly variation in growth rate—and mortality risk within the primate order is not as straightforward.

In the case of primates, growth rate variation within the order may be better explained by differential mortality threats at particular stages of life, or "age-specific mortality," as well as mortality risks not directly linked to predation, such as population and resource density. Different models emphasize different phases of life, but most link growth rates to mortality threats during the juvenile or adult period. Juvenile- and adult-focused models are not necessarily mutually exclusive, and growth rate variation likely reflects trade-offs that attempt to most effectively mitigate both juvenile and adult mortality given a species' particular environment. Models built around juvenile mortality propose that the *slow* juvenile growth of some primate species is the product of *high* juvenile mortality (e.g., Janson & van Schaik, 1993). Such models specifically link juvenile mortality to resource availability and/or foraging efficiency rather than predation risk and so will be revisited in the review of these selective pressures' effect on growth rate. Other hypotheses contend that within-order variation follows the general mammalian trend, with ecological settings that carry high adult mortality risk favoring relatively fast growth and/or early maturation overall in both human (Kramer et al., 2009) and nonhuman primate populations (Charnov, 1993). These models assume that an organism has no control over the adult mortality rates to which it is subjected, but do not explicitly state whether mortality is due to predation, resource availability, or other causes such as infectious disease. When adult mortality risk is high, argues Charnov (1993), females must reproduce young—in other words, before they die. Since it takes time to reach adult body size, and survival and fertility are often size-dependent (Mangel

& Stamps, 2001), ecological settings that carry high adult mortality would therefore favor fast growth rates and smaller body sizes that are easier to achieve. When adult mortality is low, delayed maturity is permitted, and individuals can invest energy in themselves—rather than offspring—over a longer period of time (Charnov, 1993). Growth is thus able to continue unencumbered until the onset of sexual maturity, at which point energetic resources are shifted towards reproduction. In other words, individuals subject to low adult mortality can "afford" a longer pre-reproductive growth period so slower growth is only allowed once constraints related to mortality risk have been lifted.

Despite the diverse viewpoints regarding the life phase during which mortality plays the largest role, the influence of mortality on growth and development is supported by empirical evidence from modern human populations. South American Pumé forager girls, who experience seasonal under-nutrition, annual fluctuations in food supply, harsh epidemiological conditions, no health care, and a physiologically challenging environment—all of which imply high extrinsic mortality—do indeed have fast life histories as they mature quickly and reproduce early compared to other natural-fertility South American populations (Kramer, 2008; Kramer et al., 2009; R. Walker, Gurven, et al., 2006). The small body size phenotype of various pygmy populations is hypothesized to be the consequence of a trade-off between growth and reproduction in high mortality environments where selection favors early reproduction, and thus early sexual maturation, early cessation of growth, and consequently, short stature, all of which are typically characteristic of a faster life history (Migliano et al., 2007; Migliano & Guillon, 2012). These studies did not address growth rates, however, so it is not clear whether this environment led to earlier maturation and smaller statue due to faster growth over a

shorter period of time, or simply a shorter growth period. Whether small adult body size is the byproduct or a driver of high mortality rates is unresolved, however (Migliano et al., 2013), and alternative hypotheses propose that the pygmy phenotype is simply an adaptation to life in equatorial rainforests, as small body size is solution to a food-limited environment, dense forest vegetation, and a warm, humid climate (Perry & Dominy, 2009; Ramirez Rozzi et al., 2015). Like juvenile and adult mortality, rainforest environment and mortality-driven hypotheses are not mutually exclusive.

Other selective pressures that shape primate growth rates are those related to resource availability and energetics. Organisms have minimum energetic requirements that must be met to survive, and variation or uncertainty in one's food resources affects the ability to meet these needs. Because slow juvenile growth reduces metabolic costs, the ecological risk aversion hypothesis (ERAH) suggests that slow growth may be a way for juvenile primates to mitigate the risk of starvation that is encountered when unskilled juveniles must compete with skilled adults for scarce resources (Janson & van Schaik, 1993). While adult primates may guard against environmental uncertainty and periods of food scarcity through cognitive buffering (Morris et al., 2011; van Woerden et al., 2012) that provides them with an additional degree of social, behavioral, and dietary flexibility, this may not be the case for juveniles. Slow primate growth and its accompanying reduced energetic demands may therefore be an adaptation that reduces feeding competition between adults and juveniles and addresses mortality risks linked to starvation rather than predation. In wild populations, it can be difficult to tease out the relative effects of adult and juvenile mortality risk on growth rate, and predation is certainly a risk for juvenile primates, who are subject to higher predation risk than adults

(Janson & van Schaik, 1993; Stone, 2007). Under a mortality risk scenario, however, natural selection would be expected to favor fast juvenile growth, rather than the slow juvenile growth proposed by the ERAH. It nonetheless seems unlikely that *slow* juvenile primate growth could be explained by high juvenile mortality risk or high adult mortality risk.

Alternatively, even in the absence of resource competition, the correlation of slow growth with an extended juvenile growth period, large body size, and large brain size could be the product of diverting energy away from rapid body growth in order to support a large, energetically expensive brain (Foley & Lee, 1991; Kuzawa et al., 2014; Martin, 1996; Navarrete et al., 2011). Many of the same models that emphasize the role of mortality also consider the impact of the significant costs of large brains on the evolution of long, slow life histories in primates. The costs of large brains are varied; they impose constraints on both growth and reproductive rates, require a long developmental period to reach maturity, and are energetically expensive. The relationship between life history and brain size is more nuanced than a straightforward causal one that can be explained by simple models, but brain size should be considered to understand the evolution of a particular life history.

Superficial evidence of the link between brains and life history can be seen in the grade shift between strepsirrhines and haplorhines in both life history and brain size (Martin, 2003), and in the observation that many human ontogenetic idiosyncrasies (such as the period of slow early body growth) occur at the same time as major brain growth and developmental milestones (Leigh, 2001). Changes in the brain, however, do not always result in the same suite of changes in life history, growth, and development across

animals. Taxa such as snakes and lizards have relatively small brains but body size-adjusted lifespans equal to or greater than those of primates (Bronikowski et al., 2011; Charnov & Berrigan, 1993), while bats have relatively small brains yet long lifespans for their body size (Deaner et al., 2003). Comparative analyses are also sensitive to the taxonomic level at which they are performed and the taxa included in the sample. Work on strepsirrhines by Kappeler (1996), for example, failed to find a link between variation in life history and brain size, despite mammal-wide associations between the two (Harvey & Clutton-Brock, 1985) and correlations between larger brains and some typically "slow" life-history traits identified in strepsirrhine samples by other researchers (e.g., Barrickman & Lin, 2010; Catlett et al., 2010).

It is likely that increases in brain size did not drive the evolution of a particular life-history profile, but rather that the correlation between brain size and life history stems from common underlying selective pressures such as energetic constraints, particularly those on the mother (Leigh, 2004; Leigh & Blomquist, 2007). Although all brain tissue is metabolically costly for all species due to the amount of energy that is required for its maintenance (Isler & Van Schaik, 2006; Mink et al., 1981), the high encephalization quotient of humans takes these costs to an extreme. Neural tissue accounts for about 20-25% of the resting or basal metabolic rate (BMR) in adult humans (Mink et al. 1981), with an increased energetic requirement early in life that peaks around 66% of BMR between 4.2-4.4 years of age (Kuzawa, 1998). This pattern of peak brain energetic demands is inversely related to body growth patterns (Kuzawa et al., 2014), lending support to the hypothesis that human body growth slows to compensate for peak brain development. Thus, much research on the influence of the brain on life history

focuses on *Homo sapiens*, but it is expected that the same selective pressures and costs are also relevant for nonhuman primates and mammals, albeit perhaps to a lesser extent that reflects their different brain sizes and the specifics of their ecology. Though smaller primate brain sizes are known to be linked to food scarcity (i.e., decreased energy availability) and life-history variables such as shorter interbirth intervals (Taylor & van Schaik, 2007), there appears to be no trade-off between somatic and brain growth rates in primates, as somatic growth in highly encephalized species such as humans is slowest during the juvenile period, once brain growth is close to or is fully complete (Barrickman, 2016).

A slightly different argument focuses on the costs of maintaining brain tissue, suggesting that the great energetic costs of supporting a large brain prohibit large-brained species from growing quickly into adults (Ross & Jones, 1999). According to this argument, brain size primarily constrains body growth rates after weaning, once offspring are nutritionally and energetically independent of their mothers. Ross (2003) argued that this is indicated by the correlation of juvenile growth rates (rather than fetal or infant ones) with brain size. As large brains are metabolically expensive, the low juvenile somatic growth rates of modern humans could be a form a metabolic risk aversion that has positive, knock-on effects for complex learning and brain development processes (Leigh, 2001). The slow somatic growth period between weaning and puberty (which corresponds with the most brain development and the costly acquisition of skills and knowledge) may be a way to offset or subsidize the brain's metabolic costs that carries with it a relatively long pre-reproductive life as a secondary consequence (Kaplan et al., 2000; Kuzawa et al., 2014; Leigh, 2001). The timing of growth thus represents a trade-

off. The low reproductive productivity that corresponds to delayed reproduction is an evolutionary cost, the benefits of which are reaped later in the form of higher production during adulthood (Kaplan et al. 2000). This high payoff is further manifested in surpluses that can be diverted to developing altricial offspring, thus perpetuating the cycle in future generations. Given the costs of the learning period and the significant metabolic costs of a large brain, however, this is only an effective strategy if it is coupled with delayed maturation and if individuals enjoy reduced mortality risk (H. Kaplan et al., 2000). In general, models that focus on increased brain volume and its associated costs propose that large brains "anchor" slow primate growth rates (Charnov, 1993; Leigh & Blomquist, 2007; Martin, 1996), although the direction of causality in the correlation between large brain size and slow growth can be difficult to determine.

Variation in growth rate is also linked to diet and the availability of food resources. Mammalian species that specialize in abundant and reliable foods have higher mass production rates across litters than species that do not (Sibly & Brown, 2007), but many primates rely on fruits that can be seasonally unpredictable or scarce (Chapman et al., 1999). This can lead to energetic shortfalls (Dewar & Richard, 2007; Knott, 1998; S. J. Wright et al., 1999), so slow growth and the accompanying reduced metabolic costs in primates might be adaptations to inherent irregularity in food resources or periods of food scarcity (Jones, 2011). The ERAH thus predicts that folivorous primates, who would not be subjected to the same levels of uncertainty as primarily frugivorous species, should have accelerated life histories and grow faster relative to closely related frugivorous ones. This is supported by empirical evidence (e.g., Conklin-Brittain et al., 1998; Van Noordwijk & Van Schaik, 2005; Wich et al., 2007). Folivorous gorillas, for example,

experience accelerated growth rates and an extended early growth spurt compared to humans and chimpanzees (Leigh, 2001). Comparisons between congeneric populations living in different environments (e.g., Breuer et al., 2008; Yamagiwa et al., 2012) further support the idea that physical maturation schedules are connected to different ecological conditions: more frugivorous western gorilla populations wean later and undergo slower physical maturation than more folivorous mountain gorillas (Breuer et al., 2008).

Still, not all research points towards a straightforward explanation of faster growth along dietary lines. Energetic factors such as the degree of maternal investment may explain the *slower* life histories of some folivores compared to frugivores (e.g., Godfrey et al., 2004). Although not an explicit test of variation in growth rates and relying on a small sample size, a study of closely related macaques (frugivores) and colobines (folivores) failed to find evidence of life-history differences between dietary groups; rather, provisioned populations had shorter gestation lengths and inter-birth intervals than wild ones (Borries et al., 2011). These results imply that the accessibility and availability of resources—rather than food type—is a stronger driver of variation in life history in these populations. This idea is reinforced by evidence that provisioning typically accelerates life-history schedules (Asquith, 1989; Gilmore & Cook, 1981; Kiltie, 1982).

Many of these ultimate causes of interspecific variation in growth rates share a common theme: energetics. Energy budgets have been proposed to be central to the evolution of modern human features such as a large brain and a high reproductive output coupled with slow growth (Pontzer et al., 2016), and the concept of energetic trade-offs is a central pillar of life-history theory (Bogin, 2012; Jones, 2011). Energetic costs can be

addressed by adjusting body growth rate, as slow body growth is accompanied by reduced energetic requirements compared to fast growth. Energetic costs independent of extrinsic mortality rates are more comparable to density dependent effects on life history. Furthermore, growth rate differences between folivores and frugivores, as well as between provisioned and non-provisioned populations, suggests that it is ultimately the amount of energy available—regardless of its source—that plays a key role in determining the rate of growth. After removing the effects of body size, an individual's mass production rate is tightly linked to food supply and predation risk across mammals (Sibly et al., 2014), yet interspecific variation in primate growth rates has not been explored within a comparative phylogenetic framework that considers both mortality risk and available energetic resources. Indeed, the ultimate causes outlined above have largely been explored independent of one another, without testing their relative effects on primate growth rates in a multivariate analysis that simultaneously takes into account shared ancestry between species and multiple predictor variables.

Selective pressures provide evolutionary explanations for differences in growth rate and life history between species (and to some degree, between different populations of the same species), but variation in experienced environment affects individuals in a different way than they do populations. Much intraspecific variation in growth, particularly within populations, is due to developmental plasticity. While some intraspecific variability in life-history traits is the direct result of genetic variation (e.g., Baker et al., 1993; Williams-Blangero & Blangero, 1995), mechanisms such as developmental plasticity allow trait expression to vary within a population in response to local conditions (Forsman, 2015; Wells & Johnstone, 2017). Though phenotypic or

developmental plasticity has been dismissed as noise that obscures underlying adaptations (West-Eberhard, 2003), plasticity is an important aspect of adaptive flexibility and success. Developmental plasticity is the potential for genetically similar individuals to express different phenotypes depending on conditions experienced during early development (Monaghan, 2008).

Within primate populations, the availability and quality of food resources are major driving forces behind the pace of growth (Macho, 2017; Ramirez Rozzi et al., 2015). Better nutrition and higher quality diets are linked to faster growth in humans (Stein et al., 2004) and nonhuman primates (Altmann & Alberts, 2005; Borries et al., 2011; Watanabe et al., 1992). Because slower growth reduces metabolic costs, an intraspecific application of the ERAH suggests that the ability to dynamically alter growth to match local conditions may improve survival and be an effective way to address uncertainty in environment or diet (Lee & Kappeler, 2003). Though some studies claim faster growth in response to a challenging environment (e.g., Berghänel et al., 2017), the prevailing view is that slower growth (Bogin et al., 2007) is adaptive under adverse conditions.

Plasticity is greatest early in life (Wells & Johnstone, 2017) and early life conditions lay the foundations for later-life milestones across mammals (Douhard et al., 2014; O'Rand & Hamil-Luker, 2005; Pigeon & Pelletier, 2018; Roseboom et al., 2006; Tung et al., 2016; Weibel et al., 2020). Furthermore, early-life conditions affect not only focal individuals but also their offspring: mothers who themselves experienced better early life nutrition gave birth to higher birth weight and faster growing offspring than those born to malnourished mothers (Bogin et al., 2007). The rate of postnatal (i.e., post-

birth and pre-weaning) growth is sensitive to environmental conditions and the degree of maternal investment (Altmann & Alberts, 2005). Further within-population differences in experienced environment are linked to stress levels and, in hierarchical species, dominance rank. Intraspecific correlations between slow growth rate and increased survival under stressful environmental conditions are documented from the single-celled bacteria (Gray et al., 2019) and yeast (*Saccharomyces cerevisiae*; Zakrzewska et al., 2011) to fish (Nishimura et al., 2007; Slotte et al., 2018) and primates. In hierarchical primate populations, growth and development are affected by maternal rank via increased stress in low-ranking individuals (and their offspring) as well as preferential access to resources by those of higher rank (Altmann & Alberts, 2005; Onyango et al., 2008). Indeed, Lee and Kappeler (2003) found evidence of early growth-rate plasticity in response to ecological, sociological, and reproductive factors, but their analyses were only carried out at the species level and thus were unable to link local conditions to either population-level or individual variation.

1.3. Proximate causes of growth rate variation

Evolutionary hypotheses and environmental explanations of variation in life history have received substantial attention from both biologists and anthropologists, providing important insights into the ecological conditions and selective pressures that may have shaped the diverse life-history profiles found across and within taxa. The causes of variation in life history upon which selection can act are proximate mechanisms, which are comparatively less well-studied than ultimate mechanisms. At the proximate level, a number of hormones are related both directly and indirectly to growth

and reproduction and therefore substantially impact life history and behavior (e.g., Crespi et al., 2013; Holman & Wood, 2001; Holzenberger et al., 2003; Swanson & Dantzer, 2014; Wingfield & Romero, 2000).

In mammals, these hormones are produced by endocrine glands such as the thyroid and pituitary. Growth is regulated by hormones such as growth hormone (GH) and insulin-like growth factor 1 (IGF-1) (Liu et al., 1993; Swanson & Dantzer, 2014). Growth hormone production in the pituitary is stimulated by growth hormone-releasing hormone (GHRH, also called somatocrinin) and inhibited by growth hormone-inhibiting hormone (GHIH, or somatostatin), both of which are secreted by the hypothalamus. GH is synthesized and secreted in the anterior pituitary by cells called somatotrophs and has both direct and indirect effects. It is directly involved in metabolism as it triggers adipocyte triglyceride breakdown and oxidation, stimulates protein anabolism (the formation of proteins from amino acids, which is essential for the growth of new tissues), and is one of the many hormones that maintains normal blood glucose levels.

Most of GH's effects on overall body growth are indirect. GH plays an integral role in a signaling pathway called the hypothalamic-pituitary-somatotropic (HPS) axis, which produces the insulin-like growth factor 1 (IGF-1) that is essential for postnatal growth (Achermann & Jameson, 2010; M. Lu et al., 2019). Along this axis (Figure 1.1), GH produced in the pituitary targets the liver and stimulates it to synthesize IGF-1. IGF-1 is essential for brain (Webb et al., 2012), bone, and muscle growth (Liu et al., 1993), as it stimulates both the production and differentiation of chondrocytes and the differentiation and proliferation of myoblasts, as well as amino acid uptake and protein synthesis in muscle and other tissue (Kannan, 1987). IGF-1 is also part of a negative feedback loop

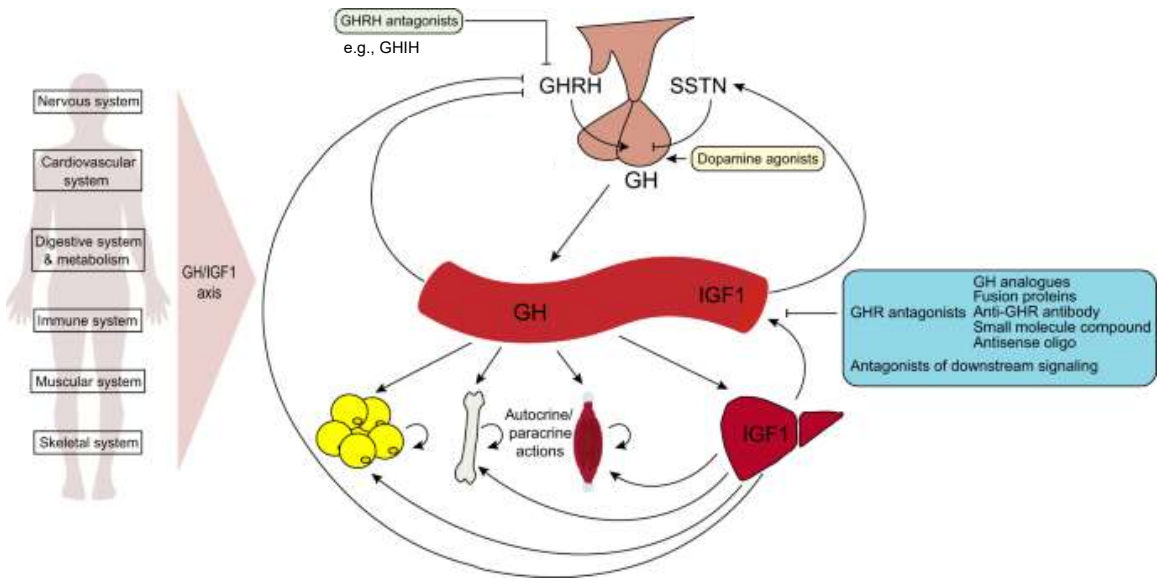


Figure 1.1. Growth hormone (GH) signaling pathway. GH is secreted from the anterior pituitary and is stimulated (indicated by arrow) by growth hormone releasing hormone (GHRH) and is inhibited (indicated by bar) by somatostatin (SSTN). GH targets the liver to produce insulin-like growth factor 1 (IGF-1). GHRH antagonists include growth hormone inhibiting hormone (GHIH) secreted by the hypothalamus. Image adapted from Lu et al. 2019.

involving GH: high levels of IGF-1 in the blood result in decreased GH secretion through direct suppression of somatotrophs and stimulation of the hypothalamus to release GHIH. The hormones produced by the GH/IGF-1 signaling pathway together result in increased growth and reproductive effort early in life, and genetic changes that trigger reduced signaling along the pathway contribute to an increased lifespan and alterations in the timing of life-history milestones (e.g., the onset of puberty) in a number of species (Holzenberger et al., 2003; Kenyon, 2010; Lithgow & Gill, 2003).

Further evidence of the key role that GH and IGF-1 play in regulating growth can be seen when these pathways are disrupted. Congenital IGF-1 deficiencies are linked to delayed skeletal maturation (Laron, 1984), and both GH (Tanner et al., 1971) and IGF-1 (Laron, 1984) deficiencies during childhood are linked to reduced growth and short

stature. When affected juveniles are treated with the deficient hormone, growth is stimulated and growth rate increases (Laron et al., 1993; Schoenle et al., 1982; Tanner et al., 1971). Conversely, pituitary tumors and disorders that cause the over-secretion of GH result in increased growth rates since higher levels of GH stimulates the liver to produce increased IGF-1 (Ayuk et al., 2004; Vierimaa et al., 2006).

Hormones mediate developmental plasticity, as hormonal axes such as the HPS axis alter levels of hormone production in response to environment and conditions experienced during development (Monaghan, 2008). Adjusting GH production can provide a mechanism to promote survival during periods of food shortage (Clemmons, 2004) and maternal malnutrition alters the hypothalamo-pituitary axis in newborn rats, resulting in retarded growth (Lesage et al., 2001).

Importantly, both hormone concentrations (Swanson & Dantzer, 2014) and pituitary gland volume (Kamilar & Tecot, 2015) are linked to species-level variation in life-history traits. In a comparative study of blood plasma IGF-1 concentrations across 41 mammalian species, higher levels of IGF-1 were linked to smaller body masses, shorter gestation periods, and younger ages at maturity (Swanson & Dantzer, 2014). Furthermore, the link between pituitary hormones and life history can be extended to the size of the gland itself. Season-specific hormone actions and changes in the size of the pituitary are observed in wild mammals, as intraspecific variation in pituitary gland size and cellular composition, which have implications for hormone production, correlate with growth rate, season, and reproductive cycle in bats (Richardson, 1979) and mongooses (Nelson & Inao, 1982). Both anterior lobe and whole pituitary volume are positively

correlated with a life-history axis that includes traits such as fetal and postnatal growth rate across a sample of 129 mammal species (Kamilar & Tecot, 2015).

Of course, other mechanisms beyond those directly related to pituitary volume and the amount of hormone secreted by the pituitary gland influence growth rates, so reducing the complex dynamics of growth and life history to simply pituitary gland products is certainly an over-simplification. Genetics, the number and affinity of protein and hormone receptors, and the number and affinity of binding proteins (Romero, 2004) all influence how hormones produced by the pituitary and other lymphatic organs behave, but current research nevertheless demonstrates that pituitary size can indeed be a proxy for hormone production and the downstream effects of those hormones on organismal biology (Kamilar & Tecot, 2015; Richardson, 1979). These results further suggest that the relationship between volume and growth rate may extend beyond the soft-tissue pituitary to hard tissue anatomy as well.

1.4. Pituitary anatomy and development

The pituitary gland is housed within the sella turcica of the sphenoid bone (Figure 1.2; Kannan, 1987). In humans, the gland occupies at minimum 80% of the sella turcica's hypophyseal fossa (Kannan, 1987). The pituitary itself is fairly small, weighing about 500-600 mg and measuring approximately 1.2 to 1.5 cm in diameter, although it can be slightly larger and vary in size with age (Macmaster et al., 2007), particularly during pregnancy (Elster et al., 1991; Scheithauer et al., 1990). The pituitary gland is enclosed in a dural covering and positioned inferior to the *diaphragma sellae*, the piece of dura mater through which the infundibulum (or infundibular process, a continuation of the pituitary

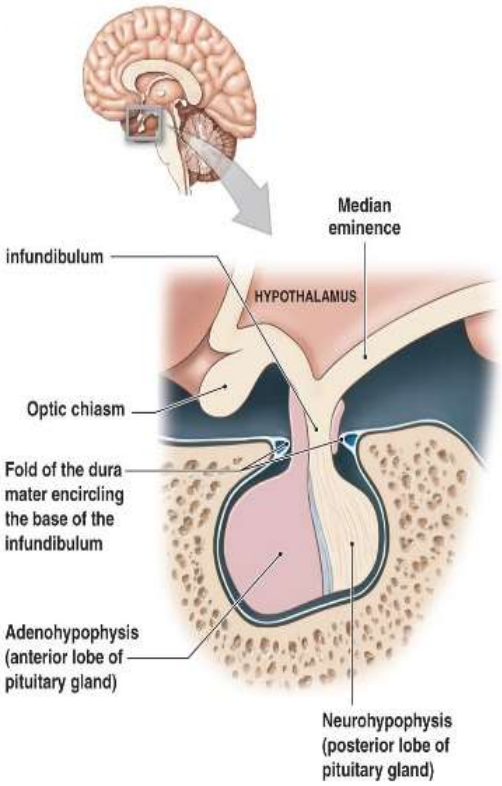


Figure 1.2. Human hypothalamic pituitary unit, sagittal section. Here, the "fold of the dura mater encircling base of the infundibulum" refers to the *diaphragma sellae*. Image from Pearson Education (2002).

stalk that connects it to the hypothalamus and the brain) passes (Fig. 1.2; Kannan 1987). Both anatomically and functionally, the gland is closely linked to the hypothalamus, the region of the forebrain that coordinates the autonomic nervous system as well as the pituitary. The median eminence of the hypothalamus is the origin of the pituitary stalk and contains the neurons that communicate with the pituitary. The hypothalamus is essential for endocrine regulation as its peptidergic neurons (i.e., neurons that secrete peptide hormones as their neurotransmitters) secrete important hormones, such as GHRH and GHIH, that communicate with the pituitary to regulate its hormone production (Kannan 1987).

Both pre- and postnatally, sella turcica and pituitary gland development are intertwined. During embryonic development, the nascent pituitary gland develops before the sella turcica (Sheng & Westphal, 1999) and it is fully formed before the sella turcica takes shape (Kjær, 2015). The medial ossification centers of the basisphenoid elements, which will form the future floor of the sella turcica, appear at a gestational age of between 15 and 17 weeks (Zhang et al., 2011), by which point all component parts of the pituitary gland already occupy the positions typical of those in adults (Solov'ev et al., 2008). The rudimentary fetal pituitary starts to form between 4 and 5 gestational weeks, with the lobes' adult positions attained by 7 weeks and functional differentiation achieved by 8 weeks when the gland's structural-functional units (such as the epithelial cords) have formed (Solov'ev et al. 2008). It thus follows that the morphology the sella turcica reflects, at least to some degree, the morphology of the pituitary gland. In humans, smaller sellae turcicae are found in individuals with smaller pituitary glands (Ferrier & Stone, 1969). Many changes in the shape of the pituitary gland result in corresponding changes in the shape of the sella turcica; sella malformations visible in profile radiographs are known to correlate with abnormal pituitary morphology and function and are therefore used in diagnosis (Kjær, 2015).

Although there is ample support for a link between sella turcica and pituitary morphology, there are a few potential qualifications. At the simplest level, while many correlations are robust, the association between the sella turcica and the pituitary gland in size and shape is not always one-to-one. In humans, sella turcica volume is known to vary with age (Alkofide, 2007; Axelsson et al., 2004; Chilton et al., 1983) and pituitary volume is known to vary with pregnancy and reproductive cycles (Elster et al., 1991;

Scheithauer et al., 1990). Furthermore, sella volume may not always reflect pituitary volume in pathological cases; the human pituitary is laterally and superiorly bound by soft tissue, which can permit expansion of the gland without a corresponding change in sella turcica volume (Kannan, 1987).

It is also possible that sella turcica morphology does not reflect only the pituitary gland. As a feature of the sphenoid bone and part of the basicranium—an area of the skull hypothesized to exhibit morphological changes (e.g., the degree of pre- and post-sella turcica flexion) in response to both posture and brain size (e.g., Ross & Ravosa, 1993)—it is plausible that sella turcica morphology could in part show signs of selection related to maintaining functional integration with the basicranium. The degree to which sella turcica morphology is affected by basicranial flexion independent of pituitary morphology has not been tested, but given that clinical tests employ sella turcica morphology to diagnose pituitary disease (e.g., Axelsson et al., 2004; Cacciari et al., 1990; Citton et al., 2016; Kjær, 2015; Kjaer et al., 1998; Schoenle et al., 1982; Spada et al., 1990) in modern *H. sapiens*, who are characterized by an extreme degree of basicranial flexion (Gould, 1975; Ross & Ravosa, 1993), this does not appear to be a concern for modern humans. Nonetheless, the lack of a close relationship between sella turcica morphology and basicranial flexion within modern humans does not mean that there is no relationship across primate species, so this may be worth keeping in mind as a potential confounding variable.

1.5. Introduction to hypotheses

Given the link between the pituitary gland and the sella turcica, sella turcica size may be a reliable proxy for the volume of pituitary hormones produced and thus be linked to the downstream effects of those hormones, such as the rate of growth. Many changes in growth rate occur throughout ontogeny, however, and the relationship between growth rate at a given age and sella turcica size in nonhuman primates is not known. In modern humans, absolute sella turcica volume increases throughout ontogeny, with volume (relative to stature) varying with age before reaching adult size (Chilton et al., 1983; Axelsson et al., 2004). Similar age-related changes in relative sella turcica size may also occur in nonhuman primates and may be linked to changes in growth via changes in hormone production. The sella turcica may thus provide a morpho-physiological connection between the proximate mechanisms that underpin the rate of growth (i.e., hormone production) and the ultimate ecological and environmental variables that structure growth rate variation both across primate species and within populations.

In this dissertation, I will explore the relationship between sella turcica size and growth in nonhuman primates at both intraspecific and interspecific levels. As very little is known about how the sella turcica varies across the primate order and across ontogeny in nonhuman primates, the overarching goal of this project is to test whether there is a consistent, predictable relationship between growth rate and various measures of sella turcica size at different taxonomic scales, using different samples. In Chapter 2, I investigate the possibility that in a well-studied *Macaca mulatta* population, relative sella turcica size varies across ontogeny and changes with growth rate in a predictable way. In doing so, I will establish whether relative sella turcica size is a reliable proxy for growth

rate across ontogeny in this nonhuman primate population by testing the hypothesis that relative size of the sella turcica tracks an individual's growth rate. This hypothesis is built upon the relationship between (a) the pituitary gland, which produces hormones that drive growth and the size of which is linked to variation in body growth rates across taxa (Monaghan, 2008; Kamilar & Tecot, 2015), and (b) the sella turcica, the bony structure within which the pituitary is housed (Figure 1.2; Kannan, 1987). Both absolute sella turcica size and sella turcica size relative to stature change across ontogeny in humans (Chilton et al., 1983; Axelsson et al., 2004), but it is not known whether these broad correlations hold in nonhuman primates, or whether ontogenetic changes in relative sella turcica size are linked to changes in growth rate.

Subsequent chapters will determine whether growth rates and relative sella turcica size reflect environmental and ecological conditions both intraspecifically (in the *M. mulatta* sample) and interspecifically (across a broad sample of 51 species). In Chapter 3, I explore how the local conditions experienced during development affect individual postnatal growth rates. Accepting that intraspecific phenotypic variation is a proxy for population-level developmental plasticity (Forsman, 2015; Lee & Kappeler, 2003), I examine the mediating effect of environment on growth by testing the hypotheses that (a) within species, individual growth rates respond to local environment and (b) more favorable conditions will result in faster growth rates and larger body size. This approach will not directly explore how intra-individual changes in growth across ontogeny are linked to developmental conditions, but will instead assess the population-level potential for growth variation in response to environment.

In Chapter 4, I employ phylogenetically informed methods to test which ecological factors (e.g., mortality risk and availability of energetic resources due to dietary regime) and sociocognitive factors (e.g., those related to brain size or juvenile-adult competition) best account for variation in growth rates and sella turcica size at the interspecific level. Extrinsic mortality risk, brain size, the degree of juvenile-adult competition, the quality and availability of preferred dietary resources, degree of maternal investment, degree of provisioning, and shared ancestry have all been proposed to structure variation in growth rates, but the relative contribution of these selective pressures is unclear. I thus attempt to elucidate which variables are the strongest drivers of growth rate by testing the hypothesis that interspecifically, both (a) growth rates and (b) the size of the sella turcica are influenced by the same combination of ecological and sociocognitive factors.

Ultimately, this dissertation aims to explore the link between primate growth rates and their hormonal underpinnings by understanding the relationship between growth rate and the sella turcica. In doing so, it also aims to develop a novel, non-destructive method for estimating growth rates that has potential applications to the fossil record and will provide a means to evaluate the population-level and evolutionary effects of ecology and environment on growth rates in living primates.

CHAPTER 2

INTRASPECIFIC RELATIONSHIP BETWEEN SELLA TURCICA SIZE AND GROWTH RATE

2.1. Introduction

Fluctuations in the tempo of growth across ontogeny characterize many mammalian species and the timing of these changes in growth is a hallmark of modern human growth and life history. Compared to other extant primates, *Homo sapiens* is distinguished by slow childhood growth, an adolescent growth spurt, extended juvenile period, and a late age at maturity (Bogin, 1999b; Jones, 2011). Understanding when and why this distinct growth profile emerged in the hominin lineage is of great interest to paleoanthropologists. Because the pattern and pace of somatic growth have few hard tissue signatures, however, growth is difficult to trace throughout the ~7 million years of hominin evolutionary history.

Inferences about the pace of growth in fossil specimens are often made by using cross-sectional assessments of changes in juvenile stature or cranial size (e.g., Anton & Leigh, 2003), the timing of molar emergence (e.g., Kelley & Schwartz, 2010, 2012; Smith et al., 2010), or dental histology-based methods that compare dental microstructure and mineralization to skeletal development (e.g., Dean & Smith, 2009; Rosas et al., 2018; S. L. Smith, 2004). These approaches, while useful, measure changes in size during growth across a population and thus require multiple individuals of different ages to build a growth curve against which to compare a focal specimen. Although individuals within a population vary in growth and size-at-age, cross-sectional methods rely on average

measures of size-at-age and cannot measure individual growth velocities directly (Antón & Leigh, 2003). Using cross-sectional, population-level growth curves also has the tendency to underestimate dynamic changes in growth trajectories such as growth spurts (Leigh, 1996). A slight shift in focus away from the outcomes of growth (i.e., body size) towards the proximate mechanisms that regulate growth provides an alternative avenue to study growth both in living populations and in the fossil record.

At the proximate level, growth is controlled by hormones, particularly the production of growth hormone (GH) and insulin-like growth factor 1 (IGF-1), which are essential for subadult brain, bone, and muscle growth (Berelowitz et al., 1981; Billestrup et al., 1998; Clemmons, 2004; Liu et al., 1993; M. Lu et al., 2019; Shea et al., 1987; Stolar et al., 1984; Webb et al., 2012; Webster et al., 1994) . Both GH and IGF-1 are produced along the hypothalamic-pituitary-somatotropic (HPS) signaling pathway. Along the HPS hormonal axis, growth hormone-releasing hormone (GHRH) is secreted from the hypothalamus and stimulates the anterior lobe of the pituitary to produce GH, which then targets the liver to produce IGF-1 (Laron, 2001; Liu et al., 1993; M. Lu et al., 2019; Webster et al., 1994). The HPS pathway is vital for successful postnatal growth (Achermann & Jameson, 2010) and changes in the production of both GH and IGF-1 affect somatic growth patterns. Elevated GH and IGF-1 levels increase growth in laboratory settings (Shea et al., 1987), while the short stature of human populations displaying the pygmy phenotype is attributed in part to low GH production and decreased IGF-1 levels (Shea & Bailey, 1996). Among human populations that do not display the pygmy phenotype, GH (Tanner et al., 1971) and IGF-1 (Laron, 1984) deficiencies during childhood result in reduced growth and short stature; when affected juveniles are treated

with the deficient hormone, growth is stimulated and growth rate increases (Laron et al., 1993; Schoenle et al., 1982; Tanner et al., 1971).

Importantly, both hormone concentrations (Buffenstein & Pinto, 2009; Swanson & Dantzer, 2014) and pituitary gland volume (Kamilar & Tecot, 2015) are linked to species-level variation in life-history traits. In a mammal-wide study of blood plasma IGF-1, higher IGF-1 levels were linked to smaller body mass, shorter gestation, and a younger age at maturity (Swanson & Dantzer, 2014); across a smaller range of taxa, lifespan was linked to GH, IGF-1, and thyroxine (whose production is regulated by pituitary hormones), which were all secreted in lower levels in long-lived rodent and bat species than in shorter-lived ones (Buffenstein & Pinto, 2009). Swanson and Dantzer's (2014) and Buffenstein and Pinto's (2009) work directly ties life-history variation to circulating hormone levels; hormone production and the biological traits that hormones govern can also be linked to the size of the glands that produce those hormones. Recalling that the pituitary gland produces GH, both whole pituitary volume and anterior volume are positively correlated with life-history traits such as postnatal growth rate across mammals (Kamilar & Tecot, 2015), which suggests that pituitary size serves as an indicator of both the volume of pituitary hormone production (with larger pituitaries associated with greater levels of hormone secretion; Nagel et al., 1997), and the magnitude of a hormonally driven response (in this case, faster rate of growth; Kamilar & Tecot, 2015). Given the mechanistic links between aspects of growth and development and hormone production (Buffenstein & Pinto, 2009; Kamilar & Tecot, 2015; Swanson & Dantzer, 2014), as well as those between hormone production and pituitary size (Nagel et al., 1997), further exploration of structures associated with the pituitary gland may reveal

additional correlates of life-history variation. In particular, it is possible the positive correlation between pituitary gland volume and growth rate extends beyond the soft-tissue pituitary to its surrounding hard tissues.

The pituitary gland is housed within the sella turcica of the sphenoid bone (Standring et al., 2008). The glandular tissue of the pituitary develops before the sella turcica during embryonic development (Sheng & Westphal, 1999), so it follows that the morphology the sella turcica reflects, at least to some degree, the morphology of the pituitary gland. In humans, smaller sellae turcicae are found in individuals with smaller pituitary glands (Ferrier & Stone, 1969) and sella turcica dimensions are used in clinical settings to diagnose abnormal pituitary conditions (Figure 2.1.; Kjær, 2015). Given the intimate association between the pituitary and the bony sella turcica that surrounds it, sella turcica size may also be a reliable proxy for the volume of the gland, and thus the amount of pituitary hormones produced, and may be linked to the downstream effects of those hormones, such as the rate of growth.

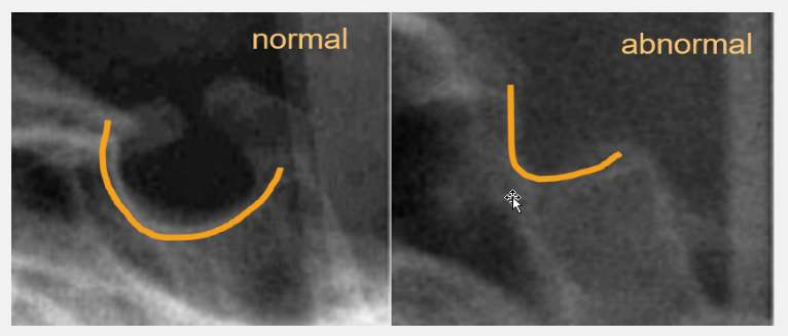


Figure 2.1. Radiographs with normal (left) and abnormal (right) sella turcica morphology used to diagnose pituitary conditions. From Kjaer 2015.

In both humans and nonhuman primates, many changes in growth rate occur throughout ontogeny (Bogin, 2009), and in nonhuman primates, the relationship between

growth rate at a given age and sella turcica size is not known. In humans, absolute and relative sella turcica volume increase across ontogeny, with sella turcica volume relative to stature varying with age before reaching stable, adult size (Axelsson et al., 2004; Chilton et al., 1983). Though these changes occur, it is unclear whether fluctuations in relative size correspond to changes in pituitary size and/or hormone production or whether they are simply idiosyncratic. Nonetheless, similar age-related shifts in relative sella turcica size may also occur in nonhuman primates. These changes may be linked to changes in pituitary hormone production and thus may reflect changes in growth. While the pituitary is also implicated in hormonal axes such as the one that produces growth-inhibiting cortisol (Achermann & Jameson, 2010), higher plasma cortisol is intraspecifically associated with smaller anterior pituitary volume (Sassi et al., 2001) through negative feedback loops (Achermann & Jameson, 2010). Increased cortisol under stressful conditions, therefore, is not expected to be linked to pituitary volume increases that would obfuscate the relationship among the pituitary, sella turcica, and growth. The sella turcica may thus provide a morpho-physiological connection between the proximate mechanisms that underpin growth (i.e., hormone production) and the ultimate ecological and environmental variables that structure growth rate variation across primates (see Chapter 1). Therefore, to evaluate potential links between proximate mechanisms and hard-tissue structures, I explore the possibility that in nonhuman primates, relative sella turcica size varies across ontogeny and changes with growth rate in a predictable way.

Building upon the relationship between the pituitary gland and the sella turcica, the bony structure that houses it, I hypothesize that the relative size of the sella turcica corresponds to an individual's growth rate. It is predicted that in the Cayo Santiago

Macaca mulatta study population, the sizes of individual sellae turcicae, relative to body size, change in tandem with changes in growth rate.[2] Because faster growth is linked to increased hormone production and larger pituitary size, and sella turcica morphology is known to correspond to pituitary morphology, I expect larger relative sella turcica sizes to be associated with faster growth. This hypothesis will be rejected if relative sella turcica size does not change across ontogeny or if growth rates are not positively correlated with relative sella turcica size in the study population.

Because this project is motivated by the need to better understand the relationship between sella turcica size and growth rate, the nature of the scaling relationship between sella turcica size and body size is not a central focus. Whether the relationship follows geometric scaling laws (e.g., sella turcica volume scales isometrically body size; Calder, 1983) or metabolic scaling laws (e.g., sella turcica volume scales at the quarter-power multiples common in biological systems; Brown et al., 2004) will not affect the predictive abilities of the models. Because the sella turcica is a bony structure (which suggests geometric scaling), but one with endocrine and metabolic links (which suggest metabolic scaling), sella turcica-body mass scaling could be reasonably expected to follow either geometric or metabolic scaling laws. Thus, this project permits an ancillary exploration of the nature of the scaling relationships between sella turcica size and body size to make inferences about whether sella turcica size simply increases geometrically as body size increases or whether it is best explained by metabolic or energetic factors that may be tied to its hormone production.

[2] In addition to the shifts in growth rate that occur across ontogeny, growth rate is also expected to change in response to experienced environment. This will be explored in Chapter 3.

2.2. Materials and methods

Study population and sample

The Cayo Santiago *Macaca mulatta* population is a well-studied, free-ranging population of rhesus macaques that, in 1938, was introduced to a 15.2 ha island that lies 1 km off the southeast coast of Puerto Rico. The population has been monitored continuously by researchers since 1956 and all individuals descend from 409 founding individuals (Widdig et al., 2016). Rainwater is available *ad libitum* and although the macaques are provisioned with commercial monkey chow, they spend about 50% of their time foraging on the island's vegetation (Marriott et al., 1989). Rhesus macaques live in social groups of multiple males and females; males disperse from their natal groups and females form stable matrilineal hierarchies (Vandenbergh, 1967). Macaques breed seasonally, although birth dates in a single breeding season may vary by up to 6 months. In 1956, a daily census began to record births, deaths, and changes in group memberships and provides pedigree, matriline, birth date, death date, and parity data (Widdig et al., 2016). Some individuals are weighed throughout life as part of routine veterinary care.

Most macaques on Cayo Santiago die of natural causes such as old age, disease outbreaks, natural disasters (e.g., hurricanes). Although up to 14% of infants die before the age of 1 year (Widdig et al., 2016), from time to time, individuals are culled to maintain a sustainable population size (Hernández-Pacheco et al., 2013). Deceased individuals are macerated and their skeletons are accessioned into the Caribbean Primate Research Center collection housed at the University of Puerto Rico in San Juan. The

associated skeletal material comprises > 2000 individuals from neonates to age 31.4 years.

From this skeletal collection, I selected a sample of non-pregnant individuals for whom body mass was recorded throughout life. To qualify for inclusion, an individual's body mass records must have contained at least 4 measurements taken throughout life and body mass measurements for adult individuals must have been taken during the juvenile growth period. Growth in the Cayo *M. mulatta* population ceases around age 6 or 7 (Leigh & Bernstein, 2006), but to ensure that no late developers were missed, the subadult sample included individuals from birth to 8 years old. Whenever possible, I sampled at least 4 males and 4 female subadults for each age between 0 and 8 years for a total of 59 subadults (Table 2.1); sufficient growth data were not available to maximize sample size in all age brackets. Similarly, I aimed to sample 2 males and 2 females at two-year intervals for each age over 8 years for a total of 29 adults (Table 2.1).

Table 2.1. Subadult and adult *M. mulatta* sample size. Sample sizes between groups are unequal because the target number of individuals was not available for all ages and sexes.

subadults			**adults**		
age (years)	male *n*	female *n*	age (years)	male *n*	female *n*
>1	2	1	**8 - 10**	2	2
1 - 2	3	4	**10 - 12**	2	2
2 - 3	4	4	**12 - 14**	2	2
3 - 4	4	5	**14 - 16**	1	2
4 - 5	4	5	**16 - 18**	1	2
5 - 6	4	4	**18 - 20**	1	2
6 - 7	3	4	**20 - 22**	1	2
7 - 8	3	5	**22+**	3	2

Linking growth rate to the sella turcica

Growth is a dynamic process and individuals experience changes in growth rate throughout ontogeny. As a physiological process, it is not subject to the same constraints as a skeletal feature such as the sella turcica. Although I seek in this project to explore the relationship between growth rate and the sella turcica, the sella turcica is fundamentally less plastic than growth and there are important considerations to acknowledge. Importantly, the sella turcica should not respond immediately to changes in hormone production and/or pituitary size. Instead, any potential changes in sella turcica morphology and/or size would be expected to be delayed by at least the 4 to 6 months that it takes cranial bone to remodel (Clarke, 2008). The sella turcica thus is expected to be a rough proxy for growth rate, not an exact predictor.

Growth rate can be measured multiple ways, and different measures may be more strongly or weakly correlated with sella turcica size depending on how (and if) the sella turcica responds to changes in pituitary size and/or hormone production. The specific aspects of growth rate that sella turcica size best reflects largely depend on (a) how tightly linked pituitary size is to the volume of hormones produced and (b) how responsive the sella turcica is to changes in pituitary size in the Cayo population. Because I did not have the means to explore these underlying assumptions by empirically measuring ontogenetic changes in hormone production and pituitary size in the study population, multiple measures of growth rate will be calculated. These different measures are intended to capture different aspects of growth in order to assess which, if any, are linked to absolute and relative sella turcica size.

Population-level growth is often modeled using mechanistic growth curve functions such as the von Bertalanffy, Gompertz, or logistic curves (Zullinger et al.,

1984). These three functions build upon the recognition that growth is not linear and model the inflection point and the asymptote. Unfortunately, the longitudinal body mass data utilized here are not easily fit by these equations[3]. Instead, I seek to approximate each individual's growth curve by fitting a parametric function that does not require underlying assumptions about the growth curve's shape to each individual's longitudinal body mass data. These curves can then be used to calculate instantaneous growth rates. Instantaneous growth rate, or the growth rate at a given point in time, is a precise measure of growth that is sensitive to the various changes in growth velocity that occur throughout ontogeny. Because the sella turcica is not expected to respond immediately to shifts in growth, less sensitive measures that time-average will also be calculated. These generalized measures simply consider the change in mass between a starting point (e.g., birth) and end point (e.g., maximum mass), thereby fitting a linear model and measuring the average change in mass over time.

Accepting for the moment that relative sella turcica size reflects changes in growth rate such that individuals with faster growth rates exhibit larger sellae turcicae relative to body size, some broad predictions can be made. For example, if the sella turcica is highly responsive to changes in growth rate, one might expect precise growth rate metrics, such as instantaneous growth rates, to be most strongly correlated with sella turcica volume. This possibility would be particularly compelling if relative sella turcica volume in subadults is most strongly correlated with the most recent instantaneous growth rate. It is likely, however, that sella turcica morphology is not plastic enough to

[3] Furthermore, the goal here is to best model the growth of each individual, rather than assess how well each individual's growth conforms to the theoretical predictions of commonly used growth curves.

respond to relatively rapid shifts in growth. Furthermore, most recent growth may not be an individual's fastest growth and sella turcica size may reflect the peak in pituitary hormone production (and thus the period of fastest growth). These specific changes may be too nuanced to be picked up in bony morphology, and it may simply be that the sella turcica reflects the sum total off all hormone production, making sella turcica size most strongly correlated with generalized measures of overall growth rate. Because there are no specific predictions about what aspects of growth (e.g., overall trends versus temporally specific events) sella turcica size may capture (beyond the assumption that absolute sella turcica size will not decrease if growth slows), multiple growth rate metrics were calculated.

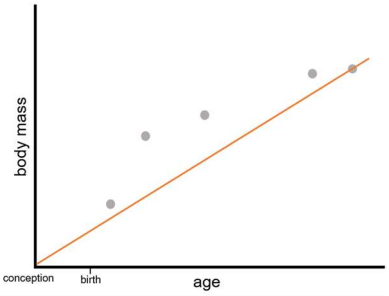
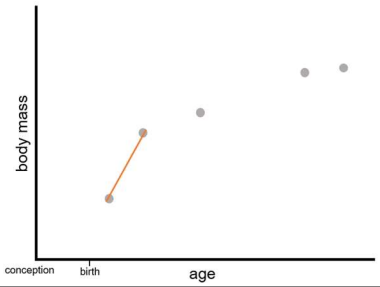
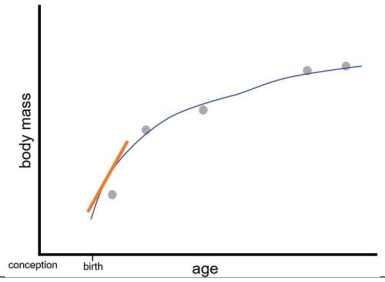
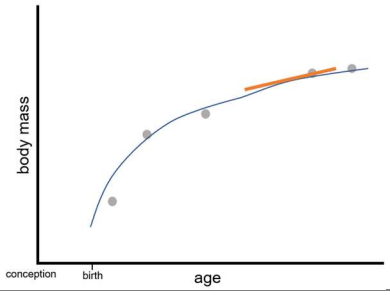
Two generalized metrics, overall growth rate ($GR_{overall}$) and maximum average growth rate ($GR_{max\ avg}$) were calculated. $GR_{overall}$ was calculated as maximum mass divided by the total growth period (age in days plus 166 days gestation; Silk et al., 1993). Because starting mass is 0, this method effectively calculates growth rate from conception and fails to account for differences in prenatal and postnatal growth rate. Instead, it averages across all of growth. Calculating growth rate from birth would be preferable, but body mass at birth was not available. As one alternative to address the oversimplifications inherent in calculating growth rate using maximum mass and age, maximum average growth rate ($GR_{max\ avg}$) was also calculated. For each individual, the change in mass between consecutive weighing events was divided by the time elapsed between those events. This effectively calculates the slopes of piecewise linear functions that fit the lines defined by consecutive (x,y) points where x =age and y=body mass and

could be considered a "generalized" (rather than specific) measure of maximum growth rate. The maximum value for each individual was selected as $GR_{max\ avg}$.

A derivative-based maximum growth rate ($GR_{max\ deriv}$) was also calculated by fitting a parametric function to each individual's body mass measurements over time using the *FlexParamCurve* package (Oswald et al., 2012) in R version 4.0.2 (R Core Team, 2020). Recalling that growth rate is the instantaneous rate of change (i.e., first derivative) of a growth curve, maximum growth rate occurs at the local maximum of the growth function's first derivative. The maximum of the first derivative occurs where the first derivative's derivative (i.e., second derivative of the original function) equals zero. Setting the second derivative equal to zero and solving for x allows one to calculate the time point (i.e., x-value) at which the first derivative's maximum (i.e., maximum growth rate) occurs. The value of the second derivative at this point is maximum growth rate (i.e., the maximum value of the first derivative and the maximum slope of the tangent line to the growth curve). Most recent growth rate (GR_{recent}) was also calculated using derivative-based methods. From the fitted growth curve, the first derivative was calculated for the point immediately preceding growth cessation (i.e., death) in the case of still-growing juveniles or immediately preceding the flattening of the growth curve in the case of adult individuals. Though the quality of all growth rate metrics depends on the quality of the growth records from which they are calculated, maximum growth rate metrics are more sensitive to the quality of growth records than $GR_{overall}$, and therefore will not be as robust to inter-individual variation in the number and frequency of body

mass measurements. The four different growth metrics ($GR_{overall}$, $GR_{max\ avg}$, $GR_{max\ deriv}$, GR_{recent}) are summarized in Table 2.2.

Table 2.2. Growth rate metrics calculated using Cayo Santiago body mass data. Slopes of the orange lines in figures represent growth rate

growth rate metric	summary and/or calculation	visual representation
$GR_{overall}$	overall change in mass from conception until maximum body mass, calculated as $\frac{max\ mass}{age\ in\ days+166}$	
$GR_{max\ avg}$	maximum rate of change in mass between two weighing events, calculated as $\frac{mass_{t_2} - mass_{t1}}{t_2 - t_1}$ where $mass_t$ is body mass at time t and t_1 and t_2 are consecutive	
$GR_{max\ deriv}$	local maximum of fitted growth function's first derivative	
GR_{recent}	first derivate at time preceding growth cessation	

Ultimately, though this project is built upon the strong, species-level correlations between postnatal growth rate and adult pituitary size (Kamilar & Tecot, 2015) and pituitary size to sella turcica size (Ferrier & Stone, 1969), there is no *a priori* reason to assume that achieved sella turcica size is linked to postnatal growth rate but not overall growth rate or maximum growth rate. The correlations of each of these growth rate measures with sella turcica size will help to inform interpretations of how and why sella turcica size changes (if at all) with respect to growth and hormone production.

Data collection

The crania of the 54 subadult and 44 adult macaques were scanned with the Bruker SkyScan 1173 microCT (μCT) scanner housed in the VizLab of the Institute of Human Origins at Arizona State University. Because scanner settings and scan resolution differed slightly for each specimen in an effort to optimize scans given differences in specimen size and field of view, parameters for each individual are listed in Appendix A. Scan reconstructions were imported into 3D Slicer v. 4.10, where they were converted from vector (RBG) volumes to a scalar grayscale volume. The volumes were cropped to reveal the sphenoid bone and the sella turcica (Figure 2.2a). Using Slicer's segmentation tool, the negative space created by the connecting the dorsum and tuberculum sellae (with the clinoid processes defining the lateral boundaries) was filled with a "virtual solid". This virtual solid was used to calculate the capacity of the empty sella turcica and approximate the volume of the pituitary gland (Figure 2.2b, c). The three-dimensional rendering of the sphenoid and sella turcica was visually inspected in all dimensions to ensure that the virtual solid volume did not spill over the lateral extent of the bony sella

turcica. The volume (mm^3) of the virtual solid was calculated from the volumetric representation using Slicer's segment statistics tool. This method utilizes voxel size calculated from z-x-y scan resolutions.

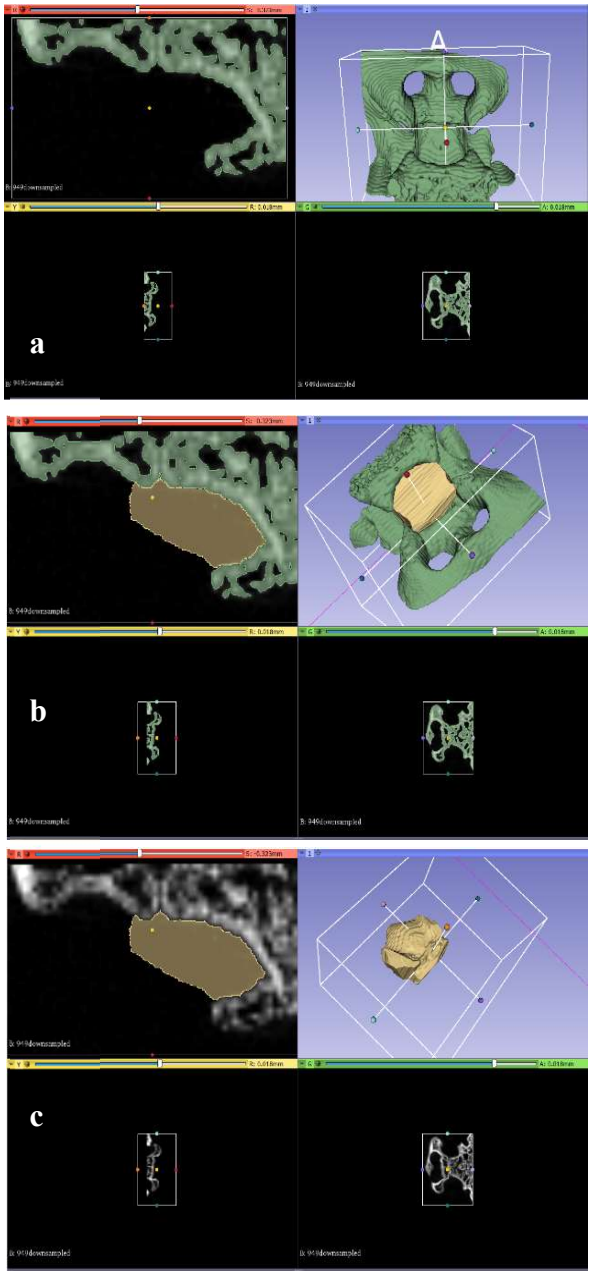


Figure 2.2. Workflow used to calculate sella turcica volume from µCT scan reconstructions. "Virtual solid" representing the volume of sella turcica is shown in gold; green indicates sphenoid bone.

Body size

Both body mass and stature were evaluated as measures of body size against which to compute relative sella turcica size. Mass and stature capture different aspects of growth and can vary independently. Though poor environmental conditions can result in stunted stature, mass generally tends to be a more plastic phenotype in response to environmental conditions (Green, 2001). The ratio of leg length to total stature (the cormic index) can be an indicator of environmental quality in modern human populations (e.g., Adak et al., 2006), but it was not possible to measure total stature in this skeletal population. Thus, body mass and humeral length were used to crudely capture two different measures of body size that are differentially affected by environmental quality. To account for effects of weight loss due to illness, the maximum body mass recorded in each individual's health record, rather than most recent body mass, was used. Humeral length at death was measured as a proxy for stature (Altmann et al., 1993). Humeri were photographed with a scale bar at the Caribbean Primate Research Center, and humeral

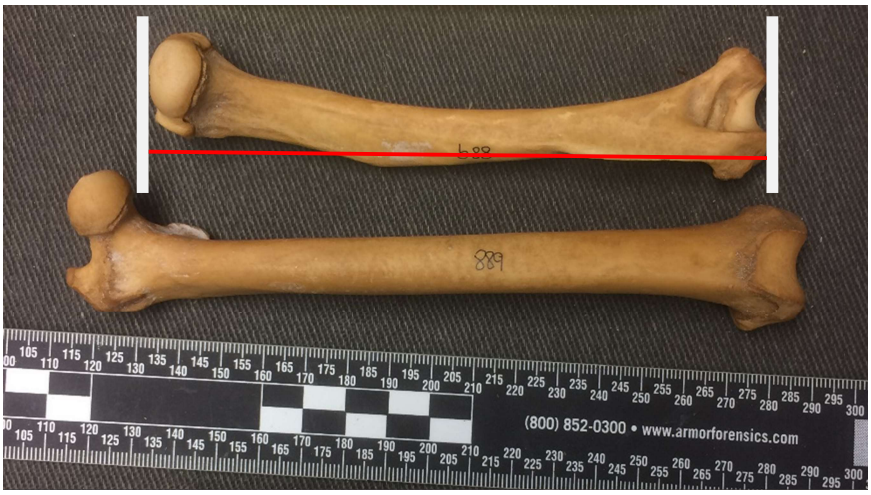


Figure 2.3. Humerus measurement protocol.

length was measured from scaled photographs using ImageJ. To approximate the use of an osteometric board, parallel lines defining the maximum proximal and distal extent of each humerus were placed on photos; maximum length was measured as the distance between the two lines (Figure 2.3). Body mass data were transcribed from veterinary records.

Analytical methods

All analyses were performed in R version 4.0.2 (R Core Team, 2020). Two sets of analyses were carried out: one with all individuals in the sample, and a second with only subadults. As males and females often exhibit differences in body mass and patterns of growth—and male children have larger sellae turcicae than female children (Chilton et al., 1983)—male and female subadults were analyzed separately. Though it was expected that subadults would exhibit stronger correlations between sella turcica size and growth rate than adults, the full sample across all ages was evaluated to assess the utility of sella turcica size as a predictor of growth rate in samples of unknown ontogenetic age. All morphological variables (body mass, humeral length, and sella turcica volume) were log-transformed to meet assumptions of normality.

Ordinary least squares linear models were fit to explore the allometric scaling relationship between sella turcica size and body size. Allometric scaling relates two biological variables (here Y and X) through the equation

$$Y=aX^b,$$

where X is some measure of body size (in this case, either body mass or humeral length), b is the allometric exponent, and a is a constant. A log-log transform yields the simple

linear equation

$$\log(Y) = \log(a) + b \log(X)$$

wherein the allometric exponent is the slope of the line relating the two variables.

Log-transformed sella turcica volume (Y) was regressed against both log-transformed maximum body mass (X) and log-transformed humeral length (X). To consider the effects of body size in models of growth rate, the residuals of these two fitted models were used as predictors in subsequent linear models (i.e., sella turcica volume residuals were used as the independent variable in ordinary least squares regressions of growth rate). Because multiple regressions are often preferred to analyses of residuals as a method of accounting for the effects of size (Freckleton, 2002), multiple linear regressions were also run with both sella turcica volume and either body mass or humeral length as predictors.

2.3. Results

All individuals

Across all individuals in this study, there was a positive relationship between sella turcica volume and maximum body mass, as well as between sella turcica volume and humeral length (Table 2.3). These relationships were negatively allometric; 95% confidence intervals for scaling exponents did not include and were smaller than values of isometry under models of both geometric scaling (isometric slope equals 1.0 for models of *sella turcica volume ~ body mass* and 3.0 for models of *sella turcica volume ~ humeral length*) and metabolic scaling (3/4, or 0.75, for models of *sella turcica volume ~ body mass* and 9/4, or 2.25, for models of *sella turcica volume ~ humeral length*). Across

all individuals, residual (i.e., size-corrected) sella turcica volume was a significant predictor of $GR_{overall}$ (coefficient = -0.002 for both stature-based and body mass-based residuals, $p < 0.001$; Table 2.3A), although R^2 values are very low (< 0.10; Table 2.4A, Figure 2.4). Multiple regressions of $GR_{overall}$ against sella turcica volume and both measures of body size yielded similar results (Table 2.4A), with sella turcica volume a statistically significant, yet weak, predictor of $GR_{overall}$ (coefficients = -0.0001, $p < 0.01$) and R^2 values again very low (= 0.08). In these multiple regressions, only sella turcica volume was a statistically significant predictor of $GR_{overall}$; body mass and humeral length were not significant predictors in the models (Table 2.4A). Neither residual-based metrics of sella turcica size nor sella turcica volume and body size in multiple regression models were significant predictors of $GR_{max\ avg}$ (Table 2.4B), $GR_{max\ deriv}$ (Table 2.4C), or GR_{recent} (Table 2.4D).

Table 2.3. Ordinary least squares linear regression models of sella turcica (ST) volume against body mass or humeral length, pooled adult and subadult sample (n = 86). Bolded entries are statistically significant predictor variables. Significance codes: *0.05, **0.01, ***0.001

model	intercept (95% CI)	predictor variable (95% CI)	adj. R^2
ST volume ~ body mass	4.13*** (3.97, 4.29)	**0.42*** (0.33-0.51)**	0.52
ST volume ~ humerus	-1.97** (-3.47, -0.47)	**1.40*** (1.09, 1.70)**	0.51

Table 2.4. Ordinary least squares linear regression models of growth rate, pooled adult and subadult sample (n = 86). Bolded entries are statistically significant predictor variables. ST = sella turcica. Significance codes: *0.05, **0.01, ***0.001

			predictor variable(s) (95% CI)		
	model	**intercept (95% CI)**	ST vol	body size	**adj. R^2**
	$GR_{overall}$				
A	**~ residual ST volume (body mass)**	0.002*** (0.002, 0.002)	**-0.002*** (-0.004, -0.0005)**	**--**	0.08
	~ residual ST volume (humeral length)	0.002*** (0.002, 0.002)	**-0.002** (NA, NA)**	**--**	0.09
	~ ST volume + body mass	0.003*** (0.003, 0.003)	**-0.0001** (-0.0002, -0.0001)**	max mass: 0.0001 (-0.0001, 0.0001)	0.08
	~ ST volume + humeral length	0.002 (0.0012)	**-0.0001** (0.00005)**	humerus: 0.00002 (0.00001)	0.08
B	$GR_{max\ avg}$				
	~ residual ST volume (body mass)	0.01** (0.0004)	0.01 (-0.033, 0.06)	--	0
	~ residual ST volume (humeral length)	0.02*** (0.005)	0.003 (NA, NA)	--	0
	~ ST volume + body mass	0.02 (0.016)	0.00004 (0.0001)	max mass: -0.0008 (0.002)	0.07
	~ ST volume + humeral length	0.013 (0.035)	-0.00001 (0.00014)	humerus: 0.00003 (0.00032)	0
C	$GR_{max\ deriv}$				
	~ residual ST volume (body mass)	0.004*** (0.001)	0 (0,0)	--	0
	~ residual ST volume (humeral length)	0.004*** (0.002, 0.004)	-0.002 (NA, NA)	--	0
	~ ST volume + body mass	0.008*** (0.0031)	-0.0001 (0.00003)	max mass: 0.0005 (0.00035)	0.02
	~ ST volume + humeral length	0.018* (0.0076)	-0.00001 (0.00002)	humerus: -0.00009 (0.00006)	0.03
D	GR_{recent}				
	~ residual ST volume (body mass)	0.004*** (0.001)	0.002 (-0.009,0.13)	--	0
	~ residual ST volume (humeral length)	0.004*** (0.001)	-0.001 (-0.01, 0.009)	--	0
	~ ST volume + body mass	0.007* (0.004)	-0.00001 (0.00003)	max mass: 0.0002 (0.0004)	0
	~ ST volume + humeral length	0.011 (0.0083)	-0.00001 (0.00003)	humerus: -0.00004 (0.00008)	0

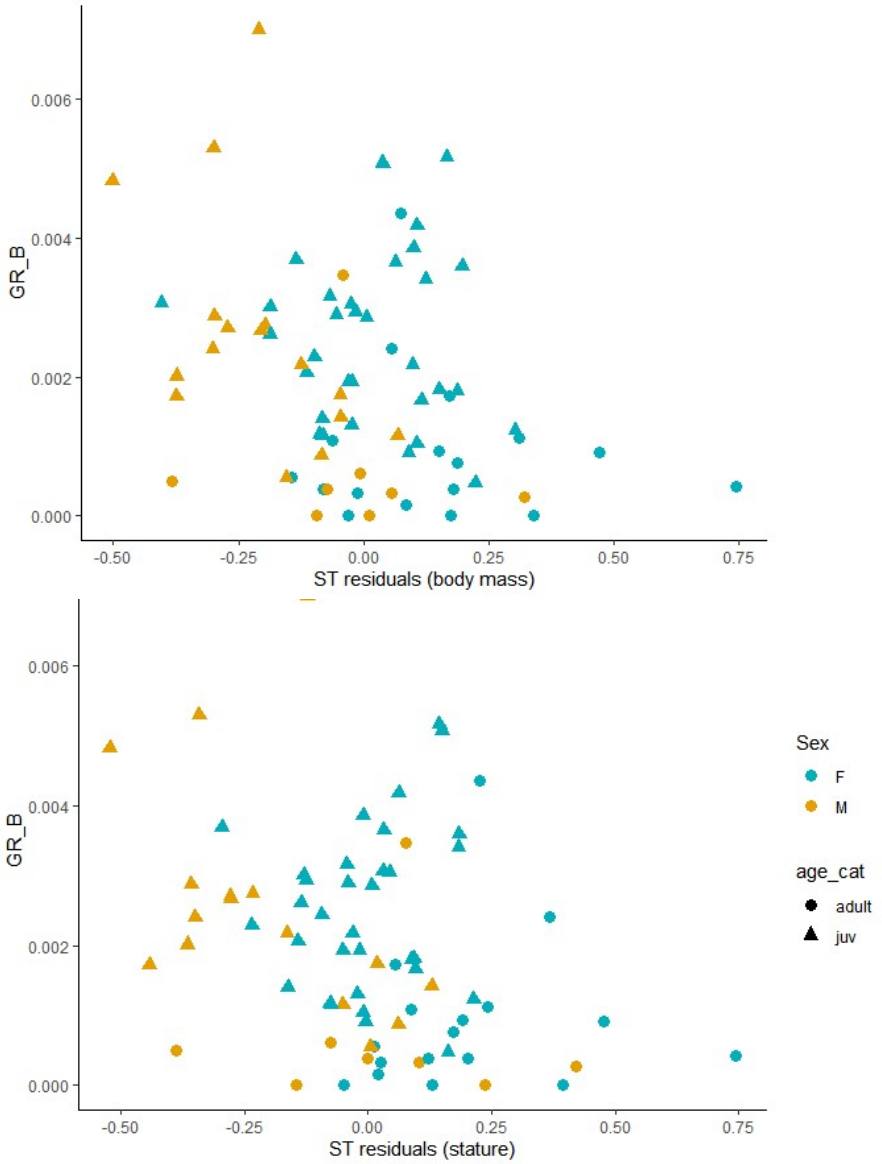


Figure 2.4. All individuals: overall growth rate plotted against residuals of sella turcica (ST) volume against body mass (top) and stature (bottom). Both variables are log-transformed.

Subadults

Within the female and male subadult samples, sella turcica volume was positively correlated with body mass and humeral length (Table 2.5A, 2.6A). As within the full sample, the relationship was negatively allometric and 95% confidence intervals for scaling exponents did not include values predicted by either geometric or metabolic

scaling. Within the female subsample, none of the predictor variables in models of growth rate was significant except for humeral length in the $GR_{max\ deriv} \sim ST\ volume + humeral\ length$ model (Table 2.5D). The largely non-significant results among females may be the result of a nonlinear relationship between growth rate and sella turcica size

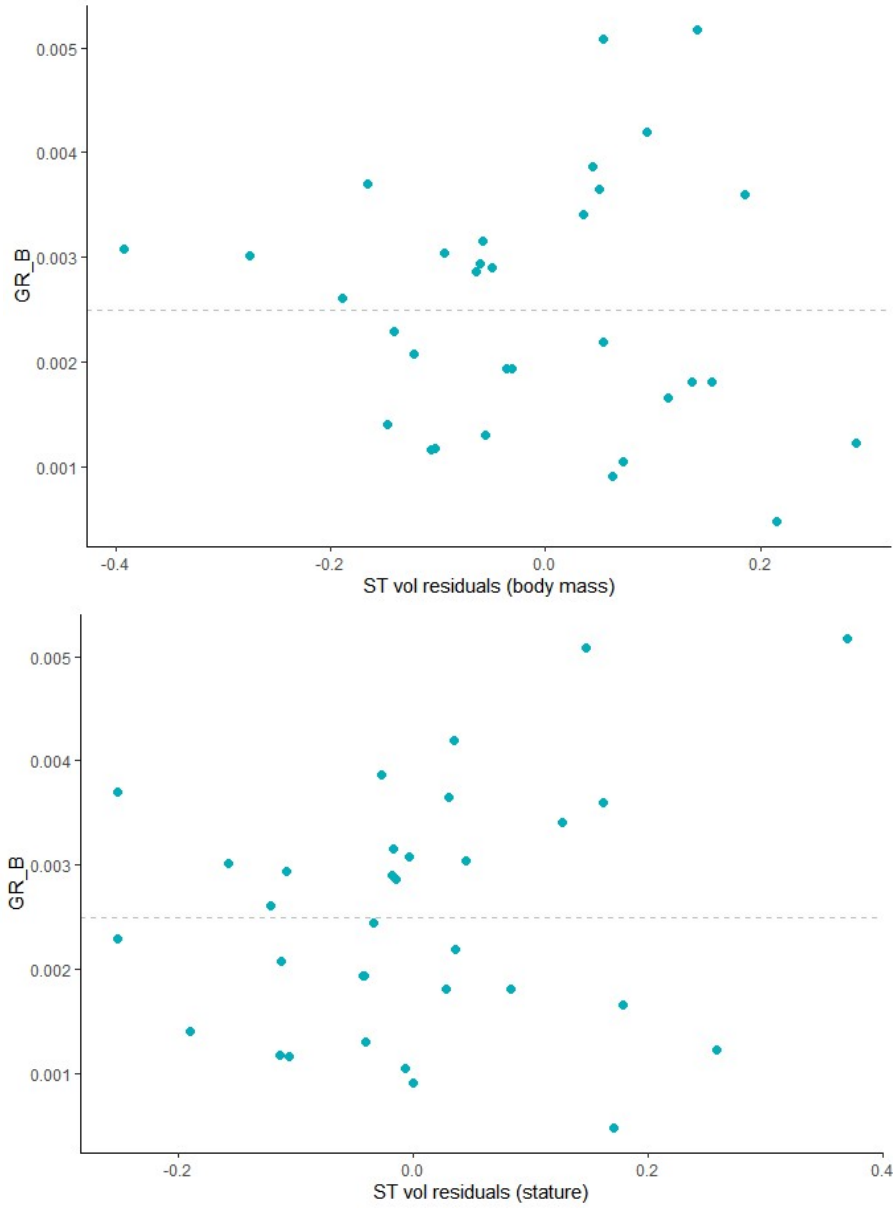


Figure 2.5. Juvenile females: growth rate plotted against residuals of sella turcica (ST) volume against body mass (top) and stature (bottom). Dashed line: $GR_{overall}$ = 0.0025 g/day.

(Figure 2.5). Visually, it appears that the direction of the relationship (i.e., positive vs. negative) between $GR_{overall}$ and sella turcica volume may depend upon the magnitude of growth—below a $GR_{overall}$ of about 0.0025 g/day (horizontal dashed line in Figure 2.5), there appears to be a negative relationship between $GR_{overall}$ and sella turcica volume,

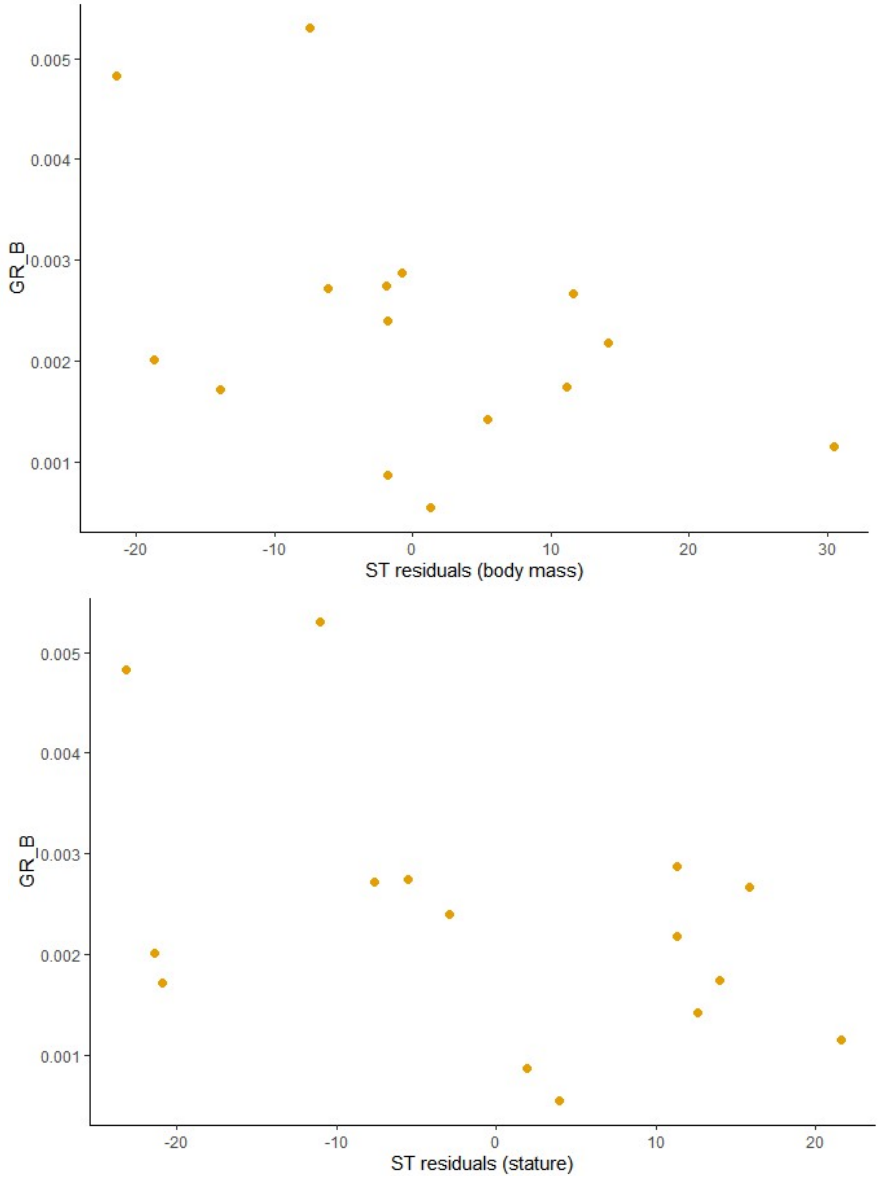


Figure 2.6. Juvenile males: growth rate plotted against residuals of ST volume against body mass (top) and stature (bottom).

while above 0.0025 g/day, the relationship appears to be positive. This possibility is explored further in the Discussion.

Among male subadults, the relationships between measures of growth rate and sella turcica volume were non-significant, with the exception of humeral length and body mass in multiple regression models of $GR_{overall}$ (Table 2.6B; Figure 2.6). Sella turcica volume approached significance in the two multiple regression models of $GR_{overall}$, as did the residuals of sella turcica size against body mass in the *$GR_{overall}$ ~ ST body mass residuals* model. In all models that approach significance, $GR_{overall}$ and sella turcica volume were negatively correlated. Though all males for which growth rate data were available were included, the number of juvenile males in the sample was much smaller than the number of juvenile females. The small male sample size prevented investigating whether males exhibit a similarly divergent, growth-rate-dependent pattern in the relationship between $GR_{overall}$ and sella turcica volume.

Table 2.5. Subadult female sample linear model results (n=35). Bolded predictors are significant at the alpha = 0.05 level; significance codes: ^0.10, *0.05, **0.01, ***0.001.

model	**intercept (95% CI)**	**predictor variable(s) (95% CI)**		**adj. R^2**
ST volume ~ body mass	4.30*** (0.09)	**0.34*** (0.22, 0.46)**		0.62
ST volume ~ humeral length	-1.77* (0.72)	**1.35*** (1.06, 1.65)**		0.71
$GR_{overall}$				
~ residual ST volume (body mass)	0.0025*** (0.0002)	-0.00017 (0.001)		0
~ residual ST volume (humeral length)	0.0025*** (0.0002)	0.0013 (-0.002, 0.005)		0.01
~ ST volume + body mass	0.0027* (0.0012)	ST vol: -0.0001 (0.0001)	body mass: -0.00004 (0.0002)	0
~ ST volume + humeral length	0.0049** (0.0015)	ST vol: 0.0001 (0.00001)	humerus: -0.00003^ (0.000016)	0.04
$GR_{max\ avg}$				
~ residual ST volume (body mass)	0.02** (0.009)	0.034 (-0.087, 0.15)		0
~ residual ST volume (humeral length)	0.02* (0.009)	0.032 (-0.13, 0.19)		0
~ ST volume + body mass	0.028 (0.05)	ST vol: -0.0002 (0.0006)	body mass: 0.004 (0.007)	0
~ ST volume + humeral length	0.034 (0.08)	ST vol: 0.0002 (0.0006)	humerus: -0.0003 (0.001)	0
$GR_{max\ deriv}$				
~ residual ST volume (body mass)	0.004*** (0.001)	-0.002 (-0.019, 0.15)		0
~ residual ST volume (humeral length)	0.004*** (0.001)	0.006 (-0.02, 0.03)		0
~ ST volume + body mass	0.016* (0.007)	ST vol: -0.00008 (0.00008)	body mass: 0.0003 (0.001)	0.08
~ ST volume + **humeral length**	0.039*** (0.011)	ST vol: 0.00006 (0.00008)	**humerus: -0.0003* (0.0001)**	0.24
GR_{recent}				
~ residual ST volume (body mass)	0.006* (0.002)	0.008 (-0.024, 0.039)		0
~ residual ST volume (humeral length)	0.006* (0.002)	0.005 (-0.37, 0.047)		0
~ ST volume + body mass	0.028 (0.05)	ST vol: -0.0002 (0.0006)	body mass: 0.004 (0.007)	0
~ ST volume + humeral length	0.02 (0.02)	ST vol: 0.00003 (0.0002)	humerus: -0.0001 (0.0003)	0

Table 2.6. Male subadult sample linear model results (n=16). Bolded predictors are significant at the alpha = 0.05 level; significance codes: ^0.10, *0.05, **0.01, ***0.001.

model	**intercept (95% CI)**	**predictor variable(s) (95% CI)**		**adj. R^2**
ST volume ~ body mass	4.12*** (0.11)	**0.29*** (0.14, 0.44)**		0.51
ST volume ~ humeral length	1.11 (0.92)	**0.71** (0.30, 1.13)**		0.46
$GR_{overall}$				
~ residual ST volume (body mass)	0.0022*** (0.0003)	-0.004^ (-0.01, 0.0007)		0.14
~ residual ST volume (humeral length)	0.0022*** (0.0003)	0.002 (-0.009, 0.001)		0.09
~ ST volume + **body mass**	0.004* (0.0017)	ST vol: -0.00004^ (0.00003)	**body mass: -0.0004** (0.0002)**	0.28
~ ST volume + **humeral length**	0.0007 (0.0016)	ST vol: 0.00004^ (0.00001)	**humerus: -0.00004** (0.000014)**	0.34
$GR_{max\ avg}$				
~ residual ST volume (body mass)	0.008*** (0.002)	0.016 (-0.018, 0.050)		0
~ residual ST volume (humeral length)	0.008*** (0.002)	0.018 (-0.14, 0.050)		0.03
~ ST volume + body mass	0.0025 (0.013)	ST vol: -0.000013 (0.0002)	body mass: 0.0006 (0.0012)	0
~ ST volume + humeral length	0.006 (0.012)	ST vol: 0.00018 (0.00015)	humerus: -0.00011 (0.00011)	0
$GR_{max\ deriv}$				
~ residual ST volume (body mass)	0.0022** (0.0006)	-0.0055 (-0.015, 0.004)		0.08
~ residual ST volume (humeral length)	0.00022** (0.0006)	0.005 (-0.015, 0.005)		0.03
~ ST volume + body mass	0.0066 (0.004)	ST vol: -0.00005 (0.00005)	body mass: 0.0002 (0.0003)	0
~ ST volume + humeral length	0.00005 (0.005)	ST vol: 0.00004 (0.00004)	humerus: -0.00001 (0.00003)	0
GR_{recent}				
~ residual ST volume (body mass)	0.003** (0.001)	0.0011 (-0.010, 0.012)		0
~ residual ST volume (humeral length)	0.003** (0.001)	0.0018 (-0.009, 0.012)		0
~ ST volume + body mass	0.0029 (0.004)	ST vol: -0.00001 (0.00005)	body mass: 0.0002 (0.0004)	0
~ ST volume + humeral length	0.005 (0.004)	ST vol: 0.00001 (0.00005)	humerus: -0.00003 (0.00003)	0

2.4. Discussion

Scaling relationships

The positive relationship between sella turcica volume and both maximum body mass and stature for the full sample and subadult subsample was expected, as larger individuals were predicted to exhibit larger sellae turcicae. Scaling exponents of sella turcica volume against both body mass and stature, however, failed to meet theoretical predictions under models of either geometric similarity or metabolic scaling. The negatively allometric *sella turcica volume ~ body mass* models (all individuals: $b = 0.42$; subadult females: $b = 0.34$; subadult males: $b = 0.29$) and *sella turcica volume ~ humerus* models (all individuals: $b = 1.40$; subadult females: $b = 1.35$; subadult males: $b = 0.71$) revealed that while sella turcica volume does increase as body size increases, it does so at a slower rate than body size. In other words, the sella turcica is smaller than would be expected under models of geometric similarity and metabolic scaling. Interestingly, the full sample (which includes adults) had a larger scaling exponent than the two subadult samples, which suggests sella turcica size becomes larger relative to body size as individuals age.

Growth rate

Analyses of the relationships between growth rate and sella turcica size revealed that sella turcica size is not positively correlated with growth rate. Unexpectedly, in all significant models, sella turcica size was *negatively* correlated with growth rate (i.e., larger sellae turcicae were linked to slower growth rates). Furthermore, the result that sella turcica volume was a significant predictor of growth rate in regression models that

employed the full (i.e., subadult plus adult) sample, but not in models that only used the subadult sample, was also surprising. The relationship between sella turcica volume and growth rate was expected to be stronger for growing individuals (i.e., subadults) than for adults. Indeed, the complete, pooled male and female adult and subadult sample was only analyzed to explore the ability of sella turcica size to predict growth rate for individuals of unknown age and/or sex.

Though sella turcica size was a significant predictor of $GR_{overall}$ in linear models utilizing the full sample, R^2 values were very low (Table 2.4A), which implies weak predictive power and cautions against using sella turcica size to infer growth rate. Though one could attempt to use these models as a tool to predict growth rate from sella turcica size, given the strength of the relationships (and bearing in mind that statistical significance does not always mean biological significance), this would be imprudent. Coupled with the non-significant predictors and low correlation coefficients in subadult the *growth rate ~ sella turcica size* models, these full-sample results suggest that the variation in sella turcica size left unexplained by body size is unlikely to be explained by differences in subadult growth rate.

There are multiple reasons why sella turcica size may be a poor predictor of growth rate. Most simply, the morphology and size of the macaque sella turcica may not be directly linked to changes in growth rate. The proposed connection between sella turcica volume and growth rate relies on many intermediate relationships, each of which has the potential to introduce sources of "error" that may cause the framework upon which the hypothesized sella turcica-growth rate connection was built to break down. Though growth is controlled by the production of pituitary hormones and higher growth

hormone concentrations are linked to increases in growth rate (Ayuk et al., 2004; Vierimaa et al., 2006), the volume of the pituitary may not change in tandem with changes in growth rate in any appreciable manner and/or pituitary size changes may not translate directly into changes in sella turcica size. Hormone production is a dynamic process that adjusts (sometimes rapidly) in response to experienced conditions (Bova et al., 2014; Clemmons, 2004; Monaghan, 2008). Although pituitary size has been shown to reflect growth hormone secretion (e.g., Nagel et al., 1997), the smaller pituitaries in such studies are often the result of pathology (rather than natural fluctuations in size), which makes it plausible that dynamic changes in hormone production do not result in substantial changes in soft tissue pituitary size. Downstream in the growth rate-sella turcica pathway, the lateral aspects of the sella turcica are bounded by the cavernous sinus (Kannan, 1987), which allows modest increases in pituitary size to occur unobstructed. Conversely, it may be that the sella turcica is consistently larger than the pituitary gland to allow space for the pituitary expansion in the event of hormone production during events such as growth spurts. In this scenario, sella turcica volume would not be sensitive to the increases in pituitary size that occur with growth rate increases and would even have the potential to substantially surpass pituitary volume during periods of slower growth (or any other hormone-linked event that is accompanied by decreases in pituitary hormone production). Moreover, any adjustments in hard-tissue morphology that do occur in response to pituitary size (whether linked to changes in growth or not) are likely to experience a lag relative to pituitary changes, as bone remodeling takes approximately 6 months (Clarke, 2008).

Even if sella turcica size can respond somewhat dynamically to changes in growth

rate and/or hormone production, there are further confounding variables linked to the timing of significant growth events. Because the size of the hard-tissue sella turcica is unlikely to decrease should pituitary size decrease, the maximum absolute size of the sella turcica size may represent maximum pituitary size and peak hormone production. In this case, if body growth continues after the peak, sella turcica size relative to body size (the metric used in these analyses) may fluctuate as time elapses. For example, consider a scenario in which maximum absolute sella turcica size is reached as the result of hitting maximum pituitary size during a juvenile growth spurt. Relative sella turcica size is by definition not a constant, so sella turcica size relative to body size will vary depending on the magnitude of the body size used to calculate it. If body growth continues for some time period after maximum absolute sella turcica size is achieved, the magnitude of sella turcica size relative to body size will differ based on how much additional body growth occurs between the time of maximum sella turcica size and the time body size is measured. In practice, this means that an individual who died soon after this peak in growth will likely have a larger sella turcica relative to body mass than an individual who survived to continue growing after this peak and died years later (e.g., an adult), despite the fact that both individuals experienced the same maximum growth rate and had same maximum absolute sella turcica size. Furthermore, following periods of peak hormone production and peak growth, increases in body mass or stature may outpace increases in sella turcica size, which would result in a smaller-than-expected sella turcica for body size and temporal shift in sella turcica size relative to body size.

Any analysis of growth is also sensitive to the metrics of growth that are used. While the theory underpinning growth rate calculations was considered carefully, the

precision and accuracy of growth rate estimates were limited by the longitudinal body mass data available. Ideally, it would have been possible to model more accurate growth curves for the study individuals to compute higher quality instantaneous growth rates. Ultimately, though, given the likely dissociation between growth rate and sella turcica size, it seems unlikely that using different growth rate metrics would have had a meaningful impact on overall interpretations and conclusions.

Nonetheless, keeping these caveats in mind, deeper explorations into the nuances of the relationship between subadult sella turcica size and growth rate revealed some complex, yet potentially intriguing, relationships. The largely non-significant results among subadult females, for example, may be the result of a nonlinear relationship between $GR_{overall}$ and sella turcica size, where sella turcica size is positively correlated with growth rate above a $GR_{overall}$ of about 0.0025 g/day, but negatively correlated with growth rate below this threshold. Further subdividing the female dataset into "$>$0.0025 g/day growth rate" and "$<$0.0025 g/day growth rate" cohorts provides some statistical support for this hypothesis. Analyses reveal that among female subadults with a growth rate above 0.0025 g/day, $GR_{overall}$ is positively correlated with sella turcica size (Table 2.7). The relationship between $GR_{overall}$ and sella turcica volume for growth rates below the 0.0025 g/day threshold is not statistically significant (Table 2.7), but this may be a product of small sample size. Further exploration (e.g., power tests) and testing with a larger sample would be worthwhile to confirm the physiological significance of this

Table 2.7. Female subadult growth rate subsamples based on growth rate.
Significant predictor variables at the alpha = 0.05 level are bolded.

model	intercept p-value	predictor variable(s) p-value		adj. R^2
$GR_{overall}$ > 0.0025 g/day				
~ residual ST volume (body mass)	0.0036 (p < 0.001)	**0.0030 (p = 0.014)**		0.31
~ residual ST volume (humeral length)	0.0034 (p < 0.001)	**0.0035 (p = 0.006)**		0.38
~ ST volume + body mass	0.0014 (p = 0.082)	**ST vol: 0.00023 (p = 0.011)**	mass: -0.0001 (p = 0.19)	0.33
~ ST volume + humeral length	0.0031 (p = 0.005)	**ST vol: 0.0003 (p = 0.003)**	**humerus: -0.0002 (p = 0.04)**	0.45
$GR_{overall}$ < 0.0025 g/day				
~ residual ST volume (body mass)	0.0015 (p < 0.001)	-0.0012 (p = 0.20)		0.05
~ residual ST volume (humeral length)	0.0016 (p < 0.001)	-0.0013 (p = 0.20)		0.05
~ ST volume + body mass	0.0022 (p = 0.009)	ST vol: -0.00001 (p = 0.18)	mass: 0.00011 (p = 0.44)	0.001
~ ST volume + humeral length	-0.001	ST vol: -0.00001 (p = 0.10)	humerus: 0.00003 (p = 0.14)	0.07

hypothesis, particularly because significant or approaching significant results may simply be artifacts of the sample used. For example, the 0.0025 g/day growth rate cut-off was not chosen for any underlying biological, physiological, or endocrinological reason; it was simply selected because it was the point at which a divergence appeared in a plot of the data. If scenarios of divergent growth trajectories around a specific growth rate are to be pursued further, it would be worthwhile to generate *a priori*, biologically informed hypotheses about growth rates that may serve as breakpoints.

Caution is urged in the event that sella turcica size as a predictor of growth rate, as it may be that the significant correlations between growth rate and sella turcica size are spurious. Nonetheless, there are some biological explanations for the few significant, negative correlations that may also shed light on the potentially divergent patterns in the female subsample. The pituitary gland secretes an array of hormones beyond those

implicated in somatic growth; the anterior pituitary produces and secretes prolactin, luteinizing hormone, follicle stimulating hormone, adrenocorticotropic hormone and thyroid stimulating hormone, while the pituitary secretes vasopressin and oxytocin that are produced by the hypothalamus (Stojilkovic, 2018; Van Tol et al., 1988). The tendency for growth rate to decrease as sella turcica size increases in the full sample and among slow-growing subadult females therefore may be linked to the production and/or secretion of other pituitary hormones (associated with the anterior pituitary, posterior pituitary, or both) that do not explicitly promote growth. Assuming a positive relationship between hormone production and pituitary size, increases in the production of these hormones could result in an increase in whole pituitary size that happens to correspond with a decrease in growth rate. Depending on the age of an individual, this decrease in growth rate and growth hormone production could be the result of normal maturation processes or could be a response to challenges and stressors, as both scenarios can be linked to pituitary hormones.

At maturation, the anterior pituitary produces gonadotropins (follicle-stimulating hormone and luteinizing hormone) that target the ovaries and testes (Marques et al., 2018). Life-history theory predicts that maturation signals a shift in energy allocation away from growth towards reproduction (Hill & Kaplan, 1999), meaning that increases in the production of gonadotropins as individuals approach sexual maturation would correspond with a tapering off in growth and the production of growth hormone. For this shift to result in an appreciable increase in pituitary or sella turcica size, however, the magnitude of increase in gonadotropin production would have to surpass the magnitude of decrease in growth hormone production. If supported, this hypothesis could also help

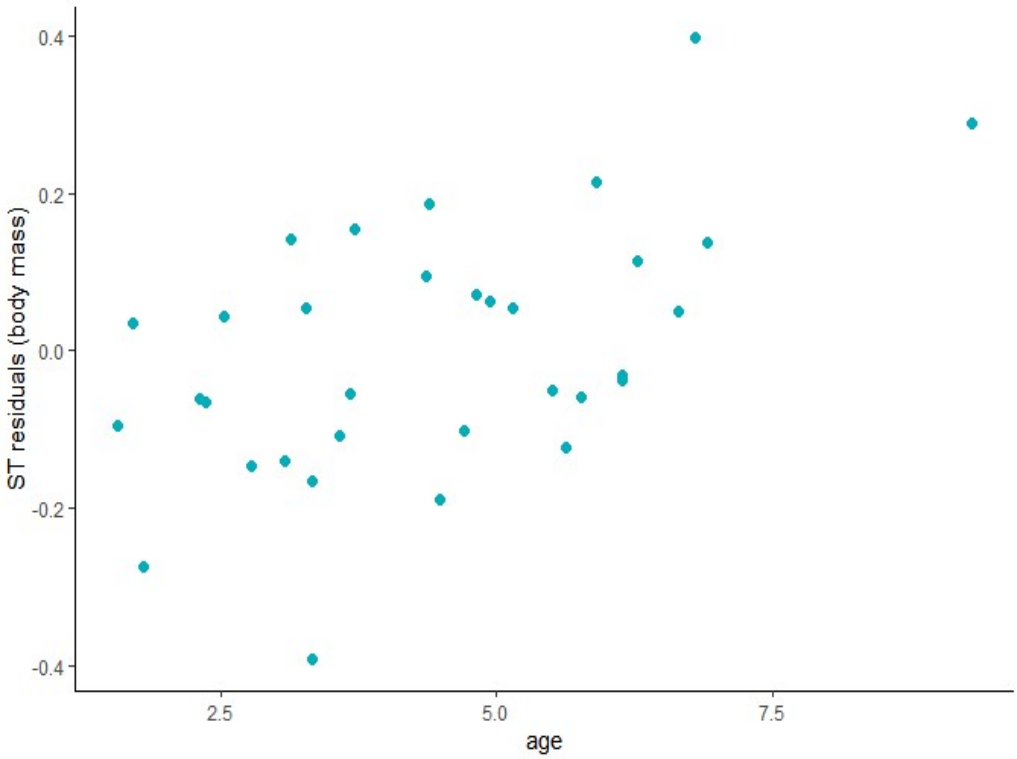


Figure 2.7. Residuals of sella turcica (ST) volume against body mass plotted against age for the female subadult sample.

Table 2.8. Linear model of residual sella turcica volume against age at death. Both the predictor variable (age at death) and intercept of the model are significant.

model	**intercept**	**predictor**	**adj. R^2**
residual ST volume (body mass) ~ age at death	**-0.207 (p = 0.003)**	**0.047 (p = 0.001)**	0.25

explain why older individuals tend to have relatively larger sellae turcicae than younger individuals. Among subadult females, for example, there is a positive relationship between body mass-adjusted sella turcica size (i.e., residuals) and age (Figure 2.7; Table 2.8). In other words, older individuals have larger sellae turcicae than predicted by their body mass, while younger individuals have smaller sellae turcicae than predicted by their body mass. Of course, this hypothesis requires sella turcica size to be a faithful reflection of pituitary size, but increases in gonadotropin production could underpin the association

between larger sella turcica size and slower growth, as well as the observation that older subadult females have relatively larger sellae turcicae than younger subadult females.

The anterior pituitary gland also produces adrenocorticotropic hormone (sometimes called cortiocotropin), which stimulates the adrenal glands to produce cortisol and is an important component of the hormonal stress response axis (Marques et al., 2018; Saffran & Schally, 1955; Smith & Vale, 2006). Therefore, stress-related pituitary hormones may play an important role in observed relationships between growth rate and sella turcica size. A stressful environment and adverse conditions are linked to increases in stress hormone production (Beehner & Bergman, 2017; Novak et al., 2013; Thayer et al., 2018). Increases in glucocorticoids (stress hormones) such as cortisol have been traditionally linked to decreased growth in mammals (Achermann & Jameson, 2010; Bellamy & Leonard, 1964; Lesage et al., 2001), although increasing evidence conversely links glucocorticoid levels to accelerated growth (Berghänel et al., 2017; Dantzer et al., 2013). Increased growth under more stressful conditions are likely linked to maternal stress responses and hormonal cues that are transmitted to offspring (Dantzer et al., 2013), so further research is necessary to tease out the relative effects of maternal cues and individual experiences on growth responses to stress.

If, however, an individual's stress hormone production is consistently linked to decreased growth, high enough concentrations of stress hormones (coupled with a large enough decrease in growth hormone production) may drive increases in pituitary size that correspond to decreased growth rate. If the slow growth in the female subadult sample is driven by increased exposure to challenges and stressors, this could potentially help to explain the negative correlation between growth rate and sella turcica size in slow-

growing individuals. Under favorable conditions, reduced stress hormone production would correspond to faster growth and increased growth hormone production. Conversely, if responses to stress in the Cayo Santiago population drive an increase in growth rates (perhaps due to maternal cues), faster growth would correspond both increased stress hormone and increased growth hormone production, which would both contribute to larger pituitary (and sella turcica) size in fast-growing individuals (for example, female subadults growing over 0.0025g/day). This scenario—the co-occurrence of both increased corticotropin and increased growth hormone to promote faster growth under stressful conditions—does not, however, provide an easy explanation for the relatively larger sellae turcicae found in slower-growing female subadults. It also does not explain the negative correlation between sella turcica size and growth rate across all individuals.

Though hypotheses linking stress to patterns of variation in growth rate found in this study are compelling, it is difficult to test them without a means to assay hormone levels ontogenetically and without understanding the specific trade-offs between growth hormone and corticotropin production in the Cayo Santiago macaque population. It is also important to investigate how levels of these two hormones interact to produce differences (if any) in pituitary and sella turcica size. Because it seems that physiological stress responses may vary slightly depending on how cues are received (i.e., maternally or from an individuals' experiences), understanding the mechanistic links between stress and growth is essential.

The anterior pituitary further produces a number of hormones beyond the gonadotropins, cortiocotropin, and growth hormone mentioned here, while the posterior

pituitary secretes two additional hormones, so it is probable that all pituitary hormones have some effect on pituitary size, thereby obscuring any potentially simple relationships between sella turcica size and any one hormone (e.g., growth hormone) or phenotype (e.g., growth rate). Though the posterior pituitary is much smaller than the anterior pituitary (Cheung & Camper, 2018) and does not produce the hormones that it secretes (oxytocin and vasopressin) itself (Baylis, 1983), it is entirely possible that potential hormone secretion-linked changes in posterior pituitary size affect whole pituitary size and thus, sella turcica size. The specific contributions of each pituitary hormone to pituitary and/or sella turcica size within the Cayo Santiago population, however, are impossible to know without directly studying hormone levels, which may be a valuable avenue for future research. Many of the scenarios outlined above are not mutually exclusive and the complex interpretations required to make sense of these results underscore the importance of understanding the proximate mechanisms that underpin growth and development. As hormone levels fluctuate in response to experienced environment and developmental conditions (Monaghan, 2008), accurately predicting hormone-linked phenotypes is difficult without considering the external influences on hormone production and their interactions.

Ultimately, though hormone-balance hypotheses are exciting to consider, dynamic changes in hormone production and/or pituitary size are likely not reflected in the more static, hard-tissue morphology of the sella turcica. Therefore, sella turcica size is likely too crude of a measure to provide meaningful information about intraspecific growth in skeletal samples. Though the significant negative correlation between sella turcica size and overall growth rate in the mixed adult-subadult sample could be used to make

inferences about growth rate differences between individuals, the model has low predictive power and should be used with caution, if at all. Given the complex, sometimes interrelated, signaling pathways of pituitary hormones and the probable unresponsiveness of sella turcica morphology, any growth rate signal in this population is unlikely to outweigh the noise. It is thus suspected that the significant growth rate-sella turcica correlations observed here may be spurious without any directly interpretable underlying biological significance, particularly as sample sizes were relatively small (<50 individuals) and a myriad other factors not investigated here (e.g., social rank, genetics, pedigree, food quantity and/or quality) may also affect growth rate. The lack of significant intraspecific relationships, however, does not preclude the possibility that predictable patterns between growth rate and sella turcica size emerge at different scales of analysis. A robust interspecific link between sella turcica size and growth rate, for example, is not predicated upon the existence of strong intraspecific, ontogenetic relationships that were hypothesized here; the strength and direction of interspecific correlations between sella turcica size and growth rate are explored in Chapter 4.

CHAPTER 3

LINKING ENVIRONMENTAL CONDITIONS TO INTRASPECIFIC GROWTH RATE VARIATION

3.1. Introduction

Within a species, individuals exhibit differences in phenotypic traits, including life-history traits such as growth rate. As variation in both life history and growth can be linked to differential fitness and success (e.g., Altmann & Alberts, 2005; Blomquist, 2009; Charnov et al., 2007; Charnov & Berrigan, 1993; Hayward et al., 2013; Kramer, 2008; S. D. Lee et al., 2019; W.-S. Lee et al., 2012; Mangel & Stamps, 2001; Migliano et al., 2007; Quesnel et al., 2018; Rollo, 2002; Stearns, 1989), understanding how and why intraspecific variation in traits such as growth emerges is relevant for understanding macroevolutionary trends and can shed light on the evolution of primate life histories. Between-population or interspecific differences in growth and life history are largely linked to evolutionary selective pressures (e.g., mortality risk) and the tradeoffs between energy devoted to growth and energy devoted to reproduction (Stearns, 1992). While intraspecific variation in growth and life-history traits also likely maximizes fitness benefits as a result of these tradeoffs in energy allocation, they are proximally determined by mechanisms that are the result of the interplay between genetics and local environment.

Some intraspecific variation is certainly the direct result of genetic variation (e.g., Baker et al., 1993; Williams-Blangero & Blangero, 1995), but mechanisms such as developmental plasticity allow trait expression and phenotypic outcomes to vary within a

population in response to local environmental conditions (Forsman, 2015; Fusco & Minelli, 2010; Moczek et al., 2011; Pfennig et al., 2010; Wells & Johnstone, 2017; West-Eberhard, 2003, 2005). Developmental plasticity is the potential for genetically similar individuals to express different phenotypes depending on conditions experienced during early development (Monaghan, 2008). It has been proposed to provide individuals with a mechanism express different phenotypes under different environmental regimes, thereby allowing them to optimize fitness without requiring different genotypes (Pfennig et al., 2010). Although the mechanisms by which environmentally induced phenotypic differences can be translated into heritable differences has long been debated (e.g., Braendle & Flatt, 2006; Waddington, 1956, 1959; West-Eberhard, 2003), it has also been hypothesized to facilitate and promote the evolution of novel traits (e.g., Moczek et al., 2011; Pfennig et al., 2010; West-Eberhard, 2005; Wund, 2012). Plasticity is greatest early in life (Wells & Johnstone, 2017), with both prenatal (e.g., Schneider et al., 1999) and postnatal (i.e., post-birth and pre-weaning) growth sensitive to experienced environment (e.g., Altmann & Alberts, 2005). Though it is clearly difficult to disentangle the effects of developmental plasticity and underlying genetic variation in a population without knowledge of individuals' genotypes, some scholars argue that intraspecific phenotypic variation, particularly with regard to aspects of development and reproduction, can serve as a proxy for population-level developmental plasticity (Forsman, 2015; Lee & Kappeler, 2003).

Within populations, the availability and quality of food resources are major driving forces behind the pace of growth (Jarrett et al., 2020; Lesage et al., 2001; Macho, 2017; Ramirez Rozzi et al., 2015; Strum, 1991), with better nutrition and higher quality

diets linked to faster growth across mammalian taxa (e.g., Drago et al., 2010; Nagy & Negus, 1993; Walkden-Brown et al., 1994) and within Primates (e.g., Altmann & Alberts, 2005; Borries et al., 2011; Watanabe et al., 1992). Differences in growth rate not only affect body mass and/or the time it takes to obtain that mass but can further impact downstream life-history traits. Altmnan and Alberts (2005), for example, found that food-enhanced baboons in a wild *Papio cynocephalus* population exhibited faster growth and reached sexual maturity earlier than naturally foraging baboons. Because slower growth reduces metabolic costs, an intraspecific application of the ERAH suggests that the ability to dynamically alter growth to match local conditions may improve survival and be an effective way to address uncertainty in environment or resource acquisition (Lee & Kappeler, 2003). Indeed, low resource availability and seasonal variation is linked to slower growth and smaller body size in a range of wild primate populations (Jarrett et al., 2020; Strum, 1991).

Growth and developmental trajectories are sensitive to social environment as well as to physical environment. Early in life, these social factors include maternal rank, maternal investment, and social bond strength. In hierarchical primate populations, growth and development are also affected by maternal rank via increased stress in low-ranking individuals (and their offspring) as well as preferential access to resources by those of higher rank (Altmann & Alberts, 2005; Onyango et al., 2008). In baboons, postnatal growth rates (Altmann and Alberts, 2005) and juvenile success (Lea et al., 2016; Lee et al., 2019) are sensitive to both environmental conditions and maternal resources, which are largely linked to maternal rank (Altmann & Alberts, 2005; Lee et al., 2019). Furthermore, challenging early life conditions have consequences for

reproductive fitness (Rödel et al., 2009) and lifespan (Tung et al., 2016). Importantly, the lasting impact of early life environment has implications beyond growth, development, and life history. There is increasing evidence that early life conditions can program later life responses to stressors (Anacker et al., 2014; Thayer et al., 2018); in humans, these early life challenges are further linked to health disparities (Bush et al., 2016).

Many investigations of growth (especially in wild or semi-wild populations) rely on cross-sectional sampling to build population-level models of growth trajectories. (e.g., Altmann & Alberts, 2005; Dean & Wood, 1981; Leigh, 1996; Turner et al., 2018; Whitten & Turner, 2009). The Cayo Santiago *Macaca mulatta* population, however, presents an opportunity to pair cross-sectional trends with individuals' growth trajectories and link local ecological and social conditions to individual variation. This chapter will therefore explore how the conditions experienced during both the prenatal and early postnatal phases of development affect individual growth rates and growth outcomes, such as age-specific and adult body size. While past work has mapped growth and developmental trajectories to general assessments of environmental quality, this project seeks to further decompose the physical environment into specific environmental variables relevant to a tropical population (e.g., dryness, precipitation level, and temperature). It additionally aims to explore how growth rates and the length of the growth period interact to produce variation in body size under different conditions.

Accepting that intraspecific phenotypic variation is a proxy for population-level developmental plasticity (Forsman, 2015; Lee & Kappeler, 2003), this project will link plasticity in growth rate and body size to early-life environment by testing two specific hypotheses: (a) within the Cayo *M. mulatta* population, individual growth rates respond

to experienced environment; (b) more favorable early-life conditions are linked to larger body mass for a given age, while poor or challenging conditions (e.g., high temperature, environmental stresses such as drought or hurricanes, low maternal rank) are linked to smaller body size. This approach will not directly explore how intra-individual changes in growth across ontogeny are linked to developmental conditions, but will instead assess the population-level potential for growth variation in response to environmental conditions. This component of the project initially proposed to investigate whether sella turcica size, as a proxy for growth rate, also exhibits evidence of developmental plasticity and varies in response to environmental conditions. Although the size of the sella turcica may vary in response to environmentally driven pituitary hormone fluctuations not linked to growth, because there was not a strong relationship between growth rate and sella turcica size (Chapter 2), this project will not explore the relationship between relative sella turcica size and environment.

3.2. Materials and Methods

Study population

Subadults (age < 8 years) within the Cayo Santiago *Macaca mulatta* sample were used to test the relationship between environmental conditions and growth (see Chapter 2, section 2.2 for an in-depth description of the study population). As a result of body-size dimorphism and thus differences in male and female growth trajectories, subadult samples were analyzed separately by sex. Female subadults were further subdivided into "high" and "low" categories for each environmental parameter; environmental parameters and binning criteria are discussed below in the *Physical and social*

environment data section. Both body mass and stature (approximated using humeral length as a proxy; see Chapter 2, section 2.2 for measurement protocols) were used as two different, yet correlated, measures of body size.

Physical and social environment data

Macaca mulatta births are seasonal, but because dates of birth can vary by up to 6 months within a cohort (Widdig et al., 2016), some variation in experienced environment is expected within each birth cohort. Instead of relying on solely on birth cohort as a proxy for potential seasonal and/or environmental differences, this project utilizes metrics specific to each individual, which serves the purpose of more directly measuring an individual's experienced environment. Daily maximum temperature ("temp"), daily evaporation ("evap"; a dryness metric measured as the mm of evaporated water as determined experimentally), and daily precipitation ("precip") data were obtained from the NOAA's Climate Data Online repository (https://www.ncdc.noaa.gov/cdo-web/) using the NOAA weather station closest to Cayo Santiago, located in Gurabao, Puerto Rico. Correlation matrices suggest that daily environment data were not strongly multicollinear, as correlation coefficients between pairs of variables are <0.65 (Figure 3.1), and variance inflation factors (VIF) confirm that the three metrics can be used as independent variables. VIFs identify the strengths of any correlations between variables and can vary from 1 to infinity; a VIF of 1 represents no correlation between the variable and any other variable (Taylor et al., 2007). VIFs for the three environmental variables and age were well below the commonly accepted critical value of 5 (Table 3.1), which

means that multicollinearity between variables is not strong enough to invalidate statistical assumptions of independence.

Table 3.1. Variance inflation factors

time period	temp	precip	evap	age
prenatal	2.02	1.24	1.72	1.13
1st year	2.33	1.41	1.61	1.35

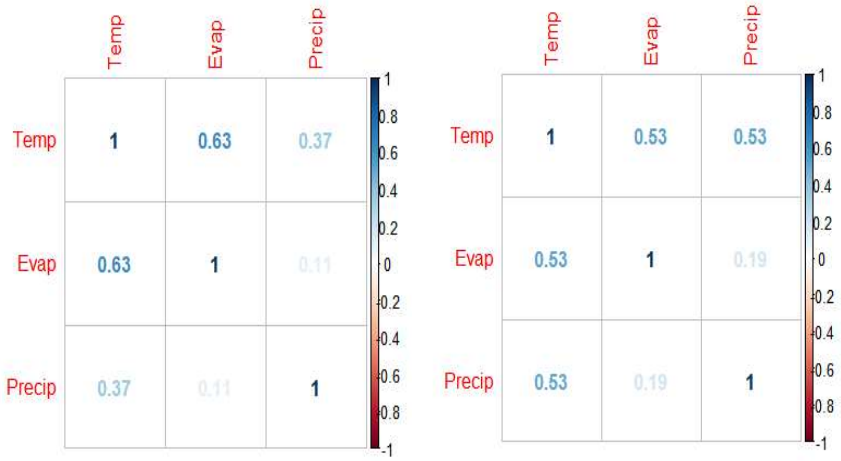


Figure 3.1. Correlation matrix between environmental variables experienced during gestation (left) and the first year of life (right). Values in cells are Pearson's correlations coefficients.

For each individual, the daily high temperature, precipitation total, and daily evaporation during the prenatal period and the first year of life were analyzed[4]. The prenatal period was defined as gestation length, which is 166 days for *M. mulatta* (Silk et al., 1993); the first year of life encompasses the first 365 days after birth. While these three environmental variables (temperature, precipitation, and evaporation) are related, all

[4] Conditions during the first, second, and third trimesters were also analyzed separately. These had no individual effects on growth so are not presented. Similarly, conditions during only the weaning period (first 180 days post-birth) were not significant predictors of growth, so only first year conditions are presented.

three were included as all capture slightly different aspects of primary productivity. Precipitation, for example, measures the total amount of rainfall, while evaporation is a measure of dryness, so it is possible to envision a scenario in which individuals experience relatively dry conditions (indicated by high evaporation) that are accompanied by bouts of high precipitation (that may then quickly evaporate). Conversely, it would also be possible for individuals to experience low evaporation (i.e., high humidity) that is accompanied by high precipitation. Admittedly, the Cayo Santiago macaques live on a tropical island that likely does not experience major temperature fluctuations, though it does experience rainfall extremes (Keellings & Hernández Ayala, 2019). Nonetheless, all three environmental variables were included in models since the more comprehensive *temperature* + *environmental* + *evaporation* model may prove valuable and informative in the event that comparisons are made to primate populations inhabiting more temperate or seasonal climates. To summarize experienced environment data for each individual, daily temperature data and daily evaporation data were averaged over each period (i.e., prenatal versus postnatal); daily precipitation data were summed to measure the total precipitation during each period.

The environmental data were used as continuous variables as well as to establish categorical "high" and "low" bins that represent differences in experienced environment. Cut-offs for environment bins were determined by examining the distribution of temperature, precipitation, and evaporation values during both the prenatal period and first year; conditions in the upper 40% were classified as "high", while conditions in the lower 40% were classified as "low". For example, an individual who experienced temperatures in the upper temperature range and precipitation in the lower precipitation

range was given a “high” temperature classification and a “low” precipitation classification. Due to the small size of the male subadult sample, only female subadults were placed into environment bins.

Dominance rank influences growth and outcomes in many mammals, including primates (e.g., Altmann et al., 2010; Altmann & Alberts, 2005; Bercovitch & Strum, 1993; Holekamp, 2019; Onyango et al., 2008; Setchell et al., 2006), and in the Cayo macaques, rank is linked to maternal rank and matriline (Missakian, 1972). Maternal rank data are not collected by the Caribbean Primate Research Center (CPRC) as part of their standard demographic census, but affiliated scientists have amassed such data over the years. Decades of maternal rank data collected by Donald S. Sade and John D. Berard were collated and synthesized by Greg Blomquist. Maternal matriline rank was not available for all individuals included in this study, so the effects of rank on growth were only analyzed for a subset of 28 individuals. Similarly, as there may be a modest genetic component to some life-history traits (e.g., age at first reproduction) within the Cayo Santiago population (Blomquist, 2012) it would have been prudent to explore the effects of matriline on growth. Unfortunately, however, matriline was only available for 18 individuals in the sample, so matriline data were not included in analyses.

Modeling growth

Environmental effects on growth are often assessed by examining differences in stature-at-age or mass-at-age between two or more conditions (Altmann & Alberts, 2005; Jarrett et al., 2020). This method relies on the fact that size is a function of growth rate and time (i.e., larger individuals for a given age achieve their larger size through faster

growth) to model cohort-level growth for each condition-based subpopulation. While there are multiple mechanistic growth curve functions that can be fit to body growth data, von Bertalanffy, Gompertz (sigmoidal), and logistic curves are the most common (Zullinger et al., 1984). The main functional difference between these models is the point at which each assumes the inflection point (where acceleration in growth changes from positive to negative) occurs (Zullinger et al., 1984). A recent analysis of growth in a mixed wild-captive vervet population confirmed that von Bertalanffy models outperformed the other commonly used growth curve models (Jarrett et al. 2020), so the von Bertalanffy model was used here.

First, von Bertalanffy growth curves were fit for humeral length and body mass in the full male and female samples as well as the male and female subadult samples. Then, to explore whether differences in growth trends between groups experiencing different climatic conditions, von Bertalanffy models were also fit for female subadults in "high" and "low" groups for each environmental parameter. The male subadult sample was too small to reliably fit convergent growth curves for environment-specific subsamples. The von Bertalanffy function models body growth as a function of time and is calculate as

$$L_t = L_\infty\left(1 - e^{-k(t-t_0)}\right)$$

where L_t is the average length at time (i.e., age) t, L_∞ is the asymptotic average length, k is the growth rate coefficient (units yr^{-1}), and t_0 is the inflection point of the growth curve. Humeral length was used as a proxy for body length. Transforming the length-based equation for use with body mass (W) yields

$$W_t = W_\infty\left(1 - \frac{1}{3}e^{-k(t-t_0)}\right)^3$$

In light of using somewhat small samples to calculate growth curve parameters, alternative methods to explore differences in mass-at-age and stature-at-age were also employed. Individuals were placed into one of two age bins: young subadults (aged under 1500 days) or old subadults (aged 1501-3000 days). Differences in body mass and humeral length between environmental condition cohorts (e.g., high or low for each environmental variable) were investigated for each of the two subadult age categories. This method could be considered to be more conservative for exploring differences in mass-at-age and stature-at-age, as there are body size differences between the youngest and oldest individuals in each age group that may obscure environmentally driven trends in body size. This method could, however, be very sensitive to under-sampled ages; for example, there could be a situation where all young (i.e., very small) individuals fall into one environmental bin and all older (i.e., larger) individuals fall into the other bin. Pairwise comparison results were thus confirmed visually using growth curves. Ultimately, this is not a perfect method, but simply another way to holistically evaluate environment-linked differences in growth.

Generalized linear models with age, continuous environmental data, and maternal rank/matriline (if available) as predictor variables were also used to analyze the relative effects of environment and age on two measures of attained body size, humeral length and body mass. These metrics were included because although humeral length and body mass are likely linked to growth rate, they capture slightly different aspects of growth than growth rate. Unlike calculations of growth rate, they do not depend on repeated body mass measurements throughout life and may not be subject to the same measurement

biases as growth rate; they may thus reveal environmental signals that growth rate cannot detect.

These analyses test for differences in growth outcomes, but larger body size can be achieved by growing faster, growing for longer, or some combination of the two. When the duration of growth is equal within a population, larger individuals are expected to exhibit faster growth rates. In practice, individuals may not all have the same growth period, and larger size may be achieved by growing at the same rate for a longer length of time, or by growing faster for a shorter length of time. This can be assessed at the cohort level by calculating maximum growth rate from the k parameter of a growth curve function and calculating when growth begins to level off as asymptotic size is reached. Due to wide 95% confidence intervals for the von Bertalanffy growth curve parameters calculated here, general trends were visually inferred from population-level growth curves.

To explore the effect of environment on growth without using achieved mass and stature indicators of growth rate, the approach employed here was to directly evaluate the correlation between environmental conditions and growth rate metrics in subadults. Doing so, however, requires caution and careful consideration of the growth rate metrics used. Because growth is non-linear and individuals may experience growth spurts or periods of slower growth, one might expect measures such as maximum growth rate (calculated using either linear models or derivatives; Chapter 2, section 2.2) to vary depending on age. Age was therefore included as a covariate in models of maximum growth rate. The average change in mass over time may also be age-dependent. For example, though the timing and duration of a growth spurt would be obscured by fitting a

linear function to body mass data that bookend a non-linear growth spurt, the presence of a growth spurt would result in a faster average growth rate. Younger individuals who may not have reached their growth spurt, or older individuals who may have experienced periods of slow growth following a growth spurt would thus be expected to have slower average growth over time than an individual who recently completed a growth spurt. Because of this, subadult ages were included as covariates in models of overall growth rate. In light of the growth curve analyses and inferences drawn about the quality of each growth metric (Chapter 2), instantaneous (i.e., derivative-calculated) growth rates were not used and only overall growth rate ($GR_{overall}$) and maximum average (linear) growth rate ($GR_{max\ ave}$) were investigated for environmental effects. Though the von Bertalanffy growth curves that were fit to population-level data are more sophisticated models of growth than the $GR_{overall}$ and $GR_{max\ ave}$ calculations, von Bertalanffy curves were not fit to each individual's longitudinal body mass data.

Finally, if differences in body size outcomes are the result of differences in growth rate, rather than duration of growth, we might expect growth rate to be a predictor of body size (although age is expected to be the strongest predictor of body size). Setting aside environmental effects as the mediator of differences in growth rate, body size was analyzed as a function of overall and maximum growth rate, with age as a covariate. Because maximum body mass was used to calculate $GR_{overall}$, body mass was only modeled as a function of $GR_{max\ avg}$ and age. Humeral length was modeled as a function of age and both $GR_{overall}$ and $GR_{max\ avg}$. Potential confounding effects due to the relationship between measures of body size and growth rate are addressed in the discussion.

Analytical methods

All analyses were performed in R version 4.0.2 (R Core Team, 2020). Body size metrics (humeral length and body mass) were log-transformed to meet statistical assumptions. As stature and body mass can vary independently of one another, with body mass a stronger indicator of body condition, both mass and humeral length were modeled for all body size analyses. The effects of growth rate on achieved body mass and humeral length were assessed using generalized linear models with growth rate (either $GR_{overall}$ and $GR_{max\ avg}$) and age as predictor variables. Though standardization of variables is not necessary for linear regression, variables were standardized to permit comparison of standardized beta coefficients within models.

The effects of environment on body size and individual growth rates ($GR_{overall}$ and $GR_{max\ avg}$) were tested through multiple generalized linear models (GLMs) with age and environmental variables during either the prenatal period or first year of life as predictors; when available, maternal rank was also included as a predictor. Matriline data were not included due to missing data and resultant small sample size. Because lower ranking individuals were poorly sampled, it could be argued that maternal rank should be considered a categorical random effect in a generalized linear mixed model (Gelman & Hill, 2006)[5], but maternal rank was here included as a co-predictor in linear models following methods in the primate literature (e.g., Murray et al., 2006; Petrullo et al.,

[5] Some statisticians, (e.g., Gelman & Hill, 2006), argue that when certain groups (in this case, ranks) are poorly represented in the data, treating the categorical variable as a random effect in a generalized linear model is preferred because its coefficient will be estimated using partial pooling technique. Shrinkage estimators like partial pooling partially base each group's effect (i.e., coefficient) estimate on the more abundant data from other groups, which can mitigate the risk of obtaining poor estimates for groups that are poorly represented in the data (Gelman & Hill, 2006).

2019). Given sex differences in growth trajectory, males and females were analyzed separately. Two sets of models were run: (1) generalized linear models with the full sample, but no rank data, and (2) generalized liner models with only individuals for whom maternal rank data were available. Akaike's Information Criterion, with a correction for small sample size (AICc), was used to determine the best fit models and to evaluate which predictor variables explain the most variation in growth rate and the two measures of body size (body mass and humeral length). The best-fitting models have the lowest AICc scores. To avoid too stringently limiting models based on ΔAICc values alone (Grueber et al., 2011) and to allow the reader access to all significant models, all models with significant predictors were reported here.

Von Bertalanffy growth curves were fit to body mass and humeral length for the full male population, full female population, male subadult population, and female subadult population the *minpack.lm*, and *FSA* packages in R. 5000 bootstrap iterations were used to generate 95% confidence intervals for all growth function parameters. Only female subadults were further subdivided by environmental conditions ("high" or "low") to compare variation in growth curve parameters in relation to environment. For each environmental parameter (evaporation, precipitation, and temperature) during each developmental period (prenatal or first year), "high" condition females and "low" condition females were modeled separately using the same protocol used for the full samples. Differences in achieved body mass and humeral length between "high" and "low" prenatal and first year environmental variable cohorts were tested for young subadult females (<1500 days) and older females (1501-3000 days) using Kruskal-Wallis rank sum tests (preferred over Mann-Whitney U tests due to tied ranks).

3.3. Results

When effects of environment were not considered, age was (as expected) the best predictor of both body mass and humeral length for male and female subadults, as indicated by significant beta coefficients for age but not for growth rate (Table 3.2). Neither overall growth rate ($GR_{overall}$) nor maximum average growth rate ($GR_{max\ avg}$) was a significant predictor of body mass or humeral length for females (Table 3.2), although $GR_{overall}$ was a nearly significant predictor of humeral length in the male sample (Table 3.2). Population-level male and female loess-fitted growth curves are presented in Figure 3.2.

Table 3.2. Models of growth rate and age as predictors of body size. Significance codes: ^0.1; *0.05; **0.01; ***0.001; NS = not significant.

		standardized beta			
	model	growth rate	age	R^2	p-value
female	mass ~ GR_{max} + age	NS	0.87***	0.73	<0.001
	humerus ~ $GR_{overall}$ + age	NS	0.87***	0.69	<0.001
	humerus ~ GR_{max} + age	NS	0.85***	0.69	<0.001
male	mass ~ GR_{max} + age	NS	0.88***	0.77	<0.001
	humerus ~ $GR_{overall}$ + age	0.21^	0.81***	0.85	<0.001
	humerus ~ GR_{max} + age	NS	0.92***	0.82	<0.001

Results of linear models testing the correlations between environmental conditions and both body size (mass and humeral length) and growth rate are presented in in Tables 3.3 (females) and 3.4 (males). (Models in which no independent variables were significant predictors of body size or growth rate are not reported in the tables.) As maternal rank was only a significant predictor in very few GLMs of body mass using the female subadult subsample, these results can be found in Appendix B (Table S3.1). In all

environmental models of body size, age was again the best predictor of both female (Table 3.3) and male (Table 3.4) body mass and humeral length, as indicated in Tables 3.3 and 3.4. Adding growth rate to the model did not improve the fit of models with body mass or humeral length as the dependent variable, so growth rate was not included as a predictor of body size in the models presented in Tables 3.3 and 3.4.

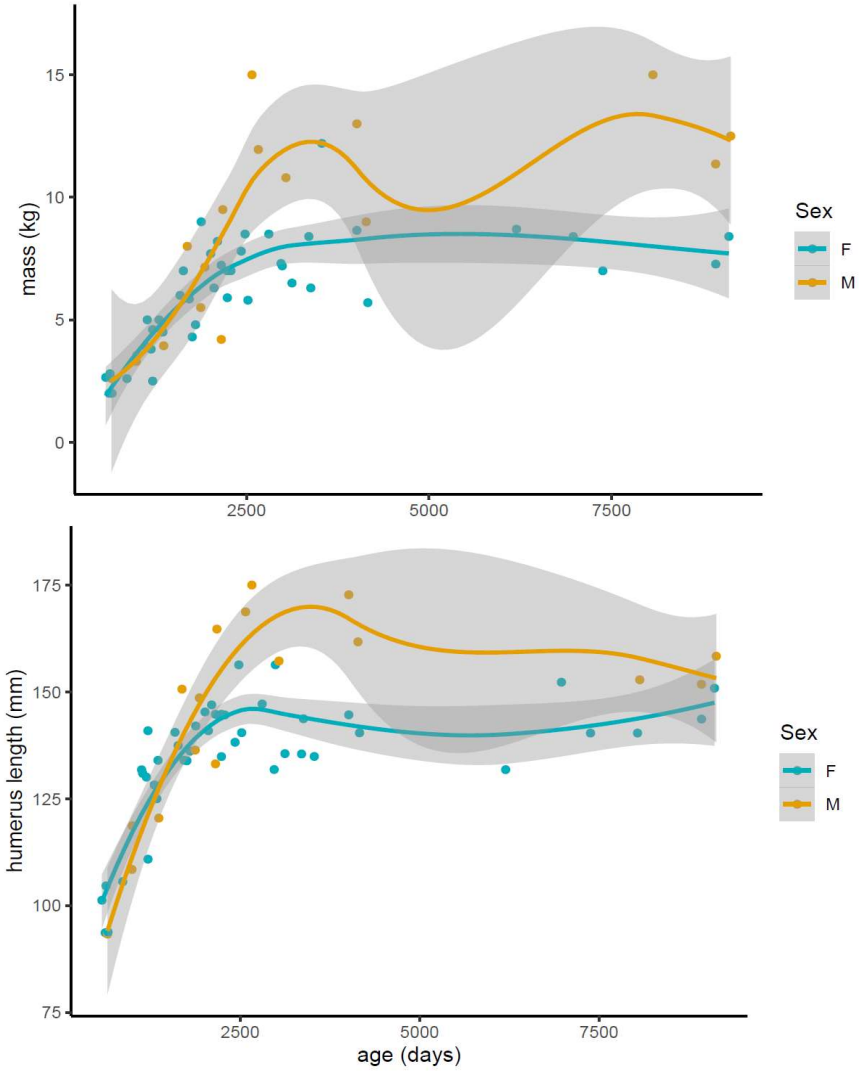


Figure 3.2. Loess-fitted growth curves for body mass (top) and humeral length (bottom) in the Cayo Santiago *M. mulatta* sample.

Among females, temperature and evaporation during the prenatal period had nearly significant negative effects on humeral length and body mass (indicated by a ^ in the temp and evap cells in Table 3.3 Part A), while precipitation and temperature during

Table 3.3 Part A. Linear models of growth rate and body size for female subadults (n=36); bolded predictors are significant at the alpha = 0.05 level. Significance level codes: ^0.1; *0.05; **0.01; ***0.001. No models of GR_{max} were significant; non-significant predictors are indicated by empty cells in the table. Variables not included in a model indicated by --.

		standardized beta coefficients						**linear model**	
	model	evap	precip	temp	age	AICc	ΔAICc	R^2	p-value
	$GR_{overall}$								
	~ **evap + temp** + age	**0.44***	--	**-0.49***		101.44	0.00	0.18	0.02
	~ **evap + temp**	**0.44***	--	**-0.56****	--	101.63	0.19	0.15	0.03
	~ evap + precip + **temp**	0.41^		**-0.48***	--	103.09	1.65	0.14	0.05
	~ **evap** + precip + **temp** + age	**0.42***		-0.44^		103.48	2.04	0.17	0.05
	~ age	--	--	--	-0.31^	103.63	2.19	0.07	0.07
	~ evap + age		--	--	-0.34^	105.04	3.60	0.06	0.13
	~ evap + precip		-0.30^	--	--	105.77	4.33	0.04	0.18
prenatal conditions	Humeral length								
	~ **age**	--	--	--	**0.83*****	62.37	0.00	0.69	<0.01
	~ temp + **age**	--	--	-0.19^	**0.89*****	62.60	0.23	0.71	<0.01
	~ evap + **age**	-1.07^	--	--	**0.86*****	63.53	1.16	0.70	<0.01
	~ evap + precip + **age**			--	**0.90*****	63.83	1.46	0.71	<0.01
	~ precip + temp + **age**	--			**0.91*****	64.08	1.71	0.71	<0.01
	~ evap + temp + **age**		--		**0.89*****	64.51	2.14	0.71	<0.01
	~ evap + precip + temp + **age**				**0.91*****	65.82	3.45	0.71	<0.01
	Body mass								
	~ temp + **age**	--	--	-0.15^	**0.91*****	57.21	0.00	0.75	<0.01
	~ evap + **age**	-0.15^	--	--	**0.89*****	57.22	0.01	0.75	<0.01
	~ evap + precip + **age**			--	**0.92*****	58.05	0.84	0.75	<0.01
	~ **age**	--	--	--	**0.86*****	58.14	0.93	0.74	<0.01
	~ evap + temp + **age**		--		**0.91*****	58.81	1.60	0.75	<0.01
	~ temp + precip + **age**	--			**0.92*****	58.93	1.72	0.75	<0.01
	~ evap + precip + temp + **age**				**0.92*****	60.35	3.14	0.75	<0.01

Continued on next page

the first year of life were significantly negatively correlated with body mass and humeral length, respectively (Table 3.3 Part B). No environmental variables were significant predictors of body mass or humeral length in male subadults (Table 3.4). Among females,

Table 3.3 Part B. Continued from previous page

first year conditions	standardized beta coefficients						linear model	
model	evap	precip	temp	age	AICc	ΔAICc	R^2	p-value
$GR_{overall}$								
~ **evap + temp**	**0.61****	--	**-0.58****	--	95.11	0.00	0.29	<0.01
~ **evap + temp** + age	**0.58****	--	**-0.54****		97.37	2.26	0.27	<0.01
~ **evap** + precip + **temp**	**0.61****		**-0.60****	--	97.46	2.35	0.28	<0.01
~ **evap** + precip + **temp** + age	**0.59****		**-0.57***		99.86	4.75	0.25	0.01
~ evap + age	0.28^	--	--	-0.29^	102.67	7.56	0.12	0.04
~ evap	0.30^	--	--	--	103.91	8.80	0.06	0.08
~ **evap** + precip	**0.34***		--	--	104.04	8.93	0.09	0.08
~ evap + precip + age	0.31^		--		104.06	8.95	0.12	0.07
Humeral length								
~ **temp** + **age**	--	--	**-0.21***	**0.91*****	62.05	0.00	0.72	<0.01
~ precip + **age**	--	-0.18^	--	**0.88*****	62.71	0.66	0.71	<0.01
~ precip + temp + **age**	--			**0.87*****	63.40	1.35	0.72	<0.01
~ evap + precip + **age**			--	**0.87*****	63.40	1.35	0.72	<0.01
~ evap + **age**		--	--	**0.82*****	63.95	1.90	0.71	<0.01
~ evap + temp + **age**		--		**0.90*****	64.22	2.17	0.71	<0.01
~ evap + precip + temp + **age**				**0.90*****	65.31	3.26	0.71	<0.01
Body mass								
~ temp + **age**	--	--		**0.91*****	55.89	0.00	0.74	<0.01
~ **precip** + **age**	--	**-0.18***	--	**0.91*****	56.02	0.13	0.76	<0.01
~ evap + precip + **age**		-0.17^	--	**0.91*****	58.24	2.35	0.75	<0.01
~ precip + temp + **age**	--			**0.92*****	58.32	2.43	0.75	<0.01
~ evap + **age**		--	--	**0.86*****	59.71	3.82	0.73	<0.01
~ evap + precip + temp + **age**				**0.91*****	60.77	4.88	0.75	<0.01
~ evap + temp + **age**		--		**0.90*****	61.27	5.38	0.73	<0.01

prenatal and first year temperature were negatively correlated with growth rate (Table 3.3 Part A), while first year and prenatal evaporation were significantly positively correlated with overall growth rate (Table 3.3 Part B); no environmental variables were significant

Table 3.4 Part A. Linear models of growth rate and body size for male subadults (n=17). Significance codes: ^0.1; *0.05; **0.01; ***0.001; non-significant predictors are indicated by an empty cell. All full models are shown; additional $GR_{max\ avg}$ models that were not significant are not included here.

		standardized beta coefficients						**linear model**	
	model	evap	precip	temp	age	AICc	ΔAICc	R^2	p-value
prenatal conditions	$GR_{overall}$								
	~ **age**	--	--	--	**0.52***	48.02	0.00	0.23	0.03
	~ precip + age	--		--	0.42^	48.71	0.69	0.26	0.04
	~ temp + age	--	--		0.50^	50.49	2.47	0.18	0.10
	~ evap + **age**		--	--	**0.52***	50.58	2.56	0.17	0.10
	~ precip + temp + age	--			0.43^	51.64	3.62	0.20	0.12
	~ evap + temp + age		--		0.50^	53.48	5.46	0.14	0.19
	~ evap + precip + temp + age					55.10	7.08	0.14	0.23
	Humerus length								
	~ **age**				**0.92*****	22.30	0.00	0.83	<0.01
	~ precip + **age**				**0.91*****	24.79	2.49	0.82	<0.01
	~ temp + **age**				**0.91*****	24.84	2.54	0.82	<0.01
	~ evap + **age**				**0.92*****	24.87	2.57	0.82	<0.01
	~ evap + temp + **age**			--	**0.91*****	27.70	5.40	0.81	<0.01
	~ evap + precip + **age**		--		**0.91*****	27.74	5.44	0.81	<0.01
	~ precip + temp + **age**				**0.90*****	27.77	5.47	0.80	<0.01
	~ evap + precip + temp + **age**				**0.91*****	31.14	8.84	0.80	<0.01
	Body mass								
	~ **age**	--	--	--	**0.89*****	27.14	0.00	0.77	<0.01
	~ evap + **age**		--	--	**0.93*****	27.84	0.70	0.78	<0.01
	~ temp + **age**	--	--		**0.91*****	29.21	2.07	0.76	<0.01
	~ precip + **age**	--		--	**0.87*****	29.58	2.44	0.76	<0.01
	~ evap + precip + **age**			--	**0.90*****	30.40	3.26	0.77	<0.01
	~ evap + temp + **age**		--		**0.92*****	30.81	3.67	0.77	<0.01
	~ temp + precip + **age**	--			**0.89*****	31.59	4.45	0.76	<0.01
	~ evap + precip + temp + **age**				**0.90*****	33.87	6.73	0.75	<0.01

Continued on next page

predictors of overall growth rate in males (Table 3.4). Together, these results indicate some influence of early life climatic conditions on female body size and growth rate, but not on male body size and growth rate.

Table 3.4 Part B. Continued from previous page

		standardized beta coefficients						**linear model**	
	model	evap	precip	temp	age	AICc	ΔAICc	R^2	p-value
first year conditions	$GR_{overall}$								
	~ evap + **age**		--	--	**0.54***	50.32	0.00	0.19	0.09
	~ precip + **age**	--		--	**0.55***	50.41	0.09	0.18	0.10
	~ temp + **age**	--	--		**0.53***	50.46	0.14	0.19	0.10
	~ evap + precip + **age**			--	**0.56***	53.17	2.85	0.12	0.21
	~ evap + temp + **age**		--		**0.54***	53.30	2.98	0.12	0.20
	~ temp + precip + **age**	--			**0.55***	53.38	3.06	0.12	0.21
	~ evap + precip + temp + **age**				**0.57***	56.62	6.30	0.06	0.34
	Humeral length								
	~ temp + **age**	--	--		**0.91*****	24.73	0.00	0.82	<0.01
	~ precip + **age**	--		--	**0.92*****	24.78	0.05	0.82	<0.01
	~ evap + **age**		--	--	**0.92*****	24.87	0.14	0.82	<0.01
	~ precip + temp + **age**	--			**0.93*****	27.26	2.53	0.81	<0.01
	~ evap + temp + **age**		--		**0.92*****	27.55	2.82	0.81	<0.01
	~ evap + precip + **age**			--	**0.93*****	27.77	3.04	0.80	<0.01
	~ evap + precip + temp + **age**				**0.94*****	30.33	5.60	0.80	<0.01
	Body mass								
	~ precip + **age**	--		--	**0.92*****	28.41	0.00	0.78	<0.01
	~ temp + **age**	--	--		**0.90*****	28.70	0.29	0.77	<0.01
	~ evap + **age**		--	--	**0.89*****	29.62	1.21	0.76	<0.01
	~ precip + temp + **age**	--			**0.92*****	31.20	2.79	0.76	<0.01
	~ evap + precip + **age**			--	**0.92*****	31.36	2.95	0.76	<0.01
	~ evap + temp + **age**		--		**0.89*****	31.62	3.21	0.76	<0.01
	~ evap + precip + temp + **age**				**0.91*****	34.57	6.16	0.74	<0.01

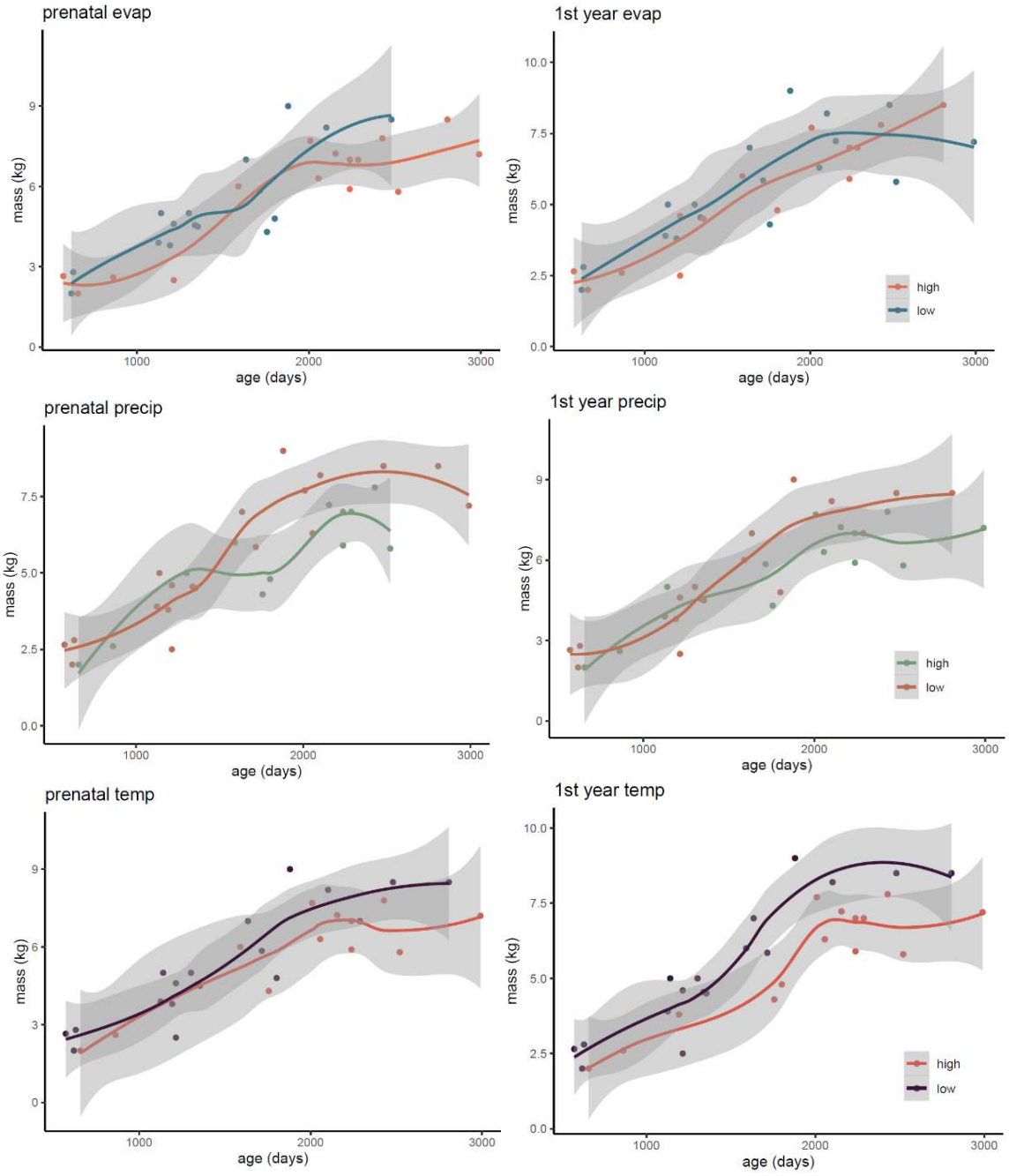


Figure 3.3. Female subadult body mass growth curves for high and low environment categories. Prenatal conditions are on the left; first year conditions on the right.

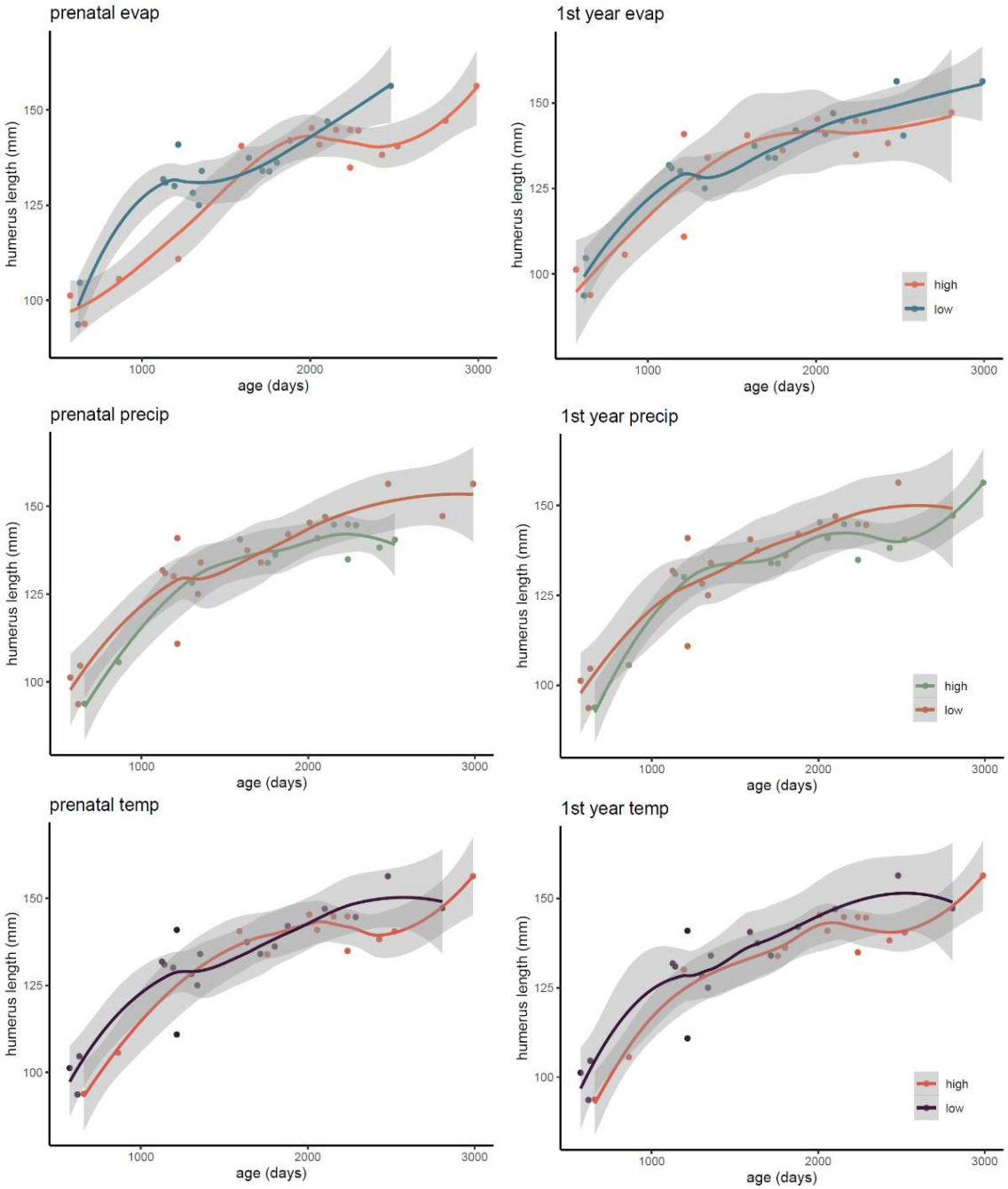


Figure 3.4. Female subadult humeral length growth curves for high and low environment categories. Prenatal conditions are on the left; first year conditions on the right.

Visual representations of general population-level trends in female body size with age under different climatic conditions ("low" versus "high") are presented in Figure 3.3 (body mass) and Figure 3.4 (humeral length). These plots fit loess curves to the data; von Bertalanffy growth model parameter estimates are presented in Table 3.5 (body mass) and Table 3.6 (humeral length). Among subadult females, average asymptotic mass (W_∞) and humeral length (L_∞) estimates are all higher for individuals who experienced higher evaporation levels, lower precipitation, and lower temperatures during both their prenatal period and first year of life. The 95% confidence intervals for W_∞ and L_∞ estimates under high versus low conditions, however, do overlap and are quite large in some cases (e.g., high prenatal and first year evaporation).

Female subadult body mass and humeral length growth curves (Figures 3.3 and 3.4) reveal divergent trends in body size given environmental conditions. Younger individuals appear more similar, with differences becoming more pronounced as individuals age. These trends appear to be stronger for body mass than for the growth of the humerus. Pairwise comparisons between high- and low-condition groups lend modest support to this observation (Table 3.7). Kruskal-Wallis tests revealed significant differences in body mass (and nearly significant differences in stature) between older subadults experiencing high versus low prenatal precipitation (Table 3.7). These results should be considered critically, however, as the significant differences in body mass for young juveniles under different evaporation conditions could be attributed to all of the youngest (and smallest-bodied) individuals falling into the high evaporation category.

Table 3.5. Body mass growth parameter estimates. Von Bertalanffy curve could not be reasonably fit to male subadult data. Confidence intervals based on 5000 bootstrap iterations.

		W_∞			k			t_0		
sample		**est.**	**SE**	**95% CI**	**est.**	**SE**	**95% CI**	**est.**	**SE**	**95% CI**
all male		14.09	2.13	10.96, 20.70	0.0008	0.0003	0.0003, 0.0018	1172.81	247.09	679.56, 1729.60
subadult male		NA	NA	NA	NA	NA	NA	NA	NA	NA
all female		8.47	0.51	7.62, 9.67	0.0012	0.0003	0.0007, 0.0018	742.92	104.09	490.98, 915.60
subadult female		9.01	1.46	7.35, 18.64	0.0010	0.0004	0.0003, 0.0018	761.06	121.51	568.56, 1714.41
prenatal	high precip	8.11	2.36	6.23, 13.45	0.0011	0.0007	0.0001, 0.0027	758.23	203.97	492.81, 1219.63
	low precip	9.63	1.62	7.68, 13.08	0.0010	0.0004	0.0003, 0.0019	800.23	130.34	600.88, 1729.98
	high temp	7.56	1.19	6.23, 13.61	0.0014	0.0007	0.0004, 0.0050	747.36	151.05	518.33, 1293.29
	low temp	12.26	4.68	8.22, 22.19	0.0007	0.0004	0.0001, 0.0015	999.888	386.71	665.88, 1819.8
	high evap	8.95	2.03	6.99, 28.65	0.0010	0.0005	0.0002, 0.0021	809.28	199.14	569.99, 2869.06
	low evap	5.64	0.48	4.90, 8.63	0.0308	0.0982	0.0010, 1.6838	615.73	33.98	351.06, 747.59
1st year	high precip	7.56	1.08	6.41, 14.36	0.0014	0.0007	0.0004, 0.0030	722.70	127.57	484.57, 1300.74
	low precip	11.94	4.31	8.16, 21.20	0.0007	0.0004	0.0001, 0.0016	977.48	350.99	658.54, 1730.27
	high temp	8.45	1.95	6.59, 31.31	0.0010	0.0006	0.0002, 0.0025	841.18	189.20	583.67, 3207.88
	low temp	11.78	3.12	8.51, 18.47	0.0008	0.0003	0.0002, 0.0015	945.55	232.51	682.13, 4520.07
	high evap	15.07	11.39	8.29, 40.15	0.0005	0.0004	0.0001, 0.0012	1394.11	1166.6	733.68, 3961.79
	low evap	6.11	0.44	5.38, 7.29	0.0038	0.0841	0.0016, 0.3287	617.47	28.39	360.54, 696.39

Table 3.6. Humerus growth parameter estimates. Confidence intervals based on 5000 bootstrap iterations. Von Bertalanffy curve was a poor fit for the subadult male sample (italics)

		L_∞			k			t_0		
sample		**est.**	**SE**	**95% CI**	**est.**	**SE**	**95% CI**	**est.**	**SE**	**95% CI**
all male		166.36	6.84	155.97, 180.33	0.0011	0.0003	0.0006, 0.0020	-40.98	268.86	-682.02, 316.60
subadult male		*470.32*	*1446*	*171.83, 2502.0*	*0.00011*	*0.0005*	*0.00002, 0.009*	*-143*	*1983*	*-2541.78, 4.74*
all female		143.46	2.05	139.82, 147.63	0.0019	0.0004	0.0013, 0.0028	9.06	127.33	-283.67, 213.80
subadult female		150.75	4.53	144.26, 163.26	0.0013	0.0003	0.0008, 0.0019	-196.69	182.57	-667.29, 70.97
prenatal	high precip	143.51	2.99	139.49, 150.89	0.0019	0.0005	0.0012, 0.0029	113.57	142.00	-213.11, 308.24
	low precip	157.43	7.76	146.39, 181.20	0.0011	0.0003	0.0005, 0.0018	-356.21	268.19	-1104.01, 13.57
	high temp	149.64	6.52	142.26, 169.26	0.0014	0.0005	0.0006, 0.0027	-58.61	268.81	-727.66, 296.70
	low temp	154.14	8.01	143.35, 181.76	0.0012	0.0004	0.0005, 0.0020	-309.24	269.03	-1092.50, 57.35
	high evap	**162.94**	**16.23**	**147.11, 274.55**	**0.0001**	**0.0004**	**0.0002, 0.0014**	**-632.27**	**474.11**	**-2215.33, -72.81**
	low evap	**136.26**	**2.18**	**132.80, 141.42**	**0.0270**	**0.0290**	**0.0032, 0.1270**	**577.03**	**51.00**	**213.91, 611.17**
1st year	high precip	145.35	3.52	140.69, 155.13	0.0019	0.0006	0.0010, 0.0030	109.57	179.59	-377.74, 331.94
	low precip	156.15	9.30	144.14, 189.21	0.0011	0.0004	0.0005, 0.0019	-335.21	291.56	-1156.05, 43.62
	high temp	148.70	5.14	142.48, 165.97	0.0014	0.0005	0.0007, 0.0024	-47.03	233.20	-673.14, 258.06
	low temp	156.20	9.63	143.38, 190.25	0.0011	0.0004	0.0005, 0.0020	-317.97	268.87	-1143.61, 60.29
	high evap	147.54	7.31	138.63, 183.16	0.0014	0.0006	0.0004, 0.0028	-137.41	306.09	-1273.63, 231.04
	low evap	138.60	2.46	134.51, 144.21	0.0250	0.0320	0.0029, 0.1070	575.70	61.13	180.69, 610.82

Table 3.7. Mean body mass (kg) and humeral length (cm) estimates for female subadults under different environmental conditions. Subadults are classified as either young (<1500 days) or old (1501-3000 days). Bolded rows indicate that there is a statistically significant difference between mass or stature estimates for individuals under different conditions.

		mass							
environment variable		young				old			
		high	**low**	**H-stat**	**p-value**	**high**	**low**	**H-stat**	**p-value**
pre-natal	precip	3.20	3.63	0.26	0.61	**6.20**	**7.58**	**4.70**	**0.03**
	temp	2.30	3.75	2.83	0.09	6.52	7.36	1.56	0.21
	evap	**2.44**	**4.01**	**5.03**	**0.02**	6.94	6.81	0.01	0.93
1st year	precip	3.35	3.61	0.22	0.64	6.51	7.73	2.03	0.15
	temp	2.80	3.75	2.08	0.15	6.46	7.58	1.80	0.09
	evap	3.14	3.66	1.48	0.22	6.84	6.93	0.02	0.89

		length							
		young				old			
		high	**low**	**H-stat**	**p-value**	**high**	**low**	**H-stat**	**p-value**
pre-natal	precip	109.2	120.3	1.02	0.31	139.8	145.2	3.28	0.07
	temp	99.7	121.0	1.19	0.17	142.0	143.1	0.16	0.69
	evap	**102.9**	**124.3**	**1.42**	**0.06**	143.4	141.0	1.18	0.27
1st year	precip	115.1	118.9	0.10	0.75	141.4	143.9	0.87	0.35
	temp	109.8	120.1	0.71	0.40	141.8	143.5	0.40	0.53
	evap	114.4	120.6	0.02	0.87	141.5	143.3	0.01	0.93

3.4. Discussion

Collectively, these results provide modest support for the hypothesis that growth rate and attained body mass are linked to the environmental conditions experienced early in life. Although (unsurprisingly) the best predictor of overall body size is age (Tables 3.2-3.4), some of the variation in body size left unexplained by age seems to be explained by environmental differences. The effect of environment on body mass and humeral length (as a proxy for body size) throughout ontogeny is more pronounced in female subadults than in male subadults, although this may be due to the small male subadult sample size. Indeed, particularly for the male subadult sample, removing environmental variables frequently improved the fit of the model (as evidenced by lower AICc values;

Table 3.4). Unfortunately, small sample sizes prevented a thorough analysis of the effect of rank (and the interactions between rank and experienced environment) on growth, but in general, the best fitting linear models of subadult humeral length and body mass included age and at least one environmental variable. It is worth reinforcing, however, that all environmental effect sizes are small compared to the (expected) large effects of age on body size. Relatively high R^2 values (0.69- 0.83 for models of humeral length and body mass) suggest that linear models fit the log-transformed body size data fairly well.

The nature of the relationship between environmental variables and body size was somewhat unexpected. First-year precipitation and temperature were negatively correlated with body mass and stature, respectively. While temperature effects may not mean much biologically, as maximum temperatures do not fluctuate wildly on tropical Cayo Santiago (standard deviation = 1.36° F), the negative correlation between precipitation and growth may be linked to hurricane effects or more generally, rainy-season health effects. During the Cayo Santiago rainy season, when precipitation increases, parasite load within the population tends to increase as well (Snyder-Mackler, pers. comm.). Morbidity and/or mortality linked to parasites and infectious diseases are linked to reduced growth rate and body condition (Bozzoli et al., 2009; Stulp & Barrett, 2014), which may underpin the greater plasticity of body mass in response to environment compared to stature. Links to disease and health would be an interesting avenue for future research. It would also be valuable to explore how environmental variation within a developmental period (rather than simply environmental minima, maxima, or averages) affect growth, especially within primate populations that experience more extreme conditions than the Cayo Santaigo macaques.

Nonetheless, despite the small environmental effects, it is intriguing that environmental conditions experienced postnatally and during the first year of life were linked to growth differences later in life. The youngest individual included was approximately 1.5 years old, and divergences in growth trajectory given early environmental conditions were most pronounced for older subadults over ~1500 days (i.e., ca. 4 years; Figures 3.3, 3.4). This result is consistent with studies revealing that early-life environments and maternal conditions can program later-life responses in other traits (Anacker et al., 2014; Rödel et al., 2009; Tung et al., 2016). These effects are frequently linked to stress responses of the hypothalamic-pituitary-adrenal axis (Smith & Vale, 2006), so it may be that similar mechanisms program the environmentally sensitive growth responses of the hypothalamic-adrenal-somatotropic axis. In other words, perhaps growth does not simply respond dynamically to current conditions; instead, perhaps the trajectory and/or pace of growth is pre-programmed based on the conditions experienced prenatally and/or during the first few months of life (A. Lu et al., 2019), utilizing the mechanisms of developmental plasticity to adjust growth an developmental outcomes in such a way to optimize later-life fitness. The persistence of early life effects has been mechanistically linked to epigenetic mechanisms such as DNA methylation (Anacker et al., 2014; Bush et al., 2016) so it would be worthwhile to explore the epigenetic signatures of early life and maternal conditions on growth.

While this analysis specifically sought to explore the relationship between early-life conditions and later growth, an exploration of environmental effects throughout the growth period would be a complement to the above approach. If there are significant differences between early-life environment and later-life environments, it may be that

conditions experienced more recently in an individual's life have a different or greater effect on growth outcomes than early-life conditions. More thorough investigations of individual responses to potential differences between early-life environments and later-life environments may further shed light on predictive adaptive response hypotheses, which address the fitness effects of "mismatches" between early and later life environments (Wells, 2005).

In mammals, challenging conditions have been implicated shorter growth periods (Migliano et al., 2007), as well as both faster (Berghänel et al., 2017) and slower (Jarrett et al. 2020) rates of growth. A population's response to challenges likely depends on what is most energetically and reproductively favorable given its specific, local ecology as well as the nature of the stressor. These results suggest that smaller body sizes in the Cayo population may be linked, at least in part, to slower growth rates under challenging conditions. Accepting that unfavorable or challenging conditions will result in smaller body size, the "challenging" environment is characterized by higher precipitation, lower evaporation, and higher temperatures (Tables 3.5, 3.6). Though von Bertalanffy parameter estimates were too wide to robustly test differences in growth rates and growth periods between conditions, some general inferences can be made by comparing the effects of environment on growth outcomes and growth rate.[6]

[6] Caution should be used in the application of von Bertalanffy growth model parameters, as 95% confidence intervals are very large for some samples' parameter estimates. Average asymptotic humeral length estimate for subadult males, for example, is 470 mm, while the 95% confidence interval ranges from 172 mm to an absurd 2502 mm. Although not as extreme, body mass estimates for the female high first year evaporation condition range from 8 to 40 kg (an unbelievably large body mass for a female rhesus macaque). This is likely due to the small sample sizes of these subsets.

While growth rate was not a significant predictor of body size (in models that did not include environmental variables), first-year evaporation is significantly positively correlated with overall growth rate (best-fitting model beta coefficient = 0.61; Table 3.3). In other words, individuals who experienced the more favorable condition (higher first year evaporation) have larger estimated body masses than those who experienced the less favorable condition, lower evaporation (high W_∞ = 8.95; low W_∞ = 5.64). Similarly, first year temperature is negatively correlated with overall growth rate (best fitting model beta coefficient = -0.58; Table 3.3), and temperature is also negatively correlated with body mass (beta coefficient = -0.21; Table 3.3). These trends, however, are not consistent for the precipitation condition, as precipitation is not a significant predictor of growth rate in any model, but high first year precipitation appears to have a significant effect on body mass (Table 3.3). Similarly, there visually appears to be an effect of prenatal precipitation on body mass (Figure 3.3), but this visual trend is not confirmed by linear model results (Table 3.3). Inconsistencies may be due to the error inherent in comparing linear models, as well as differences between categorical and continuous environmental variables. Conclusions would be stronger if they could be supported by statistically significant differences in von Bertalanffy growth parameter estimates (Tables 3.5, 3.6). Further exploration with additional body mass and stature data to more effectively model differences in both the pace and duration of growth would be valuable to tease out whether differences in environmental conditions result in changes to the pace of growth, duration of growth, or both.

While age and environment were weak predictors of growth rate ($R^2 < 0.29$ for all models), this may be linked to the growth rate metrics themselves. As discussed in

Chapter 2 (section 2.5), the measures of growth used here are only proxies, so it is perhaps unsurprising that they were not strongly linked to early life conditions or age. Growth is not linear, so building growth curves with only a few points likely linearizes the curve and obscures important shifts in growth that may be linked to age and affected by environmental conditions. For example, even though environmental effects on growth rate were only investigated for subadults, if $GR_{overall}$ and $GR_{max\ avg}$ metrics were not sensitive enough to capture subtle temporal changes, and instead model growth as constant throughout the subadult period, one might not expect to be able to identify environment-driven differences in age-specific growth rate. A potential solution to this problem would be to amass more detailed longitudinal body mass data to permit the calculation of more sophisticated measures of individual growth rate, such as those based on the *k* parameter that was calculated at the population-level. While this would not permit a direct assessment of environment on individual growth—it would require assigning individuals to "high quality" and "low quality" groups—it would make it possible to link maximum growth rate to the time at which that growth rate occurs and thus directly compare age-specific growth rates between categorical environment cohorts.

It is worth noting that these results derive from a population of individuals who died as juveniles, so they may not reflect the growth trajectories of healthy macaques who died of natural causes later in life. This effect may be more pronounced for body mass than stature (body mass reflects body condition and therefore may vary more drastically in unwell individuals) and could underpin the larger visual effects of environment on body mass (Figure 3.3) than on humeral length (Figure 3.4). Furthermore, because the Cayo Santiago population is provisioned, these results may not

be directly applicable to wild populations. Food availability is a major driver of growth, and wild populations may spend more energy foraging for less-nutritious food than the provisioned Cayo macaques. This may translate into slower growth in wild populations (Altmann et al., 1993; Turner et al., 2016). Sample sizes prevented separate analyses for pre-weaned infants and self-foraging juveniles, but juveniles often have reduced foraging efficiency compared to adults as the result of inexperience or small size (Altmann, 1980; Boinski & Fragaszy, 1989).

Ultimately, these results suggest that growth within the Cayo Santiago population is plastic. The observed differences in growth between individuals who experienced different environments fits within the broader narrative that early life experiences have lasting phenotypic effects. This idea opens up a number of avenues for future research, particularly with respect to the epigenetic and hormonal mechanisms that underpin environmentally mediated variation in growth. The results also have implications for the evolution of growth and life history. Differences in growth and development likely have meaningful fitness consequences (e.g., Blomquist, 2009; Hayward et al., 2013; Kramer, 2008; W.-S. Lee et al., 2012; Mangel & Stamps, 2001; Quesnel et al., 2018; Rollo, 2002; P. C. Wright, 1999) and plastic changes in traits are important for the short-term persistence of a population (e.g., Moritz & Agudo, 2013). Although adaptive change is a phenomenon that operates on a larger scale, understanding the short-term mechanisms upon which long term adaptive change in populations depends is crucial.

CHAPTER 4

INTERSPECIFIC EFFECTS OF SOCIOECOLOGICAL SELECTIVE PRESSURES ON GROWTH RATE AND THEIR LINKS TO SELLA TURCICA SIZE

4.1. Introduction

Just as growth and development are essential for the success of individuals, the aggregate effects of individual success are necessary for species to persist and thrive. Aspects of growth directly impact juvenile survival, age at first reproduction, and lifetime reproductive output, which means that the trajectory of growth can influence evolutionary success (Charnov & Schaffer, 1973; Dmitriew, 2011; Walker et al., 2006). Although primates as a group are characterized by long lifespans, late ages at maturity, and small litter sizes that position them on the "slow" end of the (simplified) mammalian life-history spectrum (Austad & Fischer, 1992; Bogin, 1999b; Charnov & Berrigan, 1993; Jones, 2011; Kappeler et al., 2003; Pagel & Harvey, 1993; Promislow & Harvey, 1990; Stearns, 1992; R. Walker, Burger, et al., 2006), variation in the timing, duration, and pace of growth exists within the order. Interspecific differences in the timing of growth and developmental events, like all life-history traits, are shaped by selective pressures related to trade-offs in energy allocation and acquisition throughout life.

Key among the ecological variables that shape growth rate is extrinsic mortality. Age-specific extrinsic mortality has long been hypothesized to play a major role in driving interspecific variation in growth rates and the pace of life across mammals (Charnov, 1993; Charnov & Schaffer, 1973; Kozłowski & Weiner, 1997; Williams, 1957), with higher mortality rates, especially during the juvenile period, typically linked to faster growth, development, and life history (e.g., Bronikowski et al., 2011; Charnov &

Schaffer, 1973; Janson & van Schaik, 1993; Jones, 2009; Kramer, 2008; Kramer et al., 2009; Mangel & Stamps, 2001; Shattuck & Williams, 2010). Primates grow slowly compared to many other mammals (Jones, 2011), which likely reflects the low extrinsic mortality risk afforded by the primate lineage's long evolutionary history of arboreality (Martin, 1990; Shattuck & Williams, 2010). While this observation makes it reasonable to hypothesize that primate populations experiencing higher mortality due to a terrestrial lifestyle would exhibit faster growth than more arboreal populations, mortality risk is not solely the product of substrate use (or any other single ecological trait for that matter).

Within the order Primates, the relationship between growth rate and mortality varies depending on which ecological and growth variables are considered. Because much growth occurs during the juvenile period, many models focus on juvenile mortality, proposing that the *slow* juvenile growth of some primate species is the product of *high* juvenile mortality (see below; Charnov & Schaffer, 1973; Janson & van Schaik, 1993). Other research focuses on adult mortality, contending that within-order variation follows the mammalian trend and that ecological settings that entail high adult mortality favor relatively fast growth to reach maturation earlier (e.g., Charnov, 1993; Kramer et al., 2009). In addition to the distinction between adult and juvenile mortality, a key difference between these high- versus low-mortality hypotheses is the actual cause of mortality. High extrinsic mortality linked to predation (of both adults and juveniles), for example, is posited to favor faster growth, while mortality linked to resource availability, energetics, and the threat of starvation—particularly during the juvenile period—is proposed to favor slower growth. Because slow growth reduces metabolic costs, the ecological risk aversion hypothesis (ERAH) suggests that slow growth may be a way for

juveniles to mitigate the risk of starvation that is encountered when unskilled juveniles must compete with skilled adults for scarce resources (Janson & van Schaik, 1993). Thus, slow growth—and the reduced energetic demands that accompany it—may be an adaptation that reduces juvenile-adult feeding competition and addresses population density-dependent mortality risks, such as competition for resources (Clutton-Brock et al., 1987; Fowler, 1981; Hernández-Pacheco et al., 2013; Purvis & Harvey, 1995) that may not directly linked to predation.

Even in the absence of juvenile-adult competition and accompanying selective pressures favoring slower growth, the correlation of slow growth with an extended juvenile growth period, large body size, and large brain size could be the product of diverting energy away from rapid body growth in order to support a large, energetically expensive brain (Foley & Lee, 1991; Kuzawa et al., 2014; Martin, 1996; Navarrete et al., 2011). In models that focus on increased brain volume and its associated costs, large brains are proposed to "anchor" slow primate growth rates by imposing constraints on growth and reproductive rates, requiring a long developmental period to achieve maturity, or a combination of the two (Chapter 1, section 1.2; Charnov, 1993; Leigh & Blomquist, 2007; Martin, 1996). Although it is often difficult to evaluate the direction of causality in studies of the correlations between brain size and the timing of various growth and developmental milestones, research largely suggests that primate species with larger absolute and relative brain sizes would be expected to exhibit slower growth rates. Furthermore, the influence of energetically and developmentally expensive brains and food acquisition challenges on growth rates need not be mutually exclusive. Among modern humans, for example, the ecological challenges related to acquiring food are

posited to be the strongest predictor of human growth patterns (Koster et al., 2020), with the developmental trajectory of human brains proposed to allow for longer learning periods that are, presumably, linked to greater foraging efficiency (González-Forero & Gardner, 2018).

Between-population variation in growth rates and trajectories has also been directly linked to diet and the availability of food resources. Mammalian species that specialize in abundant or reliable foods have faster body mass production rates than species that do not (Sibly & Brown, 2007). Because many primates rely on fruits that can be seasonally unpredictable or scarce (Chapman et al., 1999), slow growth and the accompanying reduced metabolic costs may be adaptations to inherent irregularity in resources or environmentally linked periods of food scarcity (in addition to juvenile-adult competition-imposed scarcity posited by hypotheses such as the ERAH) (Janson & van Schaik, 1993; Jones, 2011). Environmental uncertainty and unpredictability often favors the evolution of bet-hedging strategies (Dewar & Richard, 2007; Ellis et al., 2009; Richard et al., 2002; Stearns, 1976) such as slow growth, so an interspecific application of the ERAH predicts that folivorous primates, who would not be subjected to the same levels of uncertainty as primarily frugivorous species, should have accelerated life histories and exhibit faster growth relative to closely related frugivores. This is supported by empirical evidence (Conklin-Brittain et al., 1998; Van Noordwijk & Van Schaik, 2005; Wich et al., 2007). Folivorous gorillas, for example, experience accelerated growth rates and an extended early growth spurt compared to chimpanzees and humans (Leigh, 2001). Comparisons between congeneric populations living in different environments (e.g., Breuer et al., 2008; Yamagiwa et al., 2012) further support the idea that physical

maturation schedules are connected to ecological conditions: more frugivorous western gorilla populations wean later and undergo slower physical maturation than more folivorous mountain gorillas (Breuer et al., 2008).

While some research reveals that aspects of life history are *slower* in folivores compared to frugivores (e.g., Godfrey et al., 2004) or indicates no difference between the two dietary regimes (e.g., Borries et al., 2011), these results are likely linked other aspects of ecology or energy availability such as the degree of maternal investment or provisioning, both of which have been shown to accelerate life-history schedules (Asquith, 1989; Gilmore & Cook, 1981; Kiltie, 1982). In strepsirrhines, for example, the observation that typically folivorous taxa (e.g., indriids) grow more slowly and reach reproductive maturity later than more frugivorous taxa (e.g., some lemurids) may reflect different solutions to the ecological problems posed by Madagascar's variable environment (Dewar & Richard, 2007; Godfrey et al., 2004). As the indriid dentition develops quickly (consistent with the predictions of Janson and van Shaik's (1993) ERAH and likely the result of the mechanical demands of the indriid diet), a decoupling of patterns of dental and somatic development occurs (Godfrey et al., 2004). Godfrey and colleagues (2004) argue that, given the different breeding strategies of the two groups, slow indriid growth rates reflect lower maternal investment during the periods that juveniles are dependent upon their mothers and thus, increased energy savings for mothers, with the different developmental strategies in lemurids and indriids reflecting the availability of preferred resources during periods of environmental stress. (Godfrey et al., 2004). Ultimately, research on both strepsirrhines and haplorhines largely implies that the accessibility and availability of resources and energy—rather than food type—are

stronger drivers of variation in life history in many primate populations.

At its core, interspecific variation in growth rate is linked to energy budgets and the energetic trade-offs that underpin life-history theory. Because slow body growth is accompanied by reduced energetic requirements compared to fast growth, energetic costs can be addressed by adjusting body growth rate. After removing the effects of body size, individuals' body mass production rates are tightly linked to food supply and predation risk across mammals (Sibly et al., 2014). Yet, interspecific variation in primate growth rates has not been explored in a comparative phylogenetic framework that considers both mortality risk and available energetic resources. Indeed, many of the ultimate drivers of variation in growth rate are interrelated, making it difficult to link primate growth rates exclusively to any one variable. To date, however, ultimate drivers have largely been explored independently, without testing their relative effects on primate growth in a multivariate analysis that takes into account species' shared ancestry. Thus, this project employs phylogenetically informed methods to test which ecological factors (e.g., mortality risk, the availability of energetic resources due to diet) and sociocognitive factors (e.g., those related to brain size or juvenile-adult competition) best account for variation in growth rate across primates.

In light of the relationship between pituitary volume, growth rates, and hormones across mammals (Chapter 1, section 1.3) and the close anatomical relationship between the pituitary gland and the sella turcica (Chapter 1, section 1.4; Chapter 2, section 2.1), this chapter addresses the hypotheses that (a) sella turcica size is correlated with growth rate across primate species and (b) sella turcica size is also linked to the same ecological

and sociocognitive variables that underpin variation in growth rate.[7] Given the complex

Table 4.1. Variables proposed to underpin variation in growth rate and predicted correlation to growth rate. "-" indicates a predicted negative correlation between two variables; "+" indicates a predicted positive correlation between two variables. NDVI = normalized differential vegetation index (see section *4.2. Materials and methods: life history and ecological data*)

ultimate cause	proxy	predicted correlation	selected source(s)
brain size	brain mass	- *larger brain = slower growth*	Foley & Lee, 1991; Martin, 1996; Charnov, 1993; Navarrete et al., 2011
juvenile-adult competition	group size*	- *larger group = greater competition = slower growth*	Janson & van Schaik, 1993
extrinsic mortality risk due to predation	substrate use (arboreal = 1; semi-arboreal = 2; terrestrial =3)	+ *more terrestrial = higher predation = faster growth*	Charnov & Schaffer, 1973; Martin, 1990; Charnov, 1993; Kozlowski & Weiner, 1997; Shattuck & Williams, 2010; Bronikowski et al., 2011
	group size*	- *larger group = individuals buffered from predation = slower growth*	
food availability	percent leaves in diet*	+ *greater reliance on leaves = increased food availability = faster growth*	Conklin-Brittain et al., 1998; van Noordwijk & van Schaik, 2005; Wich et al., 2007; Breuer et al., 2008; Jones, 2011
diet seasonality	CV of NDVI	- *more variation = uncertain resources = slower growth*	Knott, 2001; Sibly & Brown, 2007; Jones, 2011

* Starred proxies are those that may be also be secondarily linked to other ultimate causes and thus could reasonably be predicted to have a different relationship to growth rate (e.g., group size may be linked to higher disease transmission, which would be expected to have a positive relationship with growth rate, while a higher percentage of leaves in the diet may be linked to lower diet quality, which would be expected to have a negative relationship with growth rate).

[7] Though the intraspecific relationship between sella turcica size has already been tested in a *M. mulatta* population (Chapter 2), intra- and interspecific trends need not mirror each other. The nature of an intraspecific relationship (in the *M. mulatta* case, a weak relationship) is not necessarily informative for an interspecific one, as different forces are at play at different levels of analysis.

relationship between primate mortality risk and growth rate (e.g., Jones, 2011; Kramer et al, 2009; Shattuck & Williams, 2010), it is expected that the strength of the correlations between independent variables and both growth rate and relative sella turcica size will be greater for variables related to the availability of energetic resources than for mortality risk. Specific predictions regarding the direction and strength of the correlation between each independent variable and both growth rate and sella turcica size are listed in Table 4.1 (see *Materials and Methods* for rationale underlying proxy choice and predicted relationships). It is expected that the magnitude and direction of each variable's correlation in analyses that use growth rate as the dependent variable will mirror those that use relative sella turcica size as the dependent variable. If the strength and direction of correlations to sella turcica size are the same as those to growth rate, this will be interpreted as evidence that the drivers of interspecific variation in growth rate also underpin variation in sella turcica size and, likely, hormone production.

4.2. Materials and methods

Sella turcica data

Volumetric sella turcica measurements were collected from microCT scanned primate crania of 51 primate species (each represented by a total of 2-19 adult individuals; Table 4.2) that were generated by Lynn Copes and Lynn Lucas (Copes et al., 2016). These scans are hosted on morphosource.org, although this project utilized copies of the scans stored in the IHO Visualization Lab. Sella turcica volume for each specimen was measured following the protocol used to measure sella turcica volume for the Cayo Santiago sample (Chapter 2.2). A sample of 328 specimens was measured; sella turcica

volume was averaged for each species' male, female, and pooled male and female subsamples. Descriptive summary statistics are provided in Appendix C (Table S4.8).

Table 4.2. Interspecific sella turcica sample. Samples are separated by sex; M = male, F = female.

	Species	*n*		Species	*n*
Strepsirrhini	*Eulemur fulvus fulvus*	M=5; F=7	Catarrhini	*Papio anubis*	M=2; F=2
	Hapalemur griseus	M=4; F=4		*Theropithecus gelada*	M=1; F=1
	Lemur catta	M=4; F=3		*Mandrillus leuchophaeus*	M=1; F=3
	Varecia variegata variegata	M=2; F=6		*Mandrillus sphinx*	M=2; F=2
	Microcebus murinus	M=1; F=5		*Lophocebus albigena*	M=6; F=5
	Propithecus diadema	M=2; F=2		*Cercocebus torquatus*	M=4; F=7
	Propithecus verreauxi verrauxi	M=2; F=3		*Macaca fascicularis*	M=4; F=8
	Avahi laniger	M=1; F=5		*Macaca fuscata*	M=1; F=1
	Nycticebus coucang	M=4; F=3		*Macaca mulatta*	M=2; F=2
	Periodicticus potto	M=2; F=10		*Macaca sylvanus*	M=1; F=1
	Galago alleni	M=2; F=2		*Erythrocebus patas*	M=3; F=2
	Galago senegalensis	M=4; F=6		*Miopithecus talapoin*	M=2; F=4
	Euoticus elegantulus	M=3; F=6		*Cercopithecus mitis*	M=0; F=12
Platyrrhini	*Callithrix argentata*	M=7; F=8		*Nasalis larvatus*	M=5; F=6
	Saguinus mystax	M=6; F=5		*Trachypithecus cristata*	M=4; F=15
	Aotus trivirgatus	M=4; F=8		*Presbytis hosei*	M=2; F=4
	Saimiri sciureus	M=7; F=4		*Presbytis rubicunda*	M=4; F=7
	Cebus capucinus	M=2; F=8		*Piliocolobus badius*	M=5; F=6
	Sapajus apella	M=7; F=10		*Colobus polykomos*	M=6; F=6
	Ateles geoffroui	M=2; F=17		*Homo sapiens*	M=2; F=2
	Alouatta caraya	M=3; F=3		*Pan troglodytes troglodytes*	M=2; F=13
	Alouatta palliata	M=2; F=10		*Pan paniscus*	M=2; F=3
	Cacajao calvus	M=1, F=1		*Gorilla gorilla gorilla*	M=5; F=6
	Chiropotes albinasus	M=0; F=2		*Pongo pygmaeus*	M=1; F=2
	Pithecia pithecia	M=4, F=4		*Hylobates lar*	M=8; F=10
	Callicebus moloch	M=8; F=4		*Symphalangus syndactylus*	M=1; F=1

Life-history and ecological data

Growth, life-history data, and body mass were primarily sourced from the AnAge database (https://genomics.senescence.info/species/) and, in cases of missing data, were supplemented with data from additional published sources (Appendix C, Table S4.1). Growth and life-history variables of interest were body mass at birth, age at weaning, body mass at weaning, age at sexual maturity, and adult mass. From these metrics,

overall growth rate (OGR) was calculated as $\frac{adult\ mass - mass\ at\ birth}{age\ at\ maturity}$ and pre-weaning growth rate (WGR) was calculated as $\frac{mass\ at\ weaning - mass\ at\ bir}{age\ at\ weaning}$. Whenever possible, mass, weaning, and maturity data were collected for both males and females, but for most species, only age at maturity and adult mass were sex-specific. Thus, OGR metrics capture some aspect of sex-linked differences in growth, but WGR metrics pool males and females. Although sufficient growth data were available for 68 primate species, male-specific growth data were only available for approximately 20 species, so only female growth data were used in the analyses. This is consistent with many life-history analyses as females are the sex that bears the brunt of energetic costs related to reproduction (Archie et al., 2014; Charnov & Berrigan, 1993; Gittleman & Thompson, 1988; Hawkes et al., 1998; Key & Ross, 1999). Brain-mass data were sourced from the published literature (e.g., DeCasien et al., 2017; Isler et al., 2008). If a source provided endocranial volume, volume was converted to mass by multiplying by the density of brain tissue, 1.036 g/cm^3 (Stephan et al., 1981).

Ecological data that either directly capture, or are proxies for, variables that are hypothesized to influence growth rate, were also collected from the literature and online databases (see below for details). These data were not available for modern humans so modern humans were not included in the socioecological analyses. Ecological variables of interest are those related to the energy available to a growing individual, such as percentage of leaves in the diet and diet seasonality. The percentage of leaves in a species' natural diet was used as a proxy for food resource availability, as leaves are typically more abundant than fruits and thus can reduce pressures related to food

acquisition (Aristizabal et al., 2019; Chapman et al., 1999; Clink et al., 2017; Reyna-Hurtado et al., 2018; Saj et al., 2007). The percentage of leaves in a species' diet would thus be expected to be positively correlated with growth rate (see Table 4.1). Leaves (particularly mature leaves), however, are typically considered a low-quality food compared to fruits (Chivers & Hladik, 1980; Felton et al., 2009; Marshall & Wrangham, 2007; Masi et al., 2015; Schülke et al., 2006), so the percentage of leaves may also be linked to a species' diet quality and thus could alternatively be hypothesized to be negatively correlated with growth rate, particularly if an individual cannot acquire sufficient young leaves. Therefore, the percentage leaves proxy may in practice capture aspects of multiple variables proposed to affect growth rate in different ways. Although this proxy may be differentially correlated with growth rate, I expect that food availability (as it more directly relates to energetics) to be a stronger driver of growth rate, and thus the percentage of leaves in the diet to be positively correlated with growth rate. In any case, the direction of the relationship between growth rate and percentage of leaves in the diet, if significant, could be used to make inferences about which aspect of a folivorous diet (i.e., food availability or food quality), if any, has a stronger influence on primate growth rate.

Similarly, social-group size was included primarily as a proxy for food competition (as larger social group sizes would be expected to correspond to increased competition and thus would be expected to be associated with slower growth). Group size, however, is also linked to other variables hypothesized to influence growth rate such as predation risk—larger groups typically lower the risk of predation for any one individual (Isbell, 1994), which would also be expected to be linked to slower growth—

and disease transmission—larger groups would be expected to increase mortality risks due to disease transmission (Capitano, 2012; Davies et al., 1991; Markham et al., 2015; Nunn et al., 2015; Nunn & Heymann, 2005), which could favor the evolution of faster growth. Though each proxy was selected with one or two ultimate causes in mind, it is essential to bear in mind the multiple correlations, costs, and benefits of each when making interpretations and drawing conclusions about such a complex, dynamic, and interdependent system.

The percentage of leaves in each species' diet was gathered from published literature (McGrosky et al., 2019 and references therein), while group size data were collected from Dunbar et al. (2018) and Overdorff et al. (1999). Seasonality in vegetation was used as a proxy for seasonality in plant food availability and thus diet. The Normalized Difference Vegetation Index (NDVI) is commonly used as a measure of seasonal variation in plant productivity (e.g., Creech et al., 2016; Uno et al., 2020; van Woerden et al., 2012; Van Woerden et al., 2010) and is calculated using the amount of near-infrared and red light reflected by vegetation as measured by satellites (Pettorelli et al., 2005). NDVI data are freely available from National Oceanographic and Atmospheric Administration's satellite imagery and the Global Inventory Monitoring and Modeling System database (Pinzon & Tucker, 2014; Tucker et al., 2005) and were downloaded for the recorded collection locations of each species. Variation in diet was estimated by using the coefficient of variation ($CV = \frac{standard\ deviation}{mean}$) of each species' NDVI values across one calendar year.

Substrate use was employed as one proxy for extrinsic mortality risk due to predation. Unfortunately, direct acts of predation on primates are difficult to study, so

primary evidence of primate predation rates and predation risk are limited.[8] Predation risk is often linked to substrate use, with time spent arboreally proposed to reduce risk (e.g., Barnett & Spironello, 2012; Barnett et al., 2012; Pruetz et al., 2008). Species were assigned terrestriality values, with 1 representing primarily arboreal species, 2 representing semi-terrestrial species, and 3 representing primarily terrestrial species. Degree of terrestriality, however, may not be the best proxy for predation rate, as terrestrial primates have developed strategies to mitigate the predation risks that they face. Predation risks and risk mitigation strategies are also linked to body size (particularly as primate body size relates to predator body size; Isbell, 1994) and group size (e.g., Bettridge & Dunbar, 2012; Hill & Dunbar, 1998; Hill & Lee, 1998; van Schaik, 1983; van Schaik & Hörstermann, 1994). Although group size data were collected as a proxy for intragroup competition, it is important to note that group size is also linked to predation risk, with an individual within a larger groups afforded a buffer from predation relative to an individual in a smaller group (Hill & Lee, 1998).

It is difficult to disentangle the competing effects of predation pressures and intragroup competition costs on the evolution of primate group size, as the two are unlikely to be independent from each other. Increased group size may decrease predation risk, but it also likely increases competition for scarce food resources (Isbell, 1994;

[8] Predation risk and predation rate, though related, are distinct selective forces that are often conflated (Hill & Dunbar, 1998). Predation risk is the likelihood of an animal encountering a predator, which Hill & Dunbar (1998) argue reflects a species' or population's past experience of attacks and is thus what has historically shaped antipredator strategies. Predation rate is effectively the excess mortality in a population due to predation that cannot be controlled for by adjusting behavior (Hill & Dunbar, 1998). While it is difficult to disentangle the effects of the two on the proxies used here, I make every attempt to be deliberate in word choice when linking a proxy to either predation rate or predation risk.

Janson & Goldsmith, 1995). Group size thus likely reflects a balance between competition costs and predation benefits, and increases in one pressure (predation, for example), may relax constraints on the other (e.g., competition pressure that favors smaller group size). Alternatively, independent of group size, periods of food scarcity can increase predation risk by decreasing vigilance as individuals must search harder for food (Isbell, 1994).

Primates have developed additional anti-predator defenses beyond social living and a largely arboreal lifestyle. As smaller-bodied species are proposed to be subject to higher predation risk than larger-bodied species (Struhsaker, 1969), predation has been posited to be a selective pressure that favors increased body size (Dunbar, 1988; Hill & Dunbar, 1998); some researchers argue that body size has a stronger effect on reducing predation than group size in primates such as great apes (Isbell, 1994). Studies suggest that larger body mass is favored under high predation scenarios, as the larger body size that accompanies larger body mass would reduce some of the predation risk faced by smaller bodied-primates that are available as prey to a larger range of predators (Struhsaker, 1969). This implies that larger-bodied primates experience lower predation *rates* than smaller-bodied species, but because group size and substrate use primarily capture aspects of predation risk, here body size is evaluated in the context of predation risk—e.g., larger body mass reduces predation risk. General life-history trends (i.e., a "slower" life history as body mass increases) predict a negative relationship between body size and growth rate, and a connection between greater body mass and lower predation would be expected to further reinforce the trend for larger-bodied primates to exhibit slower growth.

Ultimately, it is clear that many aspects of primate ecology are interrelated and codependent—group size and population density are linked to predation, but are also linked to diet and feeding competition (Janson & Goldsmith, 1995); predation is also linked to body size, and aspects of body size, predation, and diet all impact growth rates. Directional relationships are further complicated when brain size is also considered, as models of brain size evolution find that between-individual competition for resources decreases adult body mass because it reduces opportunities for an individual to extract energy from the environment and, thus, energy available for body growth (González-Forero & Gardner, 2018). Organisms and their ecologies are interconnected systems, and each trait (e.g., diet, competition, predation, etc.) influences others beyond just growth, reinforcing how important it is to evaluate the relative influence of each in a holistic way. These multi-faceted relationships between and interdependence of many aspects of primate biology necessitates a multivariate analysis, which will permit an evaluation of the relative effect (and direction of effect) of the multiple variables proposed to structure growth rate across Primates.

As this study explores the drivers of growth rate variation, it is essential to recognize that growth is a complex process that is not easily tied to a single underlying variable; many of the selective pressures and processes proposed to structure growth biology are not easily distilled into simple variables that are amenable to statistical analyses. The variables used here are proxies, and some variables are linked to more than one proposed selective pressure, which may make it difficult to infer which evolutionary process is at play. Some variables (e.g., body mass) are keystones that sit at the center of the suite of characteristics that define a species and, as a result, have underlying

(potentially confounding) linkages to other variables such as group size, predation, and diet. The variables included here—body mass (BM), brain mass (BrM), percentage of leaves in the diet (% lvs), CV of NDVI (NDVI), group size (GrpSz), substrate use (SS)—were selected based on theoretical expectations that they can reasonably serve as proxies for selective pressures. Interpretations of which selective pressure these factors best capture should be carefully considered. Independent of their links to selective pressures, however, understanding the relationships between these variables and their relative influence on growth rate is valuable in and of itself.

Analytical methods

The interspecific correlation between sella turcica size and growth rate was evaluated using multiple phylogenetic generalized least squares (PGLS) regressions for the male subsample, female subsample, and pooled male and female sample. Phylogenetic comparative methods such as PGLS account for the statistical non-independence of observations when species, which are linked by shared ancestry, are used as input data (Freckleton et al., 2002; Garland & Ives, 2000; Nunn, 2011; Revell, 2010). Patterns of growth vary between strepsirrhines and haplorhines (Mumby & Vinicius, 2008), so the two suborders were analyzed separately, as well as together as components of the full primate-wide dataset. The haplorhine sample was further subdivided, with catarrhines also analyzed separately. As both body mass and brain mass are linked to growth rates and sella turcica size, these variables were included with sella turcica volume as predictors in multiple regression models of growth rate. Though multiple regressions are often preferred over residuals as a method to control for

confounding variables in regression models (Freckleton, 2002), the residuals of the linear regression model of sella turcica volume against body mass were also used as a predictor of growth rate as an alternative method to account for the relationship between body mass and sella turcica size. AICc values were used to evaluate best-fitting models (Burnham & Anderson, 2002) of overall growth rate (OGR; between birth and sexual maturity) for males, females, and the pooled sex sample. Body mass, brain mass, and sella turcica volume were log-transformed to meet statistical assumptions of normality. Phylogenetic analyses were performed using the *caper* (Orme, 2018) and *phytools* (Revell, 2012) packages in R version 4.0.2 (R Core Team, 2020). Phylogenetic tree data were downloaded from 10kTrees (Arnold et al., 2010) at www.10ktrees.fas.harvard.edu.

The relationship between OGR and WRG and socioecological variables was also tested using PGLS multiple regressions. As multi-collinearity between predictor variables in multiple regressions can produce unreliable parameter estimates (Quinn & Keough, 2007), variance inflation factors (VIFs) were evaluated. VIFs can range from 1 to infinity and reflect the degree to which variance in estimated parameters is inflated by collinearity between predictor variables. The rule of thumb diagnoses multi-collinearity with a VIF>5, although some statisticians consider a VIF of 10 the maximum upper cut-off (Quinn & Keough, 2007). Ecological variables exhibit low multicollinearity (Table 4.3), but because brain mass and body mass approach the upper acceptable threshold

Table 4.3. Variance inflation factors

BM	BrM	NDVI	% leaves	substrate	group size
9.55	9.72	1.16	1.36	1.83	2.22

(Table 4.3), separate PGLS multiple regression models were run with either BrM or BM as predictor variables, as well as with both BrM and BM as predictor variables. Given the

multiple influences of each variable, PGLS models also explored the interaction effects between group size, NDVI variation, body mass, brain mass, and percent leaves.

Because the purpose of this analysis was to assess the relative influence of each variable on growth rate, predictor variables were scaled and centered to "standardize" them so that the units of the regression coefficients are the same. Body mass, brain mass, OGR, and TGR were log-transformed prior to analysis to meet statistical assumptions of normality. As in analyses of sella turcica size as a predictor of growth rate, AICc was used to evaluate best-fitting models (Burnham & Anderson, 2002). PGLS regressions of OGR and WGR against socioecological predictor variables were repeated with sella turcica volume as the dependent variable to assess whether sella turcica size is influenced by the same ultimate selective pressures that are hypothesized to structure growth rate.

4.3. Results

Sella turcica volume and growth rate

Models that separated analyses by sex revealed the same general trends as the pooled sex models, so results for the full, mixed-sex sample are presented here to maximize sample size and facilitate the application of predictive models to mixed or unknown-sex samples. The results of male- and female-specific analyses are presented in Appendix C (Table S4.2, Table S4.3). Generally, models that included sella turcica size and a measure of body size (either body mass or brain mass) were the best predictors of overall growth rate between birth and maturity (Table 4.4). Across the order Primates, there is not a significant relationship between sella turcica volume and growth rate in best-fitting multiple regression models that include body mass and brain mass as co-

predictors (see Table 4.4 “order Primates” models with lowest ΔAICc). Although the full model *OGR ~ ST volume + BM + BrM* is the best-fitting model as indicated by AICc values (ΔAICc = 0) and has the highest adjusted R^2 estimate (0.94), sella turcica volume is not a significant predictor in the model. The residuals of sella turcica volume against body mass are similarly not a significant predictor of overall growth rate for the full primate sample (Table 4.4 “order Primates, OGR ~ ST residuals” model; Figure 4.1). Sella turcica volume, both as a co-predictor and as residuals, is similarly a poor predictor of growth rate among strepsirrhines (Table 4.4 “suborder Strepsirrhini”). It could be that

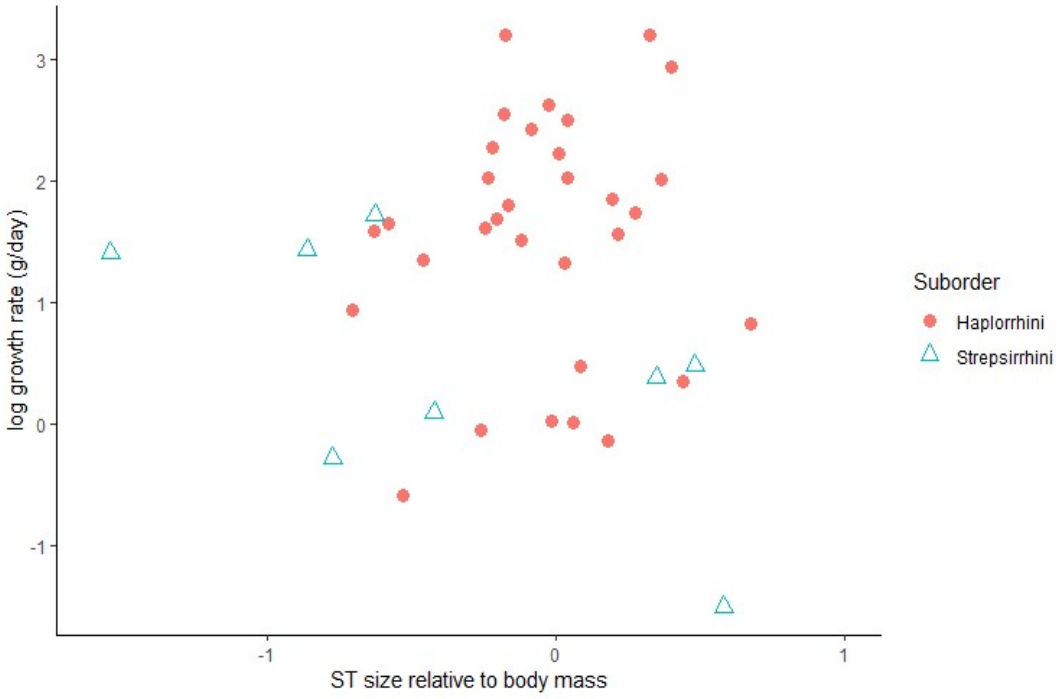


Figure 4.1. Total growth rate plotted against the residuals of sella turcica (ST) volume against body mass for the full primate sample.

Table 4.4. Parameter estimates for PGLS regressions of overall growth rate (OGR) against sella turcica size, body mass, and brain mass across different primate groups. Significant predictors at the alpha = 0.05 level are bolded; significance codes: 0.001***, 0.01**, 0.05*; -- predictor not included in model.

	model	std. beta coefficient						
	OGR ~	ST	BM	BrM	AICc	ΔAICc	adj. R^2	p-value
order Primates	ST + **BM** + **BrM**	0.01	**1.25*** **	**-0.78*** **	14.95	0	0.94	<0.001
	BM	--	**0.76*** **	--	23.02	8.07	0.81	<0.001
	ST + **BM**	0.09	**0.69*** **	--	24.24	9.29	0.81	<0.001
	ST + **BrM**	**0.41****	--	**0.43***	46.66	31.71	0.67	<0.001
	BrM	--	--	**0.85*** **	54.54	39.59	0.61	<0.001
	ST residuals	0.26	--	--	89.71	74.76	0	0.27
suborder Strepsirrhini	ST + **BM**	-0.22	**0.86*** **	--	4.11	0	0.94	<0.001
	BM	--	**0.75*** **	--	4.74	0.63	0.92	<0.001
	ST + BM + BrM	-0.27	1.73	-1.17	4.89	0.78	0.93	0.003
	BrM	--	--	**1.03*** **	5.88	1.77	0.91	<0.001
	ST + **BrM**	-0.15	--	**1.14****	6.88	2.77	0.91	0.001
	ST resid	-0.17	--	--	26.60	22.49	0	0.80
suborder Haplorhinni	**ST + BM + BrM**	**0.26***	**0.85*** **	**-0.55****	1.49	0	0.85	<0.001
	ST + BM	**0.27***	**0.48****	--	8.85	7.36	0.81	<0.001
	BM	--	**0.76*** **	--	12.08	10.59	0.78	<0.001
	ST + BrM	**0.59*** **	--	0.08	20.13	18.64	0.73	<0.001
	BrM	--	--	**0.80*** **	40.14	38.65	0.50	<0.001
	ST residuals	**0.74****	--	--	51.65	50.16	0.27	0.001
parvorder Catarrhini	**ST + BM**	**0.23***	**0.61****	--	-1.40	0	0.84	<0.001
	ST + BM + BrM	**0.23***	**0.74****	-0.23	-0.24	1.16	0.84	<0.001
	BM	--	**0.89*** **	--	0.17	1.57	0.82	<0.001
	ST + BrM	**0.49****	--	0.36	7.58	8.98	0.76	<0.001
	BrM	--	--	**1.07*** **	17.46	18.86	0.60	<0.001
	ST residuals	**0.76****	--	--	27.53	28.93	0.36	0.002

the weak, nonsignificant negative relationship between sella turcica size in strepsirrhines is driving the lack of significance in the full primate model, as the underlying biological relationship between sella turcica size and growth appears to be different in strepsirrhines compared to haplorhines (Figure 4.1).

Among haplorhine primates, sella turcica volume is significantly positively correlated with growth rate in both multiple regression models and residual-based models (Table 4.4; Figure 4.2). The best fitting model of haplorhine total growth rate includes

sella turcica volume, brain mass, and body mass as predictors (R^2 = 0.85; next-best fitting model ΔAICc = 7.36); sella turcica volume and body mass are positively correlated with growth rate, while brain mass is negatively correlated with growth rate (Table 4.4). Models of haplorhine growth rate that include sella turcica volume as a co-predictor, in general, fit better than models that employ body mass or brain mass without considering sella turcica volume, as indicated by ΔAICc and R^2 values. Plots of total growth rate against the residuals of sella turcica size against body mass indicate a grade-shift between catarrhines and platyrrhines, with all platyrrhines except for the three atelid species exhibiting slower growth rates than catarrhines, despite similar sella turcica volumes relative to body mass (Figure 4.2).

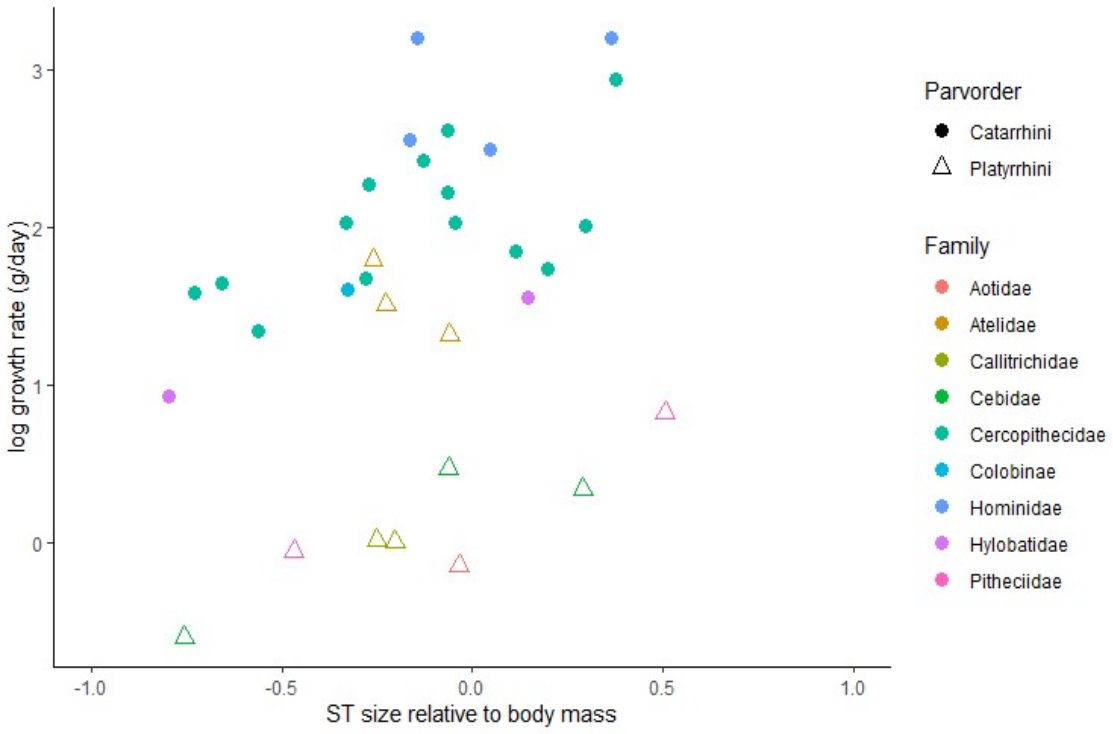


Figure 4.2. Total growth rate plotted against the residuals of sella turcica (ST) volume against body mass for the haplorhine primate sample.

Among catarrhines, there is also a strong positive correlation between sella turcica volume and growth rate (Table 4.4; Figure 4.3). Within this clade, models that include sella turcica volume are generally better fitting models of growth rate than models that only include body mass or brain mass, although the *OGR* ~ *BM* model has a ΔAICc < 2, suggesting that it is an equally good fit as the *OGR* ~ *BM* + *ST* and *OGR* ~ *BM* + *BrM* + *ST* models. Models that include sella turcica volume, however, do have slightly higher adjusted R^2 values than models that do not. Though *OGR* ~ *ST* + *BrM* models have weaker fit than models that include BM for the full haplorhine and catarrhine samples, ST volume has the strongest effect on growth rate in these models; this is likely because ST volume captures aspects of body size, which is a consistently strong predictor of growth rate in all models across taxonomic level (Table 4.4).

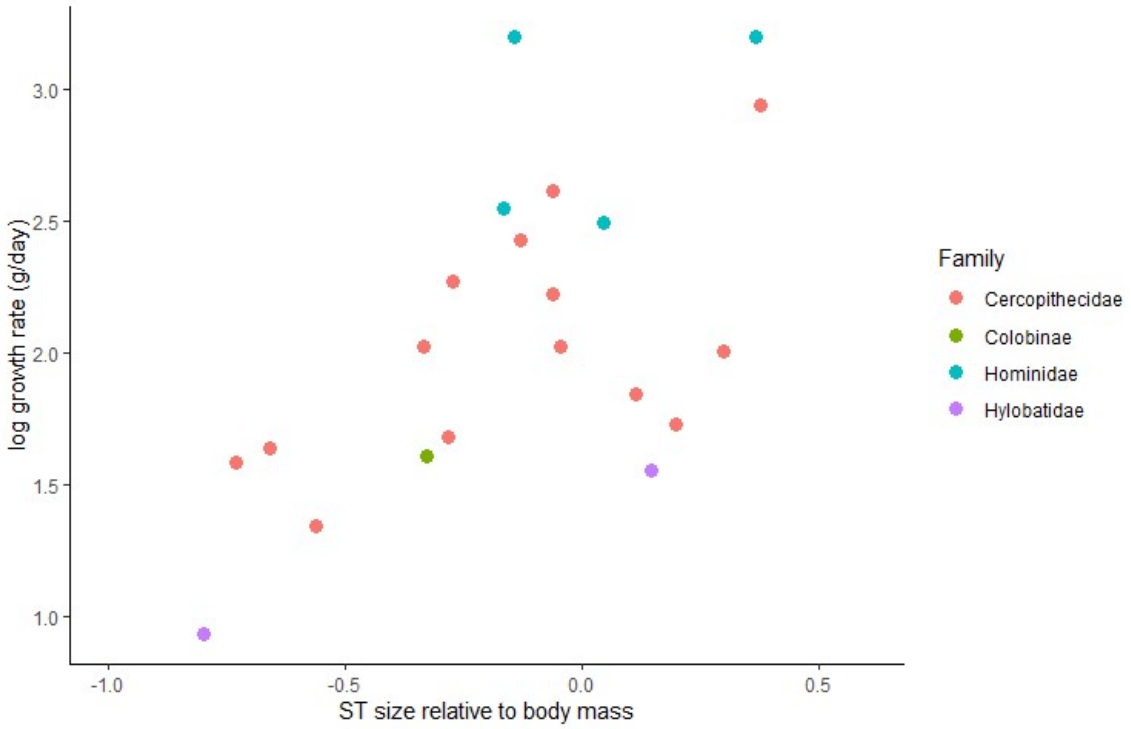


Figure 4.3. Total growth rate plotted against the residuals of sella turcica (ST) volume against body mass for the catarrhine primate sample.

Socioecological variables

Multiple PGLS regression models of both pre-weaning growth rate (WGR; Table 4.5) and overall growth rate (OGR; Table 4.6, Table S4.4, Table S4.5) reveal that body mass and/or brain mass are most strongly correlated with growth rate. Best-fitting multiple PGLS regression models of pre-weaning growth rate rarely included body mass and brain mass in the same model, so all results are presented in Table 4.5. Separately, body mass and brain mass were equally good predictors of pre-weaning growth rate, as indicated by each size variable (i.e., brain mass or body mass) on its own being among models with a ΔAICc < 2; when included, the coefficient of variation in NDVI was significantly (or nearly significantly) negatively correlated with pre-weaning growth rate in these models (Table 4.5). Interaction effects between ecological variables in models of pre-weaning growth rate were non-significant (interaction term p-values = 0.17 to 0.41).

Table 4.5. Parameter estimates for PGLS regressions of pre-weaning growth rates (n=30) against socioecological predictor variables. Significant predictors at the alpha = 0.05 level are bolded; significance codes: 0.001***, 0.01**, 0.05*, 0.10^; -- predictor not included in model.

	standardized beta coefficient								linear model	
model WGR ~	BM	BrM	NDVI	% lvs	GrpSz	SS	AICc	Δ AICc	adj. R^2	p-value
BM + NDVI	**0.41***	--	-0.29^	--	--	--	26	0	0.58	0.006
BrM + NDVI	--	**0.40***	**-0.31***	--	--	--	26.5	0.48	0.56	0.007
BM	**0.49****	--	--	--	--	--	26.6	0.61	0.44	0.008
BrM	--	**0.47***	--	--	--	--	27.5	1.47	0.40	0.01
NDVI	--	--	**-0.39***	--	--	--	30.2	4.23	0.26	0.04
BM + NDVI + SS	**0.39***	--	**-0.34***	--	--	-0.13	30.4	4.41	0.57	0.01
BM + GrpSz	**0.48***	--	--	--	0.04	--	30.9	4.88	0.38	0.04
BM + % lvs	**0.49***	--	--	-0.03	--	--	30.9	4.89	0.38	0.04
BM + SS	**0.48***	--	--	--	--	-0.01	30.9	4.94	0.38	0.04
BM + BrM	0.46	0.02	--	--	--	--	30.9	4.94	0.38	0.04
BrM + NDVI + SS	--	**0.38***	**-0.36***	--	--	-0.13	31.0	4.99	0.55	0.02

Table 4.6. Model selection overall growth rate including both brain mass and body mass as predictors (n = 68 species). Significance codes: 0.001***, 0.01**, 0.05*, 0.10^; -- predictor not included in model.

	standardized beta coefficient							linear model		
model OGR ~	BM	BrM	NDVI	% lvs	GrpSz	SS	AICc	Δ AICc	adj. R^2	p-value
BM + BrM + NDVI	**1.22*** **	**-0.53*** **	**-0.08***	--	--	--	-3.4	0	0.94	<0.001
BM + BrM + NDVI + SS	**1.20*** **	**-0.51*** **	**-0.08***	--	--	0.04	-1.4	2.01	0.94	<0.001
BM + BrM	**1.20*** **	**-0.49*** **	--	--	--	--	-1.4	2.04	0.93	<0.001
BM + BrM + NDVI + GrpSz	**1.21*** **	**-0.51*** **	**-0.09***	--	-0.03	--	-0.08	2.58	0.94	<0.001
BM + BrM + NDVI + % lvs	**1.25*** **	**-0.56*** **	**-0.08***	-0.02	--	--	-0.07	2.75	0.94	<0.001
BM + BrM + SS	**1.18*** **	**-0.47*** **	--	--	--	0.04	0.4	3.81	0.93	<0.001
BM + BrM + NDVI + % lvs + SS	**1.24*** **	**-0.54*** **	**-0.08***	-0.03	--	0.04	1.3	4.69	0.94	<0.001
BM + BrM + % lvs	**1.22*** **	**-0.51*** **	--	-0.02	--	--	1.4	4.78	0.93	<0.001
BM + BrM + GrpSz	**1.20*** **	**-0.48*** **	--	--	-0.01	--	1.5	4.87	0.93	<0.001

In multiple regression models of overall growth rate that include both body mass and brain mass as predictors, body mass is positively correlated with growth rate, while brain mass is negatively correlated with overall growth rate (Table 4.6). More conservative multiple regression models that included only body mass or only brain mass are presented in Appendix C (Table S4.4, Table S4.5). In these models, growth rate is positively correlated with both brain mass and body mass, likely because brain mass captures variation in body size when it is analyzed independently of body mass. Best-fitting multiple PGLS regression models of overall growth rate reveal that the coefficient of variation in NDVI is significantly negatively correlated with growth rate (i.e., higher seasonal variation in vegetation is associated with a slower growth rate), although the effect of NDVI variation on growth rate is much smaller than those of body mass and brain mass (Table 4.6).

Interaction effects between ecological variables, body mass and brain mass for all models were non-significant. Interaction term p-values for the best-fitting *OGR ~ BM + BrM + NDVI* model ranged from 0.36 to 0.97, while p-values for the *OGR ~ BM + BrM + NDVI + SS* model ranged from 0.18 to 0.97. Interaction term p-values for all top-fitting models (i.e., those with ΔAICc < 4) ranged from 0.17 to 0.99. These non-significant interaction terms indicate that the effects of a variable (NDVI, for example) on growth rate are not different for different values of another (BrM, for example).

When the full primate sample was subdivided into strepsirrhine, platyrrhine, and catarrhine subsamples (Appendix C; Table S4.6, Table S4.7), models of both pre-weaning and overall growth rates had lower predictive power and fewer independent variables were significant, likely as a product of reduced sample size. General trends largely mirror those identified in the full sample, so results by taxonomic group are presented in Appendix C. As no predictor variables were statistically significantly correlated with sella turcica size in these analyses of taxonomic (suborder or parvorder) subsamples, the results of PGLS multiple regressions of sella turcica volume against socioecological variables are only presented in Appendix C.

4.4. Discussion

Sella turcica volume as a predictor of growth rate

This study reveals that models that include sella turcica volume as a co-predictor are better predictors of growth rate than models that employ body mass or brain mass alone, particularly among haplorhine primates (Table 4.4). Given the strength of body mass and brain mass as predictors of growth and life history, it is informative that an

aspect of anatomy that is a component of the proximate system that modulates growth increases the strength of models that include brain and/or body mass, and is promising for the sella turcica's utility as a predictor of growth rate in primate species represented by skeletal material, especially for catarrhine (and other haplorhine) species. Although the relationship between sella turcica volume and growth rate across Primates was not significant, models that included sella turcica exhibited better fit and higher R^2 values than order-wide models that did not (Table 4.4). The non-significance of sella turcica size as a predictor in full-order models that include both haplorhines and strepsirrhines is likely due to differences in growth between these two suborders (Kirkwood, 1985; Leigh & Terranova, 1998; Mumby & Vinicius, 2008; Vinicius & Mumby, 2013). Growth rates in strepsirrhines have been observed to vary much more around the primate mean compared to growth rates in haplorhines, with some strepsirrhine clades containing species that are characterized by fast and slow growth (Mumby & Vinicius, 2008); thus, strepsirrhine ranges of variation may obscure the "typical" primate trends driven by haplorhines. In the strepsirrhine sample included in this study, there appears to be loose negative correlation between growth rate and sella turcica size (Table 4.4, Figure 4.1), although it is not statistically significant. A negative correlation between sella turcica size and growth rate across species was unexpected, so it would be prudent to expand the strepsirrhine sample to further explore this relationship and construct more robust tests of whether there are true subordinal differences in the relationship between sella turcica size and growth rate.

The significant positive relationship between sella turcica size and growth rate in haplorhine primates likely reflects the functional relationship between hormones

produced by the pituitary gland (which is housed by the sella turcica) and somatic growth[9]. As outlined in Chapter 1, section 1.3, the pituitary gland is a critical component of the hypothalamic-pituitary-somatotropic (HPS) axis that produces growth hormone (GH) and insulin-like growth factor 1 (IGF-1), which together promote bone, muscle, and brain growth (Liu et al., 1993; Webb et al., 2012). As the scope of this project did not permit an exploration of the causal mechanisms that underpin correlations between sella turcica size and growth rate, direct investigation of whether larger sella turcica and pituitary sizes are consistently linked to higher GH and IGF-1 concentrations across primate species would be a valuable avenue for future research.

Ultimate drivers of growth rate variation

These results suggest that across primates, body mass and brain mass are the two strongest predictors of variation in growth rate, both between birth and weaning and between birth and sexual maturity (Table 4.5, Table 4.6). This is perhaps unsurprising, as body and brain mass are strong predictors of many life-history traits and the overall pace of life (e.g., Jones, 2011; Leigh, 2004; Stearns, 1976, 1983; Street et al., 2017; Western & Ssemakula, 1982). Conversely, the positive correlation between body mass and growth rate in models that include both body mass and brain mass as co-variates was somewhat unexpected. This suggests that when the effect of brain mass, which is negatively correlated with growth rate, is considered, larger body mass is accompanied by a faster growth rate. This was somewhat contrary to predictions (as larger body size is typically

[9] Of course, the same functional relationship between pituitary hormones and somatic growth exists in strepsirrhines, but the nature of strepsirrhine growth appears to complicate the hypothesized positive relationship between growth rate and sella turcica size.

hypothesized to be associated with a slower life history and thus, slower growth) but makes biological sense. Given the range of body masses found across the primate clade (Fleagle, 1985; Smith & Jungers, 1997) and constraints on what constitutes a reasonable length of time for a growth period (e.g., a 100 kg primate that lives for 35 years cannot spend 25 years growing, as it might if it were to grow at the same rate as a 10 kg primate), it is not surprising that larger-bodied species would need to grow faster than smaller-bodied species, even if they also grow for a longer period of time (Leigh, 2001; Schultz, 1969). Though the study was limited to one suborder, there is some evidence of a positive association between higher postnatal litter growth rate (rather than individual growth rate) and body mass in strepsirrhines (Kappeler, 1996). While some species' larger body sizes may be achieved by growing for longer, rather than faster, compared to other species (Stearns, 1992), these results help to confirm that interspecific differences in body size are at least partly the product of differences in growth rate, with faster growth associated with larger body mass.

The significant negative correlation between brain mass and growth rate (when body mass is included as a covariate) is consistent with previous research suggesting that large brains act as an "anchor" that favors slow growth and more generally, a slower pace of life (Charnov, 1993; Foley & Lee, 1991; Kuzawa et al., 2014; Leigh & Blomquist, 2007; Martin, 1996; Navarrete et al., 2011). The generally slower life histories of primates compared to many other mammalian groups (Harvey et al., 1987; Harvey & Clutton-Brock, 1985), and slower life histories of larger-brained primates compared to smaller-brained ones (Barrickman et al., 2008; Deaner et al., 2003; Harvey & Clutton-Brock, 1985; Leigh, 2004), have been proposed to be the result of mutually reinforcing

processes and feedback loops (Street et al., 2017) that permit and encourage the development of technological, cultural, and social skills. These results therefore have implications for understanding the evolution of large brains, as well as the emergence of technological and cultural developments (such as complex tool use, innovation, and cumulative culture) that are hypothesized to be linked to (if not made possible by) the large brains and extended life history that characterize primates and, in particular, modern humas (Barrickman et al., 2008; Navarrete et al., 2016; Reader & Laland, 2002; Schuppli et al., 2016; Sol et al., 2016; Street et al., 2017; Walker et al., 2006). Although causal links can be hard to establish using comparative methods, and it is difficult to conclude whether increases in brain size contributed to the slowing of growth rates or whether slower growth caused by external selective pressures permitted the evolution of large brains, these results support the prevailing hypothesis that large brains are associated with slow growth, likely within a feedback loop by which slow growth allows the development of large brains, which further encourage slow growth. As other large-brained mammalian (Marino et al., 2007; Patterson & Mann, 2011; Sol et al., 2008) and avian species (Lefebvre, 2013; Lefebvre et al., 2004; Overington et al., 2009; Sol et al., 2007) also exhibit innovative behaviors and technological skill, it would be interesting to further explore the nature of links between large brains, slow growth, and innovation and skill in non-primate taxa (e.g., Barton & Capellini, 2011; Bennett & Harvey, 1985; Sacher & Staffeldt, 1974).

As the focus of this study was not to test correlates of brain size, its multidimensional framework did not explicitly test whether primates with larger brain sizes are also characterized by longer growth and developmental periods, but links

between larger brain size, longer growth periods, and the pace of growth suggest a number of interesting avenues for future research. Recent work on the development of manipulation complexity suggests that there are likely developmental constrains on the evolution of highly complex manipulation skills that take a long time to acquire (Heldstab et al., 2020) such that the evolution of complex activities such as tool use is only possible for species with long growth and/or developmental periods that provide them enough time for social transmission and learning. Heldstab et al. (2020) do not propose how much time is "enough time," but life-history theory predicts that the elongation of growth and/or juvenile periods would only occur if adult survival and reproduction is increased sufficiently to outweigh its costs (e.g., Barrickman et al., 2008). It would therefore be interesting to test whether primate species with absolutely and/or relatively prolonged growth and developmental periods consistently use more complex skills and whether they enjoy increased survival and/or reproductive success relative to those that do not, as well as whether complex skills are linked to the pace of growth. Furthermore, given the links between environmental and ecological conditions such as seasonal variation in vegetation (i.e., the coefficient of variation of NDVI) and growth and developmental trajectories (Table 4.6), it would be interesting to use phylogenetically informed multivariate methods to test which environmental factors facilitate the evolution of the sort of prolonged development that accompanies complex manipulative skills.

Alternatively, the positive correlation between body mass and growth rate, but negative correlation between brain mass and growth rate may be a spurious result attributable to the inclusion of both brain mass and body mass as co-predictors in models. As brain mass and body mass exhibit a degree of collinearity that approaches the upper

acceptable limit (Table 4.3), these parameter estimates may be somewhat unreliable (Quinn & Keough, 2007) and should be interpreted with some degree of caution. Body mass, however, is also positively correlated with growth rate in models that do not include both brain and body mass as covariates (Table S4.4), suggesting that increased body mass is indeed linked to increased growth rate. Models that only include brain mass reveal a positive correlation between brain mass and growth rate (Table S4.5), although this result is likely due to the confounding effects of body size, as body mass and brain mass are strongly positively correlated.

This study reveals that body mass and brain mass are the strongest predictors of both pre-weaning and overall growth rate across the primate order, but it also provides modest support for the hypothesis that interspecific variation in growth rates is linked to environment and ecology. The coefficient of variation of NDVI, a measure of seasonal variation in vegetation, is consistently negatively correlated with both measures of growth rate investigated here. This is consistent with the predictions of the ERAH (Janson & van Schaik, 1993), which suggests that the unpredictable or seasonal availability of food resources favors slower growth as a mechanism to mitigate the risk of starvation. The CV of NDVI, however, only captures the potential for seasonally driven variation in food availability and ignores other sources of variation in access to resources (i.e., those linked to social group size and/or resource spatial distribution). In future investigations, it might be useful to include food patch density as a metric that captures the potential for food availability and competition, as food resources that occur in large patches or at high density are expected to correlate with lower intragroup feeding competition (McFarland Symington, 1988).

Although the ERAH connects the potential for limited resource access to slow growth, environmental unpredictability or uncertainty can favor the evolution of various types of "bet-hedging" strategies (Dewar & Richard, 2007; Ellis et al., 2009; Richard et al., 2002; Stearns, 1976) and has been argued to result in the evolution of both fast and slow growth in different species (Mumby & Vinicius, 2008). Deeper exploration of the influence of specific environmental conditions on growth may reveal divergent underlying patterns that are obscured by the broad trends identified here (i.e., increased environmental variation is linked to slower growth). While percentage of leaves in the diet (used as a proxy for the abundance and availability of food resources) would be predicted to be correlated with faster growth under an ecological risk aversion framework, it was not significantly correlated with growth rate in best-fitting models. This may be an artifact of data quality or the primate sample used, or may simply mean that the differences in growth rates between primate species that typically consume different proportions of leaves are not robust enough to persist when other ecological variables are also considered. While leaves are typically thought to be lower in quality than fruits, it is worth noting that many folivorous primates selectively choose nutrient and protein-rich young leaves, which may provide them with a diet comparable in quality to one that heavily emphasizes fruits (Masi et al., 2015; Snaith & Chapman, 2007).

Variable choice may also explain the weak, non-significant correlations between growth rates and both group size and substrate use. In particular, the proxies for predation risk (substrate use and group size) are crude and may not accurately capture the real risk faced by primate populations in different ecological settings. More nuanced characterizations of predation risk, such as those based on the potential for and frequency

of predator-prey interactions (e.g., Hill & Lee, 1998) may be a better metric, but unfortunately such data were not available for the wide range of species included in this study. Furthermore, although there is a difference between predation risk and predation rate (Hill & Dunbar, 1998), this study only used proxies that were proposed to capture predation risk. Each is proposed to have distinct, yet related, impacts on a species' biology (Hill & Dunbar, 1998), so investigation of the influence of predation rate on growth rate may also be lucrative. Because individuals and populations respond to both unsuccessful and successful attacks, Hill and Dunbar (1998) argue, current antipredation behavior is driven by predation *risk*, but it may be that past and/or present predation *rates*, since they reflect predation that can't be adjusted for by behavioral changes, act as stronger selective pressures on life history and growth rates. Hill and Dunbar (1998) contend that animals try to reduce predation risk so that predation rate is maintained at an "acceptable" level (i.e., a level that prevents a catastrophic population crash), so individuals and populations that are able to maintain higher reproductive rates—perhaps through faster growth rates and earlier onset of sexual maturity—can tolerate higher levels of mortality (Hill & Dunbar, 1998). Although scenarios of life history evolution typically imply that the evolution of a slow life history is only possible if extrinsic mortality is low enough to permit delayed reproduction (e.g., Bribiescas, 2020; Charnov & Berrigan, 1993), one could also flip the direction of this relationship and argue that it is not predation rate that shapes life history but rather, life-history trajectories that establish what level of predation rate is tolerable (Hill & Dunbar, 1998). Most life-history researchers propose that extrinsic mortality structures variation in growth and life history; under this alternative framework, species that grow and reproduce more slowly would be

forced into niches with antipredator strategies that reduce their predation rates. Thus, one could argue that while predation risk shapes defense strategies (Hill & Dunbar, 1998; Hill & Lee, 1998), predation rate is related to reproductive and life-history traits; future research may benefit from the inclusion of measures that reflect actual instances of primate mortality due to predation across population.

The relationships between aspects of ecology and growth rate identified here encourage further exploration of the causal links between ecology and growth rates, especially as continued fieldwork contributes additional data on growth in wild populations. Because mortality rates are proposed to be linked to differences in lifespan between conspecific males and females (Lemaître et al., 2020), it would be interesting to explore sex-specific differences in the pace of growth in a wide range of species. Male and females exhibit divergent growth patterns in a number of primate species, particularly those that are sexually dimorphic in body size (e.g., Altmann & Alberts, 2005; Leigh, 1992, 1995; Leigh & Terranova, 1998), but, unfortunately, this study was not able to obtain sex-specific growth data for a the same wide range of species for which other ecological data were available. Furthermore, although growth can respond dynamically to contemporary variation in experienced environment, the selective pressures that shaped the primate growth rates that we observe today reflect past ecological conditions, so it may be that the ecological variables used here are not those for which a species' life-history strategy was selected (akin to the evolutionary disequilibrium proposed by van Schaik & Kappeler (1996) to explain lemur activity patterns). It would thus be valuable to investigate the relationship between historical ecological and environmental conditions and present-day growth rates.

It is also important to remember that interspecific comparisons necessitate the use of "average" trait values for each species, which can obscure intraspecific variation in these traits (e.g., Chapman, 1987; Chapman & Rothman, 2009; Cords, 1986; Palombit, 1997; Richard, 1974; Sandel et al., 2016; Yamagiwa et al., 2003). Variation or flexibility in traits or trait expression may be what selection targets or favors; in the case of diet, for example, this study utilized a single value for the percentage of leaves in the diet that was averaged across populations. This metric may not actually represent the dietary composition of any one population, as there can be substantial variation in the diet of wild primate populations, both temporally and spatially (e.g., Butynski, 1990; Chapman, 1987; Chapman et al., 2003; Chapman & Chapman, 1990; Harris & Chapman, 2007). Furthermore, contemporary primate populations may utilize unconventional or atypical resources as the result of human impacts on their habitats (e.g., Altmann & Muruthi, 1988; Behie & Pavelka, 2005; McKinney, 2011; Pozo-Montuy et al., 2013; Sengupta et al., 2015), and even if "ancestral" diets could be inferred, similar levels of intraspecific variation in diet should be expected.

Though many of the variables included in this study share an underlying connection to energy availability, this research was not able to study energy budgets directly and relied on metrics such as diet quality as rough approximations for energy availability. The concept of energetic trade-offs is a key aspect of life-history theory (Bogin, 2012; Jones, 2011), and energy budgets have been proposed to be central to the evolution of modern human features such as a large brain and high reproductive output coupled with slow growth (e.g., Gurven & Walker, 2006; Pontzer, 2017; Pontzer et al., 2014, 2016; Rasmussen & Izard, 1988). Future investigations that are able to directly

measure the energetic inputs and expenditures across a range of primate species (particularly wild populations) and explore how energetic differences translate into different growth rates would shed light on the mechanistic energetic trade-offs that are hypothesized to underlie the evolution and development of life-history trajectories, as well as linked traits such as brain size. The value of an energetics-based perspective is that it has the potential to unite many of the traits explored here. Though the selective pressures that are proposed to have shaped life history and growth can be grouped into various categories (e.g., those that are linked to diet vs predation, those that are density dependent), selective pressures and resulting traits do not function independently of each other. Ultimately, this study reinforces the idea that the evolution of primate growth rates, like the evolution of all aspects of life history, is the result of selection on suites of interrelated traits that act in concert as the parts of a functioning whole. Though some aspects of ecology and environment, such as seasonal variation in vegetation, impact the pace of growth, these results are consistent with previous research that places body mass and brain mass as key determinants of growth and developmental trajectories. It also reveals a relatively strong positive relationship between sella turcica size and growth rate among haplorhine primates, which suggests that the size of the sella turcica may have utility as a proxy for reconstructing growth rate in some fossil primate species.

CHAPTER 5

DISCUSSION

5.1. Summary of results

This dissertation built upon (1) the mechanistic connections between pituitary hormones and growth and (2) the anatomical relationships of the pituitary gland to explore a potential hard tissue correlate of primate growth rate—the size of the bony structure within which the pituitary sits, the sella turcica. By studying the strength of correlations between sella turcica volume and growth rate both interspecifically and intraspecifically, it tested whether the size of the sella turcica could serve as a proxy for growth rate both across and within primate species. It also investigated the links between growth rate and a suite of socioecological variables to (1) establish how local environment affects growth within a population and (2) explore the relative roles of different selective pressures in shaping growth rates across species.

Chapter 2 took an intraspecific perspective, testing whether the size of the sella turcica tracks growth rate throughout ontogeny in the Cayo Santiago *Macaca mulatta* population. Though a weak correlation between growth rate and sella turcica size was found across all study individuals, the strength and direction of the relationship suggest that sella turcica size is not a valid proxy for growth rate within this cercopithecid population. Chapter 3 then explored the effects of local environmental variables on growth within the *M. mulatta* population. It found differences in the trajectory of growth and attained body size among female subadults who experienced different conditions during the prenatal and/or early postnatal period, suggesting that the pattern and pace of

growth can respond dynamically to the specific conditions experienced by an individual early in life.

Chapter 4 moved beyond intraspecific comparisons to explore interspecific trends in patterns of growth and sella turcica size variation across the primate order. Chapter 4 tested the hypothesis that sella turcica size is positively correlated with postnatal growth rate across primates. It found support for this hypothesis, particularly among haplorhines. In the haplorhine sample and catarrhine subsample, models that included sella turcica volume were better predictors of postnatal growth rate than models that included only body and/or brain size. This suggests that sella turcica volume may provide a novel method to probe growth rate that is grounded in the proximate hormonal mechanisms that govern growth. As this method utilizes a hard tissue structure, it may be particularly valuable for reconstructing growth in species that are represented only by skeletal material. Chapter 4 also explored the relative influence of various socioecological selective pressures on interspecific variation in growth rate. Consistent with much of the classic life-history literature (e.g., Charnov, 1991, 1993; Charnov & Schaffer, 1973; Jones, 2011; Promislow & Harvey, 1990), it found that body mass and brain mass are the strongest predictors of differences in this aspect of growth and development. Results also revealed that seasonal variation in available vegetation, a proxy for diet seasonality, was a significant predictor of growth rate across species, with slower growth linked to greater variability in diet. This is consistent with the predictions of the ecological risk aversion hypothesis (Janson & van Schaik, 1993).

Differences between the intraspecific relationship (Chapter 2) and interspecific relationship (Chapter 4) of sella turcica size to growth rate highlight that interspecific

patterns need not mirror intraspecific ones. The significant positive correlation between sella turcica volume and growth rate in the interspecific sample, yet nonsignificant negative correlation in the intraspecific sample, could be because the intraspecific study only captures a small component of the overall variation (in both growth rate and sella turcica size) that is visible in the interspecific study. This could be a scenario related to Simpson's paradox (E. H. Simpson, 1951), where the positive relationship between population-level sella turcica size and population-level growth rate masks a more variable (potentially negative) relationship between sella turcica size and growth rate among individuals of the same population. Relatedly, the phenomenon could be a result of taxon-level effects akin to those that reveal different scaling relationships when the taxonomic level at which analyses are performed is shifted from higher levels (such as the class) to the order, suborder, parvorder, or family level (Pagel & Harvey, 1988, 1989). Different trends within and between populations could also be the product of clade-specific hormonal responses; perhaps *M. mulatta* (or cercopithecids as a group) are particularly susceptive to early life challenges and stress effects (Chapter 2, section 2.4), which thus drives a unique negative relationship between growth rate and pituitary size, and thus sella turcica size, in this group.[10]

Overall, while this dissertation revealed that sella turcica volume is not a reliable predictor of growth rate within the *M. mulatta* population studied here, it suggests that the sella turcica may have utility as a predictor of growth rate at the interspecific level. The

[10] It is also possible that accurate growth rates simply could not be calculated for individuals in the Cayo Santiago population; further analyses with more detailed body mass data could reveal a positive relationship between growth rate and sella turcica size similar to that observed at the interspecific level.

strength of the relationship between sella turcica size and growth rate among haplorhine primates in particular holds promise for investigations of growth, development, and life history in the hominin fossil record and has the potential to contribute to our understanding of when and why aspects of modern human growth evolved. The next sections will thus briefly outline common methods employed to reconstruct growth and life history in the fossil record and will comment on how information provided by the sella turcica may complement current hypotheses about the pattern and pace of hominin growth and development.

5.2. Methods of life history and growth reconstruction in the fossil record

Given the unique aspects of human life history (as reviewed in Bogin, 1999b; Jones, 2011), paleoanthropologists have long been interested in understanding when and why key components of the modern human life-history profile evolved. Because life history does not fossilize, explorations of life history in the past must rely on proxies that facilitate the reconstruction of major growth and developmental milestones. Broad patterns among mammals allow one to roughly gauge age at first reproduction or adult lifespan based on biological traits such as body mass or brain size (e.g., Charnov & Berrigan, 1993; Harvey & Clutton-Brock, 1985), but the relatively tight, predictable relationship often weakens when comparing closely related species with similar body or brain size (Leigh & Blomquist, 2007; Purvis et al., 2003).

Paleoanthropologists have thus developed a number of methods to reconstruct life history in extinct species. The recognition that dental development tends to align with key life-history events (Schultz, 1935, 1960, 1969; B. H. Smith, 1989, 2000; B. H. Smith et

al., 1994) allows the timing of tooth formation and eruption sequences to provide a scaffold for reconstructing the overall pace of life in fossil specimens. In general, permanent incisors, canines, and premolars tend to emerge early compared to the molars in slow-growing, long-lived species (B. H. Smith, 2000) such as *Homo sapiens* (Godfrey et al., 2005; Liversidge, 2003; B. H. Smith, 2000). Some researchers (including Adolph Schultz, the first one to elucidate the connections between dental development and life history) have hypothesized that the protracted modern human dental eruption schedule is linked to our prolonged postnatal growth and long juvenile period (Robson & Wood, 2008; B. H. Smith, 2000). The development and emergence of teeth are linked to the timing of pre-reproductive life-history events and related milestones (B. H. Smith, 1989), with the emergence of the mandibular first molar (M1) broadly correlated with age at weaning (B. H. Smith, 1989), the masticatory competency that accompanies weaning (Sardi & Rozzi, 2007), and the cessation of brain growth (Dean, 2006; Macho, 2001; B. H. Smith & Tompkins, 1995). Later in life, age at mandibular third molar emergence is loosely correlated with age at sexual maturity across primates (Dean, 2006; B. H. Smith, 1989; Watts, 1990).[11] As a result, the timing of molar emergence (particularly that of M1)

[11] Despite broad correlations between emergence ages and life history across Primates, it is worth pointing out exceptions to rule. For example, M1 emerges on average before weaning in *Gorilla* (Macho & Lee-Thorp, 2014; B. H. Smith et al., 1994), but relatively late in some *Pongo pygmaeus* individuals, ca. 4.6 years (Kelley & Schwartz, 2010). This later age is generally compatible with *Pongo's* prolonged life history (Wich et al., 2004), but varying estimates of when *Pongo* species wean makes it difficult to evaluate when M1 emerges relative to weaning. If more historic estimates of weaning age (~3 years) (Dettwyler, 1995) are used, *Pongo* M1 emerges post-weaning. Conversely, more recent field studies have reported average weaning ages between 6.5 and 7 years (Van Noordwijk et al., 2013; Van Noordwijk & Van Schaik, 2005), which would imply that M1 emerges substantially pre-weaning. In *Pan*, there are some reports of M1 emerging, on average, before weaning (e.g., T. M. Smith et al., 2007; Zihlman et al., 2004), although more recent fieldwork at Kanyawara suggests that molar emergence does not always coincide with the introduction of solid foods, as all focal individuals continued to nurse after mandibular M1 reached occlusion (T. M. Smith et al., 2013). These discrepancies are likely the

is frequently used to make inferences about the life histories of fossil hominins (e.g., Dean et al., 2001; Kelley & Schwartz, 2010, 2012; Kelley & Smith, 2003; López-Torres et al., 2015; Nargolwalla et al., 2005; B. H. Smith, 1994).

In addition to dental development, dental microstructure is also used to track the pace of life in fossil primates. Tooth crowns and roots form accretionally through the regular secretion of enamel and dentine crystalline matrices, resulting in the production of "short-period" and "long-period" lines (Bromage, 1991; Dean, 1987, 2000, 2006; Schwartz & Dean, 2000; T. M. Smith, 2006, 2008). Though there is inter- and intraspecific variation in the frequency with which long-period growth lines appear (FitzGerald, 1998), periodicity—that is, the number of daily short period lines between adjacent long-period lines—is constant within a single tooth and in all teeth of the same individual (Dean, 1987; Kelley & Schwartz, 2010; T. M. Smith, 2006; T. M. Smith et al., 2015). The known periodicities of dental microstructure features allow rates and durations of enamel and dentine formation to be calculated (Kelley & Schwartz, 2010). Because formation ceases at death, this enables counts of growth lines to reveal how much crown and/or root had formed when an individual died and can establish a calendar age-at-death (Kelley & Schwartz, 2010; T. M. Smith et al., 2006). This effectively means that dental hard tissues can function as individual clocks that can link dental eruption sequences and patterns to calendar ages in fossil individuals. Microstructure-based methods were a major methodological breakthrough, as they permit comparisons between the dental emergence statuses of fossil hominins, modern humans, and modern great apes

result of both inter-individual and inter-population (sometimes inter-specific) variation in life history and dental development (e.g., Liversidge 2003), as well as the fact that weaning is a process, not a single event.

of the same chronological age (Bromage & Dean, 1985). Previously, chronological ages of fossil specimens had to be estimated based on patterns of dental eruption in extant species or skeletal development standards (Boyde, 1990). Microstructure-based methods have been applied to australopiths (e.g., Bromage & Dean, 1985; Lacruz et al., 2008; T. M. Smith et al., 2015), *Homo erectus* (e.g., Dean et al., 2001), Neanderthals (e.g., Guatelli-Steinberg et al., 2005; Macchiarelli et al., 2006; Ramirez Rozzi & Bermudez De Castro, 2004; T. M. Smith et al., 2010; T. M. Smith, Toussaint, et al., 2007), and archaic *Homo sapiens* (T. M. Smith, Tafforeau, et al., 2007), and have aided in the identification of differences in the timing of dental and somatic development across a range of hominin taxa.

Slightly different applications of tooth histology also relate enamel and dentine microstructure features and/or the chemical composition of teeth to major life events. Enamel can record signs of biological stress (Guatelli-Steinberg, 2001), such as the neonatal line (Beynon et al., 1991; Schwartz & Dean, 2008) and stress lines that correspond with stressful events such as injury (Schwartz et al., 2006). In modern human third molars, parturition lines may provide evidence of age at first reproduction or inter-birth intervals (Dean & Elamin, 2014), but this method has yet to be applied to late-forming hominin teeth that developed during reproductive years. Changes in the chemical composition of enamel and dentine can also be tracked; carbon, nitrogen, strontium, and barium isotopes have all allowed for explorations of early life diet (e.g., Austin et al., 2013; Bocherens et al., 2001; Fahy et al., 2014; Humphrey, 2014). Notably, barium and calcium isotopes have been used to identify a weaning signal in juvenile Neanderthal (Austin et al., 2013).

An alternative approach to inferring life history in the fossil record relies on the links among brain size, growth, and life-history milestones. This is an indirect route to life history, with the relationships between brain size and life history likely due to more proximate factors such as mortality or the energetic constraints that brains impose upon growth trajectories (Leigh, 2004; Leigh & Blomquist, 2007), but brain size is often a better predictor of primate life history than body size (Harvey & Clutton-Brock, 1985). In addition to correlations between the pace of life and adult brain size, neonate and infant cranial capacity estimates have been used to posit, for example, that Neanderthals had a life history at least as slow as, and possibly slower than, modern *H. sapiens* (Ponce de León et al., 2008). Similar work comparing fossil brain growth trajectories with those of extant humans and chimpanzees has been carried out for *H. erectus* (Coqueugniot et al., 2004) and *Australopithecus afarensis* (Alemseged et al., 2006).

The methods outlined here are invaluable and have allowed paleoanthropologists to probe the life histories of extinct species, but they do have some limitations. It can be difficult, for example, to determine the exact timing of dental development in a fossil taxon (Hublin et al., 2015). Tooth eruption is a process and emergence is an event defined based on penetration of the crown through the gingival tissues, which obviously do not preserve. Furthermore, particularly when comparing closely related extant hominoid taxa, the predictive value of some correlations between age at tooth emergence and life history weakens, and exceptions to the "rules" that hold across a wider range of primate species appear (e.g., Dirks & Bowman, 2007; Guatelli-Steinberg, 2009; Humphrey, 2010; Robson & Wood, 2008; T. M. Smith et al., 2015; T. M. Smith, Machanda, et al., 2013). This has implications for drawing conclusions about extinct hominins, as they are closely

related to extant hominoids. Beyond dental-based methods, reconstructions of life history that use brain growth trajectories are often based on estimates of fossil brain size and depend on comparisons to modern human or chimpanzee growth trajectories that may substantially overlap (Leigh, 2004; Robson & Wood, 2008).

Ultimately, since life history can vary with local conditions and aspects of life history can change in a mosaic fashion (i.e., the timing of one event can shift without parallel shifts in all features of growth and development), it is unlikely that a single feature of a fossil specimen or taxon will reveal a complete life-history reconstruction. As each method probes a slightly different aspect of growth and development, the use of a suite of methods and multiple lines of evidence, as appropriate and feasible, will provide the most complete and coherent picture of life history in the fossil record. The next section will thus outline current understandings of growth and development in the hominin fossil record, while identifying how the results of this dissertation may complement the existing body of literature and contribute new perspectives.

5.3. Life history and growth reconstructions in the hominin fossil record

It is still unresolved when the modern human combination of life-history traits evolved, but current evidence suggests that a fully modern pattern is a geologically recent phenomenon. Australopiths and early *Homo* likely had ontogenies that were, on the whole, faster than that of modern humans. A growing body of work (e.g., Dean et al., 2001; Dean & Lucas, 2009; Guatelli-Steinberg et al., 2005; Ponce de León et al., 2008; T. M. Smith et al., 2010) substantiates the hypothesis that some of the nascent features of the modern pattern emerged in a mosaic fashion throughout hominin evolutionary

history. Due to the uncertainty in establishing first and last appearance dates of some of these taxa, as well as the desire to emphasize common biological traits and ecological relationships rather than temporal ones, research into hominin life histories will be discussed here from a taxonomic perspective, rather than a temporal one.

Neanderthals

Given the connections between brain size, body size, and life history, Neanderthal life histories can be informative for hypotheses of human life-history evolution, as they were a large-brained, large-bodied group closely related to *H. sapiens* (Ponce de León & Zollikofer, 2001). On average, Neanderthal brains were absolutely larger (Trinkaus & Tompkins, 1990), but smaller relative to body size (Ruff et al., 1997) than those of modern humans. An investigation of brain growth using Neanderthal neonate and infants led researchers to conclude that Neanderthal life history was as slow as, ors even slower than, that of modern *H. sapiens* (Ponce de León et al., 2008), while further work posited that modern human and Neanderthal infants were similarly altricial (Hublin et al., 2015).

Conclusions drawn using dentition-based methods in Neandertals, however, are equivocal. A growing body of histological work on Neanderthal dental tissue has failed to come to a consensus on whether—and if so, how—Neanderthal life history differed from that of modern humans (e.g., Dean et al., 2001; Guatelli-Steinberg et al., 2005, 2007; Macchiarelli et al., 2006; Ramirez Rozzi & Bermudez De Castro, 2004; Rosas et al., 2018; T. M. Smith, et al., 2010; T. M. Smith, Toussaint, et al., 2007). Early dental histology work by Dean and colleagues (2001), for example, concluded that the crown formation times fell within the range of modern humans, with some later work arriving at

a similar conclusion (e.g., Guatelli-Steinberg, 2009; Guatelli-Steinberg et al., 2005; Macchiarelli et al., 2006).

Other dental research paints a different picture of Neanderthal life history, suggesting that major life-history events occurred at younger ages than would be predicted based on modern human dental eruption and calcification standards. Dental microstructure has been used to posit that Neanderthals have a shorter period of somatic growth than modern humans, as well as *H. antecessor* and *H. heidelbergensis* (Ramirez Rozzi & Bermúdez de Castro, 2004), and lack a modern human-like prolonged childhood period (T. M. Smith, Toussaint, et al., 2007). If, on balance, Neanderthal dental development indeed proceeded at a faster pace, Neanderthals may have experienced faster somatic development than modern humans (J.-J. Hublin et al., 2015), perhaps reflecting higher juvenile and young adult mortality rates (Trinkaus, 1995). This faster growth could be uniquely Neanderthal feature or a primitive retention from the last common ancestor, as archaic *H. sapiens* at ca. 160 ka already exhibited a dental development trajectory close to that of modern humans (T. M. Smith, Tafforeau, et al., 2007; T. M. Smith et al., 2010). Given the preservation and existing CT imagery of some Neanderthal crania (see, e.g., Ponce de León et al., 2008), it may be possible to obtain Neanderthal sella turcica measurements and thus make inferences about the pace of growth that are grounded in mechanistic connections between sella turcica size, the pituitary, and growth. This could help to evaluate whether Neanderthals exhibit somatic growth rates distinct from modern humans and could inform hypotheses about whether there are aspects of body growth rate that are unique to Neanderthals.

In addition to work on the overall pace of life, research has attempted to infer Neanderthal weaning behavior. Early analyses of dental wear (Skinner, 1997) and enamel hypoplasia (Ogilvie et al., 1989) suggested that Neanderthals weaned at a later age than modern *H. sapiens*, but these studies were carried out before the faster dental development in Neanderthals (relative to modern humans) was demonstrated (Hublin et al., 2015). Direct evidence of weaning provided by barium/calcium ratios from the Scladina juvenile Neanderthal's M1 enamel indicates early weaning relative to modern humans (and many extant hominoids) (Austin et al., 2013). The weanling died at a young age, however, and nursing appears to have ceased abruptly (Austin et al., 2013; T. M. Smith, 2013), so this evidence of early weaning may not be representative of the population as a whole. Other estimates of Neanderthal weaning age based on nitrogen isotopic signatures suggest weaning at around 4 years of age, although the study individual could be younger than its tooth eruption suggests and thus could have weaned earlier (Bocherens et al., 2001). Regardless, Neanderthal weaning age estimates from both studies contrast with modern human weaning around 3 years of age (Alvarez, 2000; Sellen, 2006). Though estimates of growth rate derived from Neanderthal sellae turcicae would not be able to speak to Neanderthal weaning age, they could help to contextualize weaning within the broader trajectory and pace of Neanderthal growth.

On the whole, most research points to a Neanderthal pattern of growth and development that diverges in some respects (e.g., weaning age, brain growth patterns, aspects of dental development) from that of modern humans, although recent work challenges the notion that Neanderthals had a fundamentally different pace of growth than modern humans (Rosas et al., 2018). Incorporating data about the overall pace of

growth from sella turcica size would contribute additional perspectives to this debate. In any case, Neanderthal growth and development likely reflects adaptations to local ecological conditions and demographic forces (e.g., extrinsic morality and energy availability) that likely varied between temporally and geographically distinct Neanderthal populations.

Homo heidelbergensis *and* H. antecessor

Compared to Neanderthals, less work has been carried out on *H. heidelbergensis* and *H. antecessor*. The large-bodied (Carretero et al., 2012) and large-brained (Bermúdez De Castro et al., 1997) hominins from Gran Dolina TD-6, attributed to *H. antecessor*, have been proposed to reveal the earliest evidence of a modern-human-like pattern and pace of dental development (Bermúdez De Castro et al., 2010) that is not linked to an increase in body mass or stature (Dean, 2016). Other research, however, posits that *H. antecessor* and *H. heidelbergensis* had shorter periods of dental growth and crown formation than modern and Upper Paleolithic *H. sapiens*, yet longer crown formation times than Neanderthals (Bermúdez de Castro et al., 2003; Lacruz et al., 2005; T. M. Smith et al., 2010). Longer crown formation time, however, may not necessarily mean slower growth in *H. antecessor* and *H. heidelbergensis* than in Neanderthals, as it is still unclear whether slow rate of enamel growth is unequivocally associated with the extended growth periods that characterize modern humans (Bermúdez de Castro et al., 2003).

Further analyses of the development of the anterior teeth relative to the molars in *H. antecessor* indicated a pattern of dental development similar to that of modern humans

(Bermúdez de Castro et al., 2003). Given the relationship between the timing of dental development and the length of growth periods, the authors concluded that *H. antecessor* may have been characterized by a prolonged period of maturation similar to those observed in modern human populations (Bermúdez de Castro et al., 2003). If the hypothesized association between a cranial capacity greater than 1000 cm^3 and a generally modern human-like life history hold (B. H. Smith, 1991), both the endocranial volume and dental development patterns of *H. antecessor* and *H. heidelbergensis* suggest that the broad trend of extended life history compared to extant apes was firmly established by the Middle Pleistocene (perhaps serving as a foundation for the emergence of the derived patterns observed in modern humans). Although a study of *H. heidelbergensis* and *H. antecessor* sellae tucicae would not be able to comment on the duration of growth periods, it could contribute to understandings of how the pace of growth in *H. heidelbergensis* and *H. antecessor* compares to that of both later hominins and extant great apes. Information provided by sella turcica size could also comment on whether a slower (or faster) pace of growth in these taxa coincides with a longer or shorter duration growth (as estimated by the established methods outlined above) relative to Neanderthals and/or modern *H. sapiens*.

Earlier Homo*:* Homo erectus*,* H. rudolfensis*, and* H. habilis

The fossil record of earlier *Homo* species provides further evidence that the modern human life-history profile encompasses a suite of derived traits that arose post-*H. erectus*. Though the modern human-like body proportions and large body and brain size of *H. erectus* raises the possibility that *H. erectus* life-history attributes might fall within

the range of modern human variation (Hawkes, 2003), this expectation is not supported by studies of fossil remains. Most analysts suggest faster development not just in smaller-bodied *H. habilis* and *H. rudolfensis*, but also in *H. erectus*, relative to *H. sapiens* and other later hominins (e.g., Bromage & Dean, 1985; Dean et al., 2001; Dean & Lucas, 2009). Crown and enamel formation times in *H. erectus* do not fall within the range of modern human variation in enamel growth (Dean et al., 2001). Tooth development in both *H. habilis* (Dean, 1995; Robson & Wood, 2008) and *H. rudolfensis* (B. H. Smith, 1991) have similarly been proposed to indicate that these species possessed life histories unlike that of modern *H. sapiens*.

Dean et al. (2001) estimated that M1 emerges at age 4.4 years and M2 at age 7.6 years in *H. erectus*, which is later than in Neanderthals, extant apes, and australopiths (Dean & Lucas, 2009), yet earlier than the *H. sapiens* average. Subsequent analyses concluded that although tooth development in early *Homo* and *H. erectus* individuals fell within the known modern human ranges (Dean, 2016; Dean & Liversidge, 2015), early *Homo* and *H. erectus* patterns were much more squarely within the range of modern chimpanzees (Dean, 2016). On the whole, these data imply that early *Homo* and *H. erectus* were likely more similar to extant great apes than modern humans in the timing of its dental development (Dean, 2016).

Homo erectus further reveals an uncoupling of some of the correlations between somatic and dental development observed in modern humans. Modern human standards for epiphyseal fusion point to an age of death around 13-13.5 years old for the *H. erectus* juvenile KNM-WT 15000 (Ruff & Walker, 1993); when this age is considered in conjunction with body mass and stature, estimates of which are within the range of a

modern 15-18-year-old (Dean & Smith, 2009; Ruff, 2007), it implies that *H. erectus* may have experienced faster somatic growth than modern humans. Dental eruption patterns, however, suggest that the individual had a dental age around 10 years (using modern human standards) (Dean & Smith, 2009), while histology suggests even faster dental development and a calendar age at death of between 7.6 and 8.8 years (Dean et al., 2001; Dean & Smith, 2009). The skeletal age of KNM-WT 15000, based on modern human standards for epiphyseal fusion, is thus likely advanced relative to dental age, so it is possible that this individual had achieved a greater proportion of adult stature than is typical of modern humans at a similar stage of dental development (B. H. Smith, 1993).

Ruff and Walker (1993) applied modern human stature growth rates to estimate KNM-WT 15000's adult stature at ~185 cm. While this height could be an overestimate due to pathology (e.g., Ohman et al., 2002), no other adult *H. erectus* specimen that hits this benchmark has been found (Hublin et al., 2015), which suggests that *H. erectus* did not experience growth rates comparable to those of modern humans. Subsequent work reevaluated growth trajectories using life-history milestones as landmarks and revised adult height to ~163 cm, estimating that adult height would have been achieved by about age 12 and concluding that growth was characterized by a slower and shorter spurt than that of modern *H. sapiens* (Graves et al., 2010). Although the initial publication notes that part of KNM-WT 15000's sphenoid is missing (Brown et al., 1985), I was not able to conclusively determine the condition or presence of a sella turcica in KNM-WT 15000. Photographs of the cranium do not instill confidence that the sella turcica is preserved (and recognizing that interspecific sella turcica-growth rate correlations may not be as robust in subadults as adults), but if the sella turcica is present, it could be used to

estimate growth rate and help evaluate whether this *H. erectus* individual grew at a slower or faster rate than modern humans.

In addition to KNM-WT 15000, both dental and postcranial remains from Dmanisi (Lordkipanidze et al., 2007, 2013) provide insights into early *H. erectus* (sensu latu) growth and development. Subadult stature and body mass estimates exceed the heights and weights of modern human children of similar estimated age (Dean, 2016), making it likely that somatic development was more similar to that of chimpanzees than modern humans. On balance, the disconnect between *H. erectus* juveniles' relatively advanced skeletal development and calendar age (as suggested by dental microstructure) make it unlikely that *H. erectus* possessed the slow childhood growth and adolescent growth spurt that characterize modern humans (Hublin et al., 2015). Although they cannot separate childhood growth from the sum total of all subadult growth, the sella

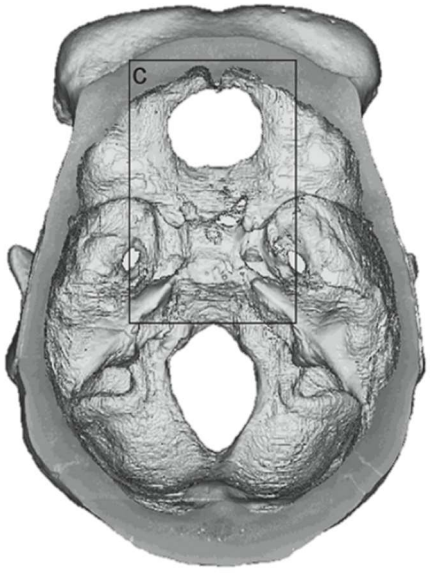


Figure 5.1. Preserved fossil sella turcica visible in the endocranial cavity of Ngawi 1 (*Homo erectus*) after digital removal of matrix (Kaifu et al. 2015).

turcica-based methods proposed in this dissertation could help to corroborate such hypotheses about the pace of somatic growth in *H. erectus,* especially given the preservation of some *H. erectus* crania (e.g., Kaifu et al., 2015; Lordkipanidze et al., 2013; Figure 5.1).

Studies of *H. erectus* brain growth are less conclusive than those of skeletal development. Coqueugniot et al. (2004) postulated that the pattern of brain growth as a proportion of adult volume in *H. erectus* was more similar to that of modern chimpanzees than modern humans, while Leigh (2006) critiqued this study and posited a more human-like pattern. Additional comparative work contra Coqueugniot et al. (2004) that corroborates Leigh (2006) hints that *H. erectus* exhibited the increased neonate brain size and faster postnatal growth rates (relative to extant apes) that characterize modern human ontogeny (Cofran & DeSilva, 2015; DeSilva & Lesnik, 2008).[12] This conclusion is further supported by neonatal brain size estimates obtained from the Gona pelvis (S. W. Simpson et al., 2008). As a corollary of physiological and obstetrical constraints that are hypothesized to accompany birthing large-brained offspring (Ponce de León et al., 2008), it has also been proposed that large neonatal brain size in *H. erectus* (~300 cc) implies secondary altriciality in this species (Cofran & DeSilva, 2015; DeSilva & Lesnik, 2006;

[12] Though much work suggests that the increased brain growth that distinguishes modern humans from chimpanzees was in place by *H. erectus*, is important to remember that estimates of brain growth rate are dependent upon adult brain size. This is not trivial, as *H. erectus* cranial capacity varies considerably both temporally (Hublin & Coqueugniot, 2006) and geographically (Antón et al., 2014; Spoor et al., 2007). Estimates of brain growth rates in this species thus depend on which adult specimens are used as a reference. Nevertheless, since *H. erectus* as a species has a smaller overall brain size than modern humans, even relatively fast brain growth rates similar to *H. sapiens* would suggest that *H. erectus* had a shorter period of brain growth, and thus possibly an earlier age at maturity (Hublin et al., 2015).

Leigh, 2006; Ruff & Walker, 1993). Though the results of this dissertation cannot speak directly to altriciality and patterns of brain growth, the strong correlations between sella turcica size, brain size, and growth rate that it revealed could provide additional information about the relationship between postnatal growth rate and brain size in *H. erectus*.

When the variable brain and body size of *H. erectus* (Lieberman, 2007) is considered in tandem with its wide geographical range, the possibility of life-history distinctions between Asian and African populations emerges as well. Bermúdez de Castro et al. (2003) found similarities in dental development between modern humans and Asian *H. erectus* populations, but not between modern humans and African *H. erectus* (=*H. ergaster*) populations, which fell intermediate between a modern human-*H. antecessor*-*H. heidelbergensis* cluster and an extant great ape-archaic hominin cluster. It is unclear whether the differences between Asian and African populations are any wider than would be expected given regional variation *in H. sapiens* (Liversidge, 2003; Reid & Dean, 2006), but in any case, the preliminary evidence of life history and growth divergence between *H. erectus* populations warrants further investigation. The results of this dissertation suggest that these potential differences may be related to differences in ecology and selective pressures (such as the seasonal variation in food resources that was identified as a predictor of growth rate variation in extant primate populations). Although I do not recommend that sella turcica size be used to make inferences about the ecological underpinnings of variation in growth rate, sella turcica size could be employed as an alternative, non-dental-based, method to explore growth rate differences between African and Asian *H. erectus*.

Australopiths

Most evidence suggests that, as a group, australopiths possessed generally "faster" life histories than later hominins, including modern humans. Studies of dental eruption (Dean, 1985) and tooth formation (Bromage & Dean, 1985; Dean et al., 2001) proposed that the dental development of *Australopithecus* species that do not exhibit the megadontia and related masticatory adaptations of *Paranthropus* species followed a chimpanzee-like pattern, with faster life histories than modern humans. Dental microstructure analyses and developmental patterns further suggest that *A. afarensis* and *A. africanus* developmental trajectories were more similar to those of modern *Pan* than modern *H. sapiens* (Bromage & Dean, 1985; Lacruz et al., 2005; T. M. Smith et al., 2015). Patterns of dental and skeletal maturation in *A. sediba* specimens similarly suggest that growth and development proceeded at a rate not significantly different from that of the extant great apes (Berger et al., 2010; Cameron et al., 2017; Dean, 2016). More recent analyses using synchrotron technology on an expanded sample of Pliocene and early Pleistocene hominins suggests that these taxa skewed more towards modern chimpanzees in terms of dental development, and, in the case of *A. africanus*, possessed a chimpanzee-like range of variation in the timing of M1 emergence (T. M. Smith et al., 2015). Interestingly, the authors concluded that one *A. anamensis* and one *A. africanus* individual exhibited a pace of dental development faster than both chimpanzees and modern humans of similar age (T. M. Smith et al., 2015), which perhaps suggests that a chimpanzee-based analogy may be inappropriate.

On the whole, dental-based methods of inferring life history support the hypothesis that gracile australopiths grew and developed at a pace more similar to extant chimpanzees than modern humans. Work by Alemseged and colleagues (2006), on the Dikika juvenile on the other hand, concluded that *A. afarensis* took longer than extant apes to achieve adult brain size and possessed a pattern of brain growth more similar to *H. sapiens* than to great apes. Growth trajectories may also indicate slower absolute brain growth in *A. afarensis* than in extant apes (Alemseged et al., 2006; Gunz et al., 2020), which could imply that the species had a longer period of parental dependence than extant apes. This conclusion is still up for debate, however, as the extent of variation in the timing of extant ape brain development is not well documented (Hublin et al., 2015).

Although increased brain size has been hypothesized to be a driver of a protracted period of growth (Kaplan et al., 2000; Martin, 1996; Ross & Jones, 1999), the increase in australopith brain size compared to extant great apes of similar body size (Falk et al., 2000) has not been hypothesized to be linked to a protraction of life history. Instead, changes in australopith brain size and shape have been proposed to be the result of reorganization and expansion of brain regions associated with tool making and use (Gómez-Robles et al., 2014; Stout et al., 2008), although recent work found few, if any, features of the *A. afarensis* brain that are indicative of humanlike brain reorganization (Gunz et al., 2020). Dean (2016) contends that if modest brain expansion was driven largely by tool use, a long and costly period of social growth and development (i.e., an extend modern human-like childhood, juvenile, or adolescent period) would not have been necessary. This argument draws upon the predictions of the social brain hypothesis (Dávid-Barrett & Dunbar, 2013; Dunbar, 2003), which proposes that increases in brain

size are linked to living in larger and more complex social groups, which require unique cognitive skills. As a result, longer growth and developmental periods were driven by both the energetic costs of maintaining this much larger (i.e., social) brain and the developmental costs of learning the social skills necessary to navigate complex social situations that accompany larger group living (Dávid-Barrett & Dunbar, 2013; Dunbar, 2003). Recent work, however, has argued that primate brain size is better explained by diet than by sociality (DeCasien et al., 2017), perhaps as the result of selective pressures linked to seasonal and/or extractive foraging (Melin et al., 2014). This potentially weakens the argument that increases in brain size are only accompanied by a longer period of growth and development if they are the result of social pressures.

Reduced canine size and dimorphism (which could imply decreased male-male competition, and perhaps fewer intra-group conflicts) also suggest that australopiths may have lived in larger social groups than extant great apes, which may have fostered more efficient food procurement and potentially reduced extrinsic mortality rates. In such a situation, prolonged growth, delayed reproduction, and redistributing energy towards

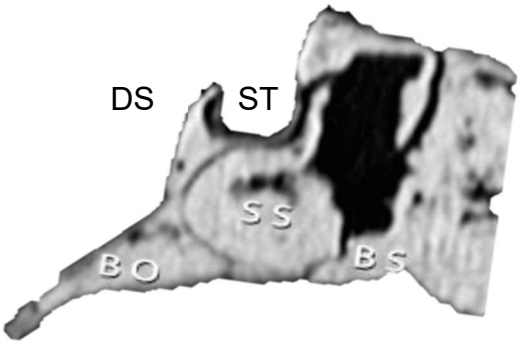


Figure 5.2. Preserved fossil sella turcica visible in a sagittal section scan of Sts 5 (*Australopithecus africanus*) sphenoid (Bonmati et al. 2008), ST = sella turcica, DS = dorsum sellae.

growing a large brain that promotes social cohesion would have been advantageous. This scenario receives comparatively less support, however, as most evidence suggests that gracile australopith taxa did not have extended growth relative to modern apes (e.g., Dean, 2016; T. M. Smith et al., 2015). Although preservation of australopith crania is often not as good as preservation in geologically younger taxa, there are some australopith crania with preserved sellae turcicae (e.g., Bonmatí et al., 2008; Figure 5.2) that may permit the estimation of growth rate and thus could contribute to the discussion about the pace of somatic growth among gracile australopiths.

Dental development data also suggest that the more megadont, or "robust," australopiths also possessed a life-history profile distinct from that of other fossil hominins, as well as from *Pan* and modern *Homo*. Bromage and Dean's (1985) analysis of *Paranthropus robustus* indicated that the timing and duration of dental development was more similar to that of modern *Pan* than *H. sapiens*, while B. H. Smith (1986) suggested that unlike other fossil hominins, *Paranthropus* was characterized by developmentally advanced anterior teeth relative to M1. Age-at-death-based estimates of M1 emergence age additionally imply a faster pace of life, or at least earlier weaning, than that observed in the extant great apes (Kelley & Schwartz, 2012). Despite the robust and gracile australopiths possessing a generally similar dental development schedule (Kuykendall, 2003), comparisons of tooth emergence ages across known-age individuals further suggest that some robust taxa may have experienced delayed M1 emergence relative to gracile taxa (Kelley & Schwartz, 2012; T. M. Smith et al., 2015).

The premolar and molar crowns of *P. boisei* appear to have formed in at least the same amount of time as, and possibly faster than, modern *H. sapiens* (Beynon & Wood,

1987; Bromage & Dean, 1985; Macho & Wood, 1995). The fast development of *P. boisei* teeth is similar to that seen in the deciduous human dentition and is particularly surprising given that crown formation time typically is positively correlated with crown height and *P. boisei'*s crowns are about twice the size of those of modern humans (Beynon & Wood, 1987). While the underlying biological reason for *P. boisei*'s shorter crown formation time is debated (Beynon & Wood, 1986, 1987; Macho, 2001), the fact remains that some aspects of dental development in the taxon appear to be accelerated. Assuming that life history does in some way map on to dental development in *Paranthropus* species, the faster rates of enamel and dentine formation compared to other members of the *Pan-Homo* clade (Robson & Wood, 2008), advanced incisor crown formation relative to M1 (Kuykendall, 2003; B. H. Smith, 1986), advanced premolar development compared to modern humans (Beynon & Dean, 1987), and even differences between the East and South African species of *Paranthropus* (Kuykendall, 2003) reinforce the mosaic nature of hominin life-history evolution.

These aspects of *Paranthropus* dental development further hint that the robust australopiths may have possessed a pattern of growth and development not observed in any living primate taxon and a uniquely derived life history compared to modern humans, the extant great apes, and the gracile australopiths (Kuykendall, 2003). Dietary differences, which are hypothesized to have driven the evolution of masticatory system differences between the gracile and robust australopiths (Grine, 1988), may also have played a role in the observed developmental differences between the two groups. *Paranthropus* has been proposed to have subsisted on lower-quality diets (e.g., (Sponheimer et al., 2013; Van Der Merwe et al., 2008) and occupied more open habitats

than gracile species (e.g., Wood & Strait, 2004), two ecological attributes that tend to be associated with earlier maturation and larger adult body size (Macho & Williamson, 2002), as well as accelerated dental development compared to closely related species of similar body size (Godfrey et al., 2001; Macho, 2001). Especially for terrestrial primates, more open habitats also tend to carry a higher mortality risk, which, along with selective pressures related to diet, could have driven the evolution of the robust australopiths' accelerated life-history features. Though additional life-history data is necessary to further test hypotheses that the robusts' unique life-history pattern reflects, at least to some degree, selective pressures related to diet, analyses of *Paranthropus* sellae tucicae, if available, could shed light on the pace of growth in the taxon and inform hypotheses about the links between growth and ecology.

5.4. Holistic reconstructions of patterns of growth, development, and life history

Each method of reconstructing aspects of life history, growth, and development in the fossil record can contribute a different perspective on growth and development. Although valuable and informative, each is also accompanied by limitations, so the most complete picture of the life history in the fossil record will combine multiple lines of evidence. Dental developmental sequences, for example, are not on their own inherently meaningful, and are only able to provide information about life history insofar as certain eruption patterns or emergence ages correlate with certain life-history features, while understanding patterns of skeletal growth requires modern reference samples. Furthermore, many methods to reconstruct hominin life history necessitate the use extant species as reference taxa against which to frame hypotheses of life-history evolution.

This can be problematic. It assumes that the life histories of extant taxa have changed little since the split from their last common ancestor, which may not be the case (Robson & Wood, 2008). It also risks erroneously placing extinct hominins on the "ape-like" or "human-like" spectrum of variation, effectively limiting the degree to which a fossil individual can exhibit a unique, species-specific life-history pattern that is not observed in the extant taxa to which it must be compared.

The novel sella turcica-based method proposed here does not eliminate all of these concerns, but has the potential to contribute additional data regarding the pace of growth that more directly builds upon pituitary hormone production, a proximate mechanism that regulates growth. Using the sella turcica to estimate growth rate would not hinge on correlations between somatic growth rate and, for example, the pattern and pace of dental development and/or the trajectory of brain growth, thus "freeing" growth rate to vary somewhat independently of these variables (while recognizing that they are, of course, interdependent to some degree). Use of the sella turcica would also make it possible to compare the pace of dental development or brain growth trajectories to the overall pace of somatic growth to evaluate if and/or how they are uncoupled in hominin species.

Ultimately, a holistic approach that allows for comparisons and connections between growth rate as estimated by sella turcica size and aspects of growth and development as estimated using other methods will provide the most complete understanding of the pace of life in fossil hominins. When the results of well-established methods are interpreted in conjunction with more recently developed ones, such as those that use enamel isotopes to trace signatures of weaning (e.g., Austin et al., 2013), the

timing of specific life-history milestones can be framed by more general pace-of-life estimates. Even misalignments between conclusions drawn using different methods (e.g., Neanderthal body and brain size suggesting high energetic requirements and thus slow growth, while some studies of dental development suggesting faster growth) can be informative, as seemingly "conflicting" life-history parameter estimates may indicate the mosaic evolution of life history and/or may signal that different methods have picked up on aspects of temporal or geographic variation between populations.

5.5. Future directions

The outcomes of this dissertation suggest multiple avenues for future research. In addition to using preserved sellae turcicae to explore postnatal growth rate in the fossil record, there are also potentially lucrative opportunities to further explore growth patterns, proximate and ultimate causes, and the hormonal regulation of processes that affect growth (such as stress responses) in living populations. The population-level negative correlation between sella turcica size and growth rate presented in Chapter 2, for example, suggests that the production of somatic growth-linked hormones does not drive differences in sella turicica size, and possibly pituitary size, within the Cayo Santiago macaque population. At the most basic level, it would thus be valuable to explore both the effect of hormone production on pituitary size, as well as the correlation between pituitary size and sella turcica size, in this population.

Beyond growth hormone, the pituitary gland produces and/or secretes a number of hormones that are central to both reproduction (S. L. Kaplan & Grumbach, 1978; Robertson et al., 2009; Saltzman et al., 2011; Stojilkovic, 2018) and stress responses

(Marques et al., 2018; Saffran & Schally, 1955; Smith & Vale, 2006). As discussed in Chapter 2, section 2.4, stress-related pituitary hormones may play a particularly important role in the observed relationships between sella turcica size and growth rate. As taxing environments and adverse conditions are linked to increases in stress hormone production (Beehner & Bergman, 2017; Novak et al., 2013; Thayer et al., 2018), environmentally linked stress responses may impact growth. Though higher levels of stress hormones such as cortisol have traditionally been linked to decreased growth in mammals (Achermann & Jameson, 2010; Bellamy & Leonard, 1964; Lesage et al., 2001), increasing evidence connects stress hormone levels to accelerated growth (Berghänel et al., 2017; Dantzer et al., 2013). It may be that the faster growth rates that occur under more stressful conditions are linked to maternal stress responses and hormonal cues that are transmitted to offspring (Dantzer et al., 2013), so further research that assays both juvenile and maternal hormone levels in primate populations would be valuable to tease out the relative effects of maternal cues and individual experiences on growth responses to stress, as well as to assess whether different sources of stress have divergent effects on growth rates (e.g., some sources accelerate growth, while others slow growth). The results presented in Chapter 3 suggest that within the Cayo Santiago macaque population, environmental conditions that favor smaller body size are also correlated with slower growth, which is consistent with other explorations of growth in wild primate populations (e.g., Altmann & Alberts, 2005; Jarrett et al., 2020). If these divergent trajectories are due to stress effects, however, they are not consistent with results suggesting that stress-inducing conditions can favor faster growth, so further research in this arena that explores

whether challenging environmental conditions are consistently linked with higher stress hormone levels in the Cayo macaque population would be worthwhile to explore.

Broadly, this project supports the hypothesis that across all primate taxa, the trajectory of growth is linked to the specific stressors and ecological problems posed by a species' environment (e.g., resource availability or competition), which can produce a range of responses in patterns of growth, development, and reproduction (e.g., Godfrey et al., 2004; Pianka, 1970). In particular, the outcomes of analyses exploring the environmental correlates of growth trajectories (Chapters 3 and 4) suggest avenues for future research that explores the intersection of developmental conditions, hormones, growth, and later life outcomes across primate species. Hormone levels fluctuate in response to experienced environment and early life conditions (Monaghan, 2008; Wingfield et al., 1998; Wingfield & Kitaysky, 2002), with both hormonal and environmental differences proposed to be linked to not only to variation in growth and life history (e.g., Berghänel et al., 2017; Bogin et al., 2007; Crespi et al., 2013; Dantzer et al., 2013; Frisancho et al., 1973; Swanson & Dantzer, 2014), but also to later life health outcomes (e.g., Cao-Lei et al., 2016; Hayward et al., 2013; Hong et al., 2020; Kittleson et al., 2006; Lu et al., 2019; O'Rand & Hamil-Luker, 2005; Tung et al., 2016). Epigenetic mechanisms such as DNA methylation have been hypothesized to be the mechanism by which environment (particularly early life environment) can permanently alter hormonal responses and affect later life health and fitness (Cao-Lei et al., 2016; Kinnally, 2014; Kundakovic et al., 2015; Labonte, 2012; Murgatroyd & Spengler, 2011; Provençal et al., 2020). As this research revealed that environmental conditions experienced by Cayo Santiago macaques during both gestation and the first year of life are linked to growth

outcomes across the juvenile period, the connection between early life environment and later life growth may be the result of similar epigenetic regulation. Epigenetic mechanisms (i.e., DNA methylation) may program growth hormone responses, but may also alter stress hormone responses and thus contribute to the effect of stress on growth. Given the links between early life environment and later life health, individuals in Cayo Santiago population who exhibit different growth trajectories may also exhibit later life health and fitness disparities, so direct explorations of the epigenetic and hormonal mechanisms that mediate the association between environment and growth variation, as well as other aspects of life history, health, and fitness, may be valuable.

Furthermore, although epigenetic changes induced by environmental conditions are small, identifying loci that are susceptible to such changes could prove invaluable for understanding the development and persistence of responses that are influenced by environment (Gluckman et al., 2009), as such research can attempt to unite mechanisms of change at the individual level with evolutionary outcomes. Different relationships between environmental conditions and growth, developmental, and reproductive outcomes can emerge depending on whether an analysis is carried out at the inter- or intraspecific level (Ellis et al., 2009), so it is essential to understand the different causes or mechanisms that operate at different levels (e.g., evolutionary selective pressures, developmental plasticity, more proximate mechanisms such as hormone production and, at the molecular scale, epigenetics). Both developmental plasticity (e.g., Pfennig et al., 2010) and hormone responses (e.g., Dantzer et al., 2013; Wingfield & Kitaysky, 2002) can allow individuals to respond to local environment independently of genetics. Though these responses can affect reproductive fitness (and thus the transmission of genes, some

of which may predispose individuals to responsiveness to the environment, to the next generation), they themselves cannot be transmitted to subsequent generations. At the molecular level, epigenetic processes can allow an organism to respond to environment through changes in its gene expression (Jaenisch & Bird, 2003), and it may be possible for some environmentally triggered epigenetic changes to be inherited from generation to generation (Feil & Fraga, 2012). In the case of stress-linked responses, for example, both genetic and epigenetic stress effects may contribute to change (and potentially adaptation) in subsequent generations (Feil & Fraga, 2012), but this is certainly a possibility that requires further research.

Ultimately, this dissertation took a multifaceted approach to understanding the determinants of growth differences between individuals and species while attempting to connect growth rates to a hard tissue proxy; the linkages between growth, hormones, ecology, and environment that it explored only scrape the surface. Much of the value in studying life history lies in its links to other biological phenomena and evolutionary dynamics, opening the door for further investigation. It is hoped that the results of these investigations will help both biologists and paleoanthropologists better understand the determinants and pace of body growth in living and extinct primate species and will contribute to a deeper understanding of the forces that have shaped the evolution of primate growth.

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APPENDIX A

CHAPTER 2 SUPPLEMENTAL TABLES

S2.1. CT scan parameters, Cayo Santiago *Macaca mulatta* cranium sample

specimen	pixel size (μm)	voltage (kV)	current (μa)	projections
CPRCMUS-00742	35.132412	125	64	1285
CPRCMUS-00867	31.546565	125	64	1161
CPRCMUS-00870	31.546565	125	64	1161
CPRCMUS-00873	35.848214	120	66	1200
CPRCMUS-00878	35.848214	125	64	1440
CPRCMUS-00882	34.414901	125	64	1333
CPRCMUS-00884	35.848214	120	66	1090
CPRCMUS-00885	29.754496	125	64	1241
CPRCMUS-00886	35.848214	125	64	1200
CPRCMUS-00888	35.848214	120	66	1058
CPRCMUS-00889	31.189518	125	64	1440
CPRCMUS-00892	35.848214	120	66	1200
CPRCMUS-00900	31.905321	125	64	1028
CPRCMUS-00902	32.981587	125	64	1125
CPRCMUS-00906	35.848214	110	70	1200
CPRCMUS-00907	35.848214	120	66	1200
CPRCMUS-00910	35.848214	108	74	1028
CPRCMUS-00914	35.848214	120	66	1200
CPRCMUS-00919	35.848214	125	64	1200
CPRCMUS-00920	32.264076	125	64	1161
CPRCMUS-00924	35.848214	125	64	1125
CPRCMUS-00925	35.848214	110	70	1200
CPRCMUS-00926	35.848214	125	64	1440
CPRCMUS-00927	31.189518	125	64	1161
CPRCMUS-00947	35.848214	125	64	1028
CPRCMUS-00949	35.848214	120	66	1241
CPRCMUS-00956	35.848214	125	64	1200
CPRCMUS-00985	35.848214	125	64	1125
CPRCMUS-00993	35.848214	110	70	1440
CPRCMUS-00994	35.848214	125	64	1090
CPRCMUS-00997	35.848214	125	64	1200
CPRCMUS-01000	33.697390	125	64	1241
CPRCMUS-01002	35.848214	125	64	1440
CPRCMUS-01148	35.848214	125	64	1285
CPRCMUS-01166	35.848214	108	70	1028
CPRCMUS-01167	35.848214	120	66	1125

CPRCMUS-01171	29.754496	125	64	1200
CPRCMUS-01174	28.679938	125	64	1565
CPRCMUS-01183	31.189518	125	64	1200
CPRCMUS-01188	35.848214	120	66	1333
CPRCMUS-01193	30.830763	125	64	1090
CPRCMUS-01194	35.848214	108	70	900
CPRCMUS-01197	35.848214	75	106	1200
CPRCMUS-01198	35.848214	120	66	1200
CPRCMUS-01545	30.830763	125	64	1200
CPRCMUS-01547	35.848214	120	66	1200
CPRCMUS-01599	34.773656	125	64	1200
CPRCMUS-01601	33.340343	125	64	1440
CPRCMUS-01607	35.848214	120	66	1241
CPRCMUS-01611	32.264076	125	64	1090
CPRCMUS-01626	35.848214	120	66	1285
CPRCMUS-01634	35.848214	125	64	1200
CPRCMUS-01644	35.848214	110	70	1125
CPRCMUS-01648	35.848214	125	64	1028
CPRCMUS-01650	35.848214	110	70	1200
CPRCMUS-01779	34.056145	125	64	1285
CPRCMUS-01782	35.848214	120	66	1028
CPRCMUS-01962	35.848214	125	64	1241
CPRCMUS-01967	34.414901	125	64	1125
CPRCMUS-02129	28.679938	125	64	1241
CPRCMUS-02130	35.132412	125	64	1800
CPRCMUS-02180	32.622832	125	64	1125
CPRCMUS-02200	33.340343	125	64	1125
CPRCMUS-02993	35.848214	120	66	1285
CPRCMUS-03030	32.264076	125	64	1200
CPRCMUS-03031	34.773656	125	64	1125
CPRCMUS-03099	30.113252	125	64	1125
CPRCMUS-03235	32.264076	125	64	1125
CPRCMUS-03358	34.056145	125	64	1125
CPRCMUS-03380	35.848214	125	64	1565
CPRCMUS-03404	30.113252	125	64	1125
CPRCMUS-03427	31.189518	125	64	1058
CPRCMUS-03448	35.848214	125	64	1125
CPRCMUS-03472	35.848214	125	64	1200
CPRCMUS-03714	34.414901	125	64	1125

CPRCMUS-03833	35.848214	125	64	1285
CPRCMUS-03957	35.848214	110	70	1200
CPRCMUS-04071	35.848214	125	64	1565
CPRCMUS-04072	35.848214	125	64	1200
CPRCMUS-04073	35.848214	120	66	1285
CPRCMUS-04074	35.848214	120	66	1800
CPRCMUS-04075	35.848214	120	66	1200
CPRCMUS-04077	34.414901	125	64	1200
CPRCMUS-04102	35.848214	125	64	1440
CPRCMUS-04215	35.848214	120	66	1714

APPENDIX B

CHAPTER 3 SUPPLEMENTAL TABLES

Table S3.1. Linear models for female subadults (n =). Significance level codes: ^0.1; *0.05; **0.01; ***0.001. Significant predictors are bolded at the 0.05 level. No models of GR_{max} were significant; all other combinations of predictors not listed were non-significant.

		standardized beta coefficient							linear model	
	model	evap	precip	temp	age	rank	AICc	ΔAICc	R^2	p-value
gestation conditions	$GR_{overall}$									
	~ evap + precip + temp + age + rank	1.69	-1.29	-0.48	-0.33	-0.34	20.63	0	0.49	0.23
	~ evap + precip + temp + rank	1.49^	-1.10	-0.62	--	-0.30	21.91	1.28	0.45	0.18
	~ evap + temp + age + rank	0.72	--	-1.02^	-0.18	-0.05	25.59	4.96	0.17	0.37
	~ evap + **precip** + age + rank	1.62^	**-1.67***	--	-0.40	-0.37	21.11	0.48	0.50	0.15
	~ evap + temp + rank	0.70	--	-1.04^	--	-0.06	24.14	3.51	0.29	0.21
	~ evap + precip + rank	1.33	-1.52^	--	--	-0.33	22.76	2.13	0.40	0.15
	~ temp + age + rank	--	--	-0.57^	-0.16	0.02	25.70	5.07	0.16	0.32
	~ precip + age + rank	--	-0.56^	--	-0.23	-0.05	26.10	5.47	0.13	0.35
	~ age + rank	--	--	--	-0.32	0.06	28.67	8.04	0	0.75
	Humeral length									
	~ evap + precip + temp + **age** + rank	-0.31	0.28	-0.14	**1.28***	0.06	21.34	5.03	0.63	0.15
	~ evap + precip + **age** + rank	-0.34	0.17	--	**1.26****	0.05	19.57	3.26	0.72	0.05
	~ evap + temp + **age** + rank	-0.10	--	-0.03	**1.25****	0.001	19.79	3.48	0.71	0.06
	~ precip + temp + **age** + rank	--	0.08	-0.17	**1.25****	0.001	19.81	3.5	0.71	0.06
	~ temp + **age** + rank	--	--	-0.09	**1.25****	-0.01	17.88	1.57	0.77	0.02
	~ evap + **age** + rank	-0.14	--	--	**1.25****	0.01	17.80	1.49	0.77	0.02
	~ precip + **age** + rank	--	-0.07	--	**1.23****	-0.02	18.10	1.79	0.76	0.02
	~ **age** + rank	--	--	--	**1.22****	-0.003	16.31	0	0.80	<0.01
	Body mass									
	~ evap + precip + temp + **age** + rank	-0.55	0.40	-0.22	**1.40****	0.04	9.13	0.8	0.91	0.02
	~ evap + precip + **age** + rank	-0.58	0.22	--	**1.37*****	0.03	9.10	0.77	0.92	<0.01
	~ evap + temp + **age** + rank	-0.25	--	-0.06	**1.35*****	-0.03	10.15	1.82	0.90	<0.01
	~ temp + precip + **age** + rank	--	0.06	-0.26	**1.35****	-0.06	11.50	3.17	0.89	<0.01
	~ temp + **age** + rank	--	--	-0.21^	**1.35*****	-0.07	9.58	1.25	0.91	<0.01
	~ evap + **age** + rank	-0.31^	--	--	**1.35*****	-0.03	8.33	0	0.92	<0.01
	~ precip + **age** + rank	--	-0.18	--	**1.31*****	-0.09	11.20	2.87	0.89	<0.01
	~ **age** + rank	--	--	--	**1.29*****	-0.05	12.00	3.67	0.88	<0.01

Table S3.1 Continued

		standardized beta coefficient							linear model	
	model	evap	precip	temp	age	rank	AICc	ΔAICc	R^2	p-value
first year conditions	$GR_{overall}$									
	~ **evap** + precip + temp + age + rank	**2.21***	2.18	2.30^	0.48	0.14	20.49	0	0.50	0.23
	~ evap + precip + **temp** + rank	1.46^	1.65	**-1.79***	--	0.21	22.14	1.65	0.43	0.15
	~ evap + temp + age + rank	0.67	--	-0.69	0.05	0.02	27.89	7.4	0	0.55
	~ evap + precip + age + rank	0.07	-0.47	--	-0.20	-0.01	31.42	10.93	0	0.88
	~ evap + temp + rank	0.63	--	-0.67^	--	0.03	25.90	5.41	0.14	0.32
	~ evap + precip + rank	0.24	-0.51	--	--	-0.07	29.69	9.2	0	0.77
	~ evap + age + rank	0.13	--	--	-0.27	0.02	30.63	10.14	0	0.91
	~ evap + rank	0.38	--	--	--	-0.07	29.06	8.57	0	0.86
	~ temp + rank	--	--	-0.59^	--	0.21	25.75	5.26	0.12	0.18
	Humeral length									
	~ evap + precip + temp + **age** + rank	0.41	-0.41	0.34	**1.37***	-0.26	14.49	2.83	0.83	0.05
	~ evap + precip + **age** + rank	0.73	-0.01	--	**1.47****	-0.24	13.66	2	0.85	0.02
	~ evap + temp + **age** + rank	0.70	--	0.04	**1.45****	-0.24	13.56	1.9	0.85	0.01
	~ precip + temp + **age** + rank	--	-0.74	0.62	**1.24****	-0.23	13.69	2.03	0.85	0.02
	~ evap + **age** + rank	0.73^	--	--	**1.47*****	-0.24	11.66	0	0.88	<0.01
	~ precip + **age** + rank	--	-0.05	--	**1.23****	-0.01	18.24	6.58	0.76	0.02
	~ temp + **age** + rank	--	--	0.18	**1.19****	-0.05	17.12	5.46	0.79	0.01
	~ **age** + rank	--	--	--	**1.22****	-0.03	16.31	4.65	0.80	<0.01
	Body mass									
	~ evap + **precip** + temp + **age** + **rank**	0.20	**-0.67***	0.24	**1.43*****	**-0.24***	-9.23	0	0.98	<0.01
	~ **evap + precip + age + rank**	**0.42***	**-0.39****	--	**1.51*****	**-0.22***	-5.32	3.91	0.98	<0.01
	~ **evap** + temp + **age** + rank	**0.68***	--	-0.25^	**1.57*****	-0.21^	4.49	13.72	0.94	<0.01
	~ **precip + temp + age + rank**	--	**-0.83****	**0.37***	**1.37*****	**-0.22***	-7.69	1.54	0.98	<0.01
	~ precip + **age** + rank	--	**-0.42***	--	**1.37*****	-0.09	5.21	14.44	0.95	<0.01
	~ evap + **age** + rank	0.47	--	--	**1.45*****	-0.21	10.09	19.32	0.91	<0.01
	~ temp + **age** + rank	--	--	-0.13	**1.31*****	-0.02	13.02	22.25	0.87	<0.01
	~ **age** + rank	--	--	--	**1.28*****	-0.04	12.00	21.23	0.88	<0.01

Table S3.2. Linear models for male subadults (n =). Significance level codes: ^0.1; *0.05; **0.01; ***0.001. Significant predictors at the 0.05 level are bolded. No models of GR_{max} were significant; all other combinations of predictors not listed were non-significant.

		standardized beta coefficient							linear	
	model	evap	precip	temp	age	rank	AICc	Δ AICc	R^2	p-value
gestation conditions	$GR_{overall}$									
	~ evap + precip + temp + age + rank	-0.17	0.26	0.28	0.19	0.25	35.57	2.98	0	0.52
	~ evap + precip + temp + rank	-0.20	0.28	0.39	--	0.25	34.52	1.93	0.03	0.42
	~ evap + temp + age + rank	-0.18	--	0.39	0.22	0..14	35.22	2.63	0	0.50
	~ evap + precip + age + rank	-0.03	0.32	--	0.28	0.22	34.63	2.04	0.02	0.44
	~ evap + temp + rank	-0.22	--	0.51	--	0.13	34.28	1.69	0.01	0.42
	~ evap + precip + rank	0.02	0.39	--	--	0.20	34.58	1.99	0	0.46
	~ temp + age + rank	--	--	0.23	0.25	0.08	33.86	1.27	0.04	0.38
	~ precip + age + rank	--	0.31	--	0.27	0.21	32.66	0.07	0.12	0.26
	~ precip + rank	--	0.39	--	--	0.21	32.59	0	0.09	0.26
	~ age + rank	--	--	--	0.37^	0.08	33.08	0.49	0.05	0.31
	Humeral length									
	~ evap + precip + temp + **age** + rank	-0.004	-0.05	-0.01	**1.02*** **	-0.05	23.75	7.44	0.78	<0.01
	~ evap + precip + **age** + rank	-0.01	-0.05	--	**1.02*** **	-0.05	21.76	5.45	0.80	<0.01
	~ evap + temp + **age** + rank	-0.002	--	-0.03	**1.02*** **	-0.03	21.93	5.62	0.80	<0.01
	~ precip + temp + **age** + rank	--	-0.05	-0.02	**1.02*** **	-0.05	21.76	5.45	0.80	<0.01
	~ temp + **age** + rank	--	--	-0.04	**1.02*** **	-0.03	19.93	3.62	0.82	<0.01
	~ evap + **age** + rank	-0.02	--	--	**1.01*** **	-0.02	19.97	3.66	0.82	<0.01
	~ precip + **age** + rank	--	-0.06	--	**1.02*** **	-0.06	19.78	3.47	0.82	<0.01
	~ **age** + rank	--	--	--	**1.22****	-0.003	16.31	0	0.80	<0.01
	Body mass									
	~ evap + precip + temp + **age** + rank	-0.09	-0.06	-0.03	**0.96*** **	-0.14	16.14	3.3	0.85	<0.01
	~ evap + precip + **age** + rank	-0.11	-0.07	--	**0.95*** **	-0.14	14.22	1.38	0.87	<0.01
	~ evap + temp + **age** + rank	-0.09	--	-0.06	**0.95*** **	-0.12	14.61	1.77	0.86	<0.01
	~ temp + precip + **age** + rank	--	-0.06	-0.11	**0.97*** **	-0.17	14.95	2.11	0.86	<0.01
	~ temp + **age** + rank	--	--	-0.13	**0.97*** **	-0.15	13.35	0.51	0.87	<0.01
	~ evap + **age** + rank	-0.12	--	--	**0.97*** **	-0.10	12.84	0	0.88	<0.01
	~ precip + **age** + rank	--	-0.10	--	**0.93*** **	-0.19	14.18	1.34	0.86	<0.01
	~ **age** + rank	--	--	--	**0.90*** **	-0.14	13.25	0.41	0.86	<0.01

Table S3.2 Continued

		standardized beta coefficient							linear	
	model	evap	precip	temp	age	rank	AICc	Δ AICc	R^2	p-value
first year conditions	$GR_{overall}$									
	~ evap + precip + temp + age + rank	-0.48^	0.06	0.26	0.35	0.04	32.41	1.91	0.19	0.29
	~ evap + precip + temp + rank	-0.47^	0.12	0.29	--	0.01	34.58	4.08	0.02	0.43
	~ **evap** + temp + age + rank	**-0.49***	--	0.29	0.36^	0.04	30.50	0	0.28	0.16
	~ evap + precip + age + rank	-0.34^	0.19	--	0.36	0.05	32.08	1.58	0.20	0.23
	~ evap + temp + rank	-0.49^	--	0.40	--	0.002	32.88	2.38	0.11	0.28
	~ evap + precip + rank	-0.32	0.27	--	--	0.01	34.01	3.51	0.03	0.39
	~ evap + age + rank	-0.23	--	--	0.41^	0.04	31.13	0.63	0.22	0.17
	~ evap + rank	-0.28	--	--	--	-0.01	33.78	3.28	0	0.41
	~ age + rank	--	--	--	0.37^	0.07	33.08	2.58	0.05	0.31
	Humeral length									
	~ evap + precip + temp + **age** + rank	-0.16	-0.04	0.13	**1.00*****	-0.04	22.11	4.1	0.80	<0.01
	~ evap + precip + **age** + rank	-0.10	-0.10	--	**1.01*****	-0.04	20.99	2.98	0.81	<0.01
	~ evap + temp + **age** + rank	-0.16	--	0.11	**0.99*****	-0.04	20.20	2.19	0.82	<0.01
	~ precip + temp + **age** + rank	--	0.001	0.01	**1.00*****	-0.03	22.00	3.99	0.80	<0.01
	~ evap + **age** + rank	-0.09	--	--	**1.01*****	-0.04	19.05	1.04	0.83	<0.01
	~ precip + **age** + rank	--	0.01	--	**1.00*****	-0.03	20.01	2	0.81	<0.01
	~ temp + **age** + rank	--	--	0.01	**1.00*****	-0.03	20.00	1.99	0.82	<0.01
	~ **age** + rank	--	--	--	**1.00*****	-0.03	18.01	0	0.84	<0.01
	Body mass									
	~ evap + **precip** + temp + **age** + **rank**	-0.15	0.03	-0.05	**0.92*****	-0.16^	13.23	3.69	0.88	<0.01
	~ **evap** + precip + **age** + rank	**-0.17***	0.004	--	**0.92*****	-0.16^	11.54	2	0.89	<0.01
	~ evap + temp + **age** + rank	-0.15	--	-0.04	**0.92*****	-0.16^	11.35	1.81	0.89	<0.01
	~ precip + temp + **age** + rank	--	0.06	-0.16	**0.92*****	-0.15	14.10	4.56	0.87	<0.01
	~ precip + **age** + rank	--	-0.03	--	**0.90*****	-0.15	15.15	5.61	0.85	<0.01
	~ **evap** + **age** + rank	**-0.17***	--	--	**0.92*****	-0.16^	9.54	0	0.90	<0.01
	~ temp + **age** + rank	--	--	-0.13	**0.93*****	-0.15	12.52	2.98	0.88	<0.01
	~ **age** + rank	--	--	--	**0.90*****	-0.14	13.25	3.71	0.86	<0.01

APPENDIX C

CHAPTER 4 SUPPLEMENTAL TABLES

Table S4.1. Sources for life-history and body mass data not available from the AnAge database.

species	**variable**	**source**
Avahi laniger	female maturity	(Godfrey et al., 2004)
	body mass	(Glander et al., 1992; Smith & Jungers, 1997)
Eulemur fulvus fulvus	weaning age	(Tarnaud, 2004)
	weaning body mass	(Leigh & Terranova, 1998)
Eulemur rufus	group size	(Overdorff et al., 1999)
Hapalemur griseus	weaning age	(Tan, 2006)
	weaning body mas	(Leigh & Terranova, 1998)
Lophocebus albigena	adult body mass	(Smith & Jungers, 1997)
Microcebus murinus	weaning body mass	(Zimmerman & Radespiel, 2013)
Piliocolobus badius	brain size	(Silcox et al., 2009)
	life history	(Struhsaker, 1975)
Presbytis hosei	age at maturity	(Zimmermann & Radespiel, 2013)
Trachypithecus cristatus	life history	(Shelmidine et al., 2009)

Table S4.2. Female-only sample parameter estimates for PGLS regressions of overall growth rate (OGR) against sella turcica size, body mass, and brain mass across different primate groups. Significant predictors are bolded; significance codes: 0.001***, 0.01**, 0.05*; -- predictor not included in model.

	model	**std. beta coefficient**						
	OGR ~	ST	BM	BrM	AICc	ΔAICc	adj. R^2	p-value
order Primates	ST + **BM** + **BrM**	0.06	**1.14*** **	**-0.70** **	7.69	0	0.95	<0.001
	BM	--	**0.67*** **	--	18.99	11.3	0.88	<0.001
	ST + **BM**	0.01	**0.66*** **	--	20.98	13.29	0.88	<0.001
	ST + **BrM**	0.19	--	**0.57** **	30.25	22.56	0.83	<0.001
	BrM	--	--	**0.77*** **	30.07	22.38	0.83	<0.001
	ST residuals	0.28	--	--	61.17	53.48	0	0.42
suborder Strepsirrhini	ST + **BM**	-0.001	**0.70***	--	7.06	6.14	0.87	0.02
	BM	--	**0.70** **	--	5.06	4.14	0.90	0.002
	ST + BM + BrM	-0.01	0.38	0.22	4.61	3.69	0.79	0.29
	BrM	--	--	**0.73** **	0.92	0	0.92	0.006
	ST + **BrM**	-0.004	--	**0.73***	2.92	2	0.89	0.05
	ST resid	-0.01	--	--	20.25	19.33	0	0.99
suborder Haplorhinni	**ST + BM + BrM**	**0.10***	**1.15*** **	**-0.72***	7.45	0	0.92	<0.001
	ST + BM	**0.12***	**0.65*** **	--	14.77	7.32	0.82	<0.001
	BM	--	**0.75*** **	--	12.62	5.17	0.88	<0.001
	ST + BrM	**0.42***	--	0.41	24.31	16.86	0.66	0.001
	BrM	--	--	**0.89*** **	25.54	18.09	0.68	<0.001
	ST residuals	**0.42***	--	--	38.48	31.03	0.14	0.03
parvorder Catarrhini	**ST + BM**	**0.24***	**1.11** **	--	8.93	1.81	0.81	0.002
	ST + **BM** + BrM	**0.15***	**1.10** **	-0.04	10.93	3.81	0.77	0.01
	BM	--	**0.91*** **	--	7.12	0	0.84	<0.001
	ST + BrM	**0.36** **	--	0.52	12.74	5.62	0.72	0.01
	BrM	--	--	**1.24** **	12.11	4.99	0.72	0.002
	ST residuals	**0.73** **	--	--	14.17	7.05	0.55	0.01

Table S4.3. Male-only sample parameter estimates for PGLS regressions of overall growth rate (OGR) against sella turcica size, body mass, and brain mass across different primate groups. Significant predictors are bolded; significance codes: 0.001***, 0.01**, 0.05*, 0.10^; -- predictor not included in model.

	model	**std. beta coefficient**						
	OGR ~	ST	BM	BrM	AICc	ΔAICc	adj. R^2	p-value
order Primates	ST + **BM** + BrM	0.06	**1.02*** **	-0.40	8.96	0	0.92	<0.001
	BM	--	**0.65*** **	--	15.52	6.56	0.88	<0.001
	ST + **BM**	0.13	**0.75*** **	--	15.39	6.43	0.88	<0.001
	ST + **BrM**	0.13	--	**0.89*** **	18.58	9.62	0.86	<0.001
	BrM	--	--	**0.76*** **	18.18	9.22	0.85	<0.001
	ST residuals	0.10	--	--	48.88	39.92	0	0.71
suborder Strepsirrhini	ST + **BM**	-0.12	**0.80***	--	6.63	1.08	0.79	0.04
	BM	--	**0.71****	--	5.55	0	0.81	0.009
	ST + BM + BrM	-0.8	1.94	-1.87	5.86	0.31	0.53	0.24
	BrM	--	--	**0.84***	6.49	0.94	0.61	0.05
	ST + BrM	-0.11	--	0.97^	7.88	2.33	0.48	0.08
	ST resid	-0.12	--	--	18.83	13.28	0	0.78
suborder Haplorhinni	**ST + BM + BrM**	**0.02***	**0.99****	**-0.38***	4.00	0	0.85	<0.001
	ST + BM	**0.02***	**0.73*** **	--	8.11	4.11	0.79	<0.001
	BM	--	**0.72*** **	--	6.15	2.15	0.80	<0.001
	ST + BrM	**0.07****	--	**0.99*** **	14.16	10.16	0.74	<0.001
	BrM	--	--	**0.92*** **	12.47	8.47	0.74	<0.001
	ST residuals	**0.13***	--	--	30.03	26.03	0.17	0.02
parvorder Catarrhini	**ST + BM**	**0.09***	**0.88****	--	2.10	0.54	0.76	0.006
	ST + BM + BrM	**0.09***	**0.83***	-0.07	4.07	2.51	0.71	0.02
	BM	--	**0.80****	--	1.56	0	0.75	0.001
	ST + BrM	**0.30****	--	**1.24****	4.48	2.92	0.72	0.009
	BrM	--	--	**0.84***	7.35	5.79	0.56	0.01
	ST residuals	**0.19****	--	--	15.36	13.8	0.19	0.01

Table S4.4. Model selection total growth rate including only body mass (n = 68 species). Significance codes: 0.001***, 0.01**, 0.05*, 0.10^

	standardized beta coefficient							linear model	
model			%				Δ		
TGR ~	BM	NDVI	lvs	GrpSz	SS	AICc	AICc	R^2	p-value
BM	0.74***					13.5	0	0.88	<0.001
BM + SS	0.75***				0.08	13.5	0.02	0.88	<0.001
BM + % lvs	0.73***		0.07			14.1	0.60	0.88	<0.001
BM + GrpSz	0.77***			-0.06		14.8	1.27	0.88	<0.001
BM + % lvs + SS	0.74***		0.05		0.07	15.3	1.75	0.88	<0.001
BM + NDVI	0.73***	-0.05				15.3	1.79	0.87	<0.001
BM + NDVI + SS	0.74***	-0.05			0.08	15.4	1.93	0.88	<0.001
BM + NDVI + GrpSz	0.76***	-0.06		-0.08		16.0	2.53	0.88	<0.001
BM + NDVI + % lvs	0.72***	-0.05	0.07			16.0	2.53	0.88	<0.001
BM + % lvs + GrpSz	0.75***		0.06	-0.05		16.3	2.76	0.88	<0.001
BM + GrpSz + SS	0.76***			-0.01	0.07	16.4	2.88	0.88	<0.001
BM + NDVI + % lvs + SS	0.74***	-0.05	0.05		0.07	17.4	3.87	0.88	<0.001
BM + NDVI + % lvs + GprSz	0.75***	-0.06	0.06	-0.06		17.9	4.37	0.88	<0.001
BM + NDVI + GrpSz + SS	0.75***	-0.06		-0.04	0.06	18.3	4.81	0.88	<0.001
BM + % lvs + GrpSz + SS	0.75***		0.05	-0.005	0.06	18.4	4.90	0.88	<0.001

Table S4.5. Model selection total growth rate including only brain mass (n = 68 species). Significance codes: 0.001***, 0.01**, 0.05*, 0.10^

	standardized beta coefficient							linear model	
model							Δ		p-
TGR ~	BrM	NDVI	% lvs	GrpSz	SS	AICc	AICc	R^2	value
BrM + % lvs	0.63***		0.20*			45.7	0	0.66	<0.001
BrM + % lvs + SS	0.64***		0.18^		0.07	47.9	2.25	0.65	<0.001
BrM	0.62***					48.4	2.70	0.60	<0.001
BrM + NDVI + % lvs	0.62***	-0.03	0.20*			48.4	2.75	0.64	<0.001
BrM + % lvs + GrpSz	0.64***		0.20*	-0.02		48.5	2.86	0.64	<0.001
BrM + SS	0.65***				0.11	49.4	3.76	0.61	<0.001
BrM + GrpSz	0.65***			-0.06		50.7	5.00	0.59	<0.001

Table S4.6. Model selection weaning growth rate including both brain mass and body mass as predictors across taxonomic groups. No weaning data were available for strepsirrhines. Significance codes: 0.001***, 0.01**, 0.05*, 0.10^; -- predictor not included in model.

		standardized beta coefficient								linear model	
	model	BM	BrM	NDVI	% lvs	GrpSz	SS	AICc	Δ AICc	adj. R^2	p-value
	no weaning data available for strepsirrhines										
haplorhines	WGR~										
	BM + NDVI	**0.41****	--	**-0.29***	--	--	--	26.0	0	0.57	<0.01
	BrM + NDVI	--	**0.40****	**-0.31***	--	--	--	26.5	0.48	0.56	<0.01
	BM	**0.48****	--	--	--	--	--	26.6	0.61	0.44	<0.01
	BrM	--	**0.46****	--	--	--	--	27.5	1.47	0.40	<0.01
	BM + NDVI + % lvs	**0.39****	--	-0.34^	--	--	-0.13	30.4	4.41	0.53	0.02
	BM + GrpSz	**0.47****	--	--	--	0.04	--	30.9	4.88	0.38	0.04
	BM + % lvs	**0.49****	--	--	-0.03	--	--	30.9	4.89	0.48	0.04
	BM + SS	**0.48****	--	--	--	--	-0.01	30.9	4.89	0.38	0.04
	BM + BrM	0.46	0.02	--	--	--	--	30.9	4.89	0.42	0.03
	BrM + NDVI + SS	--	**0.38***	**-0.36***	--	--	-0.13	31.0	4.99	0.55	0.02
	BM + NDVI + GrpSz	**0.39***	--	**-0.31***	--	0.09	--	31.0	5.01	0.55	0.02
	BrM + NDVI + % lvs	--	**0.41****	**-0.31***	0.10	--	--	31.3	5.31	0.54	0.02
catarrhines	WGR~										
	BM + BrM + NDVI + % lvs + GrpSz + SS	3.74	-2.93	0.03	-1.12	-0.52	-0.78	14.6	0	0.23	0.57
	BM + NDVI	0.41^	--	-0.27	--	--	--	16.3	1.7	0.49	0.08
	BM	**0.51***	--	--	--	--	--	16.8	2.2	0.41	0.05
	BrM + NDVI	--	0.35	-0.35	--	--	--	17.6	3	0.40	0.13
	BM + BrM	0.87	-0.40	--	--	--	--	18.1	3.5	0.36	0.14
	BM + % lvs	0.53^	--	--	-0.06	--	--	18.7	4.1	0.31	0.17
	BrM	--	0.41	--	--	--	--	19.2	4.6	0.21	0.14
	NDVI	-0.45	--	--	--	--	--	19.5	4.9	0.22	0.13

Table S4.7. Parameter estimates for PGLS regressions of overall growth rates (n=30) against socioecological predictor variables by taxonomic group. Significance codes: 0.001***, 0.01**, 0.05*, 0.10^; -- predictor not included in model.

		standardized beta coefficient								linear model	
	model	BM	BrM	NDVI	% lvs	GrpSz	SS	AICc	Δ AICc	adj. R^2	p-value
strepsirrhines	OGR~										
	BM + BrM + NDVI + % lvs + GrpSz	0.07	0.28	-0.13	-0.32	0.52	--	-12.7	0	0.98	0.10
	BM + NDVI + % lvs	**0.67****	--	0.13	-0.10	--	--	-2.5	10.2	0.95	<0.01
	BM + BrM + NDVI + % lvs	0.75^	-0.10	0.09	-0.12	--	--	-1.2	11.5	0.92	0.05
	BM + NDVI	**0.66*****	--	0.16^	--	--	--	-0.3	12.4	0.93	<0.01
	BM + % lvs	**0.65****	--	--	-0.14	--	--	0.95	13.65	0.91	<0.01
	BM	**0.62****	--	--	--	--	--	2.9	15.6	0.88	<0.01
	BM + BrM	**0.80***	-0.20	--	--	--	--	3.2	15.9	0.88	<0.01
	BM + GrpSz	**0.56***	--	--	--	0.10	--	3.9	16.6	0.87	<0.01
	BrM + GrpSz	--	**0.41***	--	--	0.36^	-	7.6	20.3	0.77	0.02
	BrM	--	**0.49***	--	--	--	--	12.9	25.6	0.49	0.05
haplorhines	OGR~										
	BM + BrM + NDVI	**1.32*****	**-0.57*****	**-0.09***	--	--	--	-6.1	0	0.96	<0.01
	BM + BrM + NDVI + SS	**1.30*****	**-0.55*****	**-0.09***	--	--	0.03	-3.0	3.08	0.95	<0.01
	BM + BrM	**1.37*****	**-0.63*****	**--**	--	--	--	-2.9	3.12	0.94	<0.01
	BM + BrM + NDVI + % lvs	**1.23*****	**-0.49***	**-0.10***	0.03	--	--	/2.6	3.43	0.95	<0.01
	BM + BrM + NDVI + GrpSz	**1.31*****	**-0.56*****	**-0.10***	--	-0.01	--	-2.4	3.64	0.95	<0.01
	BM + NDVI + % lvs	**0.73*****	**--**	**-0.14****	**0.15****	--	--	-0.3	5.79	0.94	<0.01
	BM + BrM + SS	**1.36*****	**-0.61*****	--	--	--	0.02	-0.1	6.00	0.94	<0.01
	BM + BrM + % lvs	**1.44*****	**-0.69****	--	-0.03	--	--	0.2	6.23	0.94	<0.01
	BM + BrM + GrpSz	**1.38*****	**-0.63*****	--	--	0.02	--	0.2	6.28	0.94	<0.01
catarrhines	OGR ~										
	BM + BrM	**0.87*****	-0.23^	--	--	--	--	-3.2	0	0.93	<0.01
	BM + BrM + % lvs	**1.04*****	**-0.39***	--	-0.08	--	--	-0.2	2.99	0.94	<0.01
	BM + BrM + GrpSz	**0.91*****	**-0.28***	--	--	0.05	--	0.0	3.23	0.94	<0.01
	BM	**0.69*****	--	--	--	--	--	0.3	3.50	0.91	<0.01
	BM + BrM + NDVI	**0.87*****	**-0.26***	-0.04	--	--	--	0.4	3.58	0.94	<0.01
	BM + BrM + SS	**0.83*****	-0.18	--	--	--	0.02	1.4	4.58	0.92	<0.01
	BM + NDVI	**0.68*****	--	-0.07	--	--	--	2.2	5.38	0.90	<0.01
	BM + GrpSz	**0.69*****	--	--	--	0.05	--	3.1	6.28	0.90	<0.01
	BM + SS	**0.67*****	--	--	--	--	0.05	3.4	6.56	0.91	<0.01

Table S4.8. Interspecific sella turcica volume descriptive statistics

species	mean	std deviation
Alouatta caraya	141.05	5.85
Alouatta palliata	164.30	19.21
Aotus trivirgatus	40.46	7.23
Ateles geoffroyi	206.38	67.23
Avahi laniger	19.52	1.38
Cacajao calvus	118.88	1.15
Callicebus moloch	29.99	9.85
Callithrix argentata	18.14	2.26
Cebus capucinus	96.72	34.55
Lophocebus albigena	108.43	13.50
Cercocebus torquatus	251.32	12.42
Cercopithecus mitis	133.24	32.43
Chiropotes albinasus	151.36	4.34
Piliocolobus badius	173.09	36.32
Colobus polykomos	202.52	32.96
Erythrocebus patas	219.82	34.46
Eulemur fulvus fulvus	16.00	3.01
Eulemur rufus	15.58	4.11
Galago alleni	22.02	0.38
Galago senegalensis	4.93	2.17
Gorilla gorilla gorilla	1753.50	199.30
Hapalemur griseus	26.77	7.38
Hylobates lar	94.02	13.36
Lemur catta	36.41	6.90
Macaca fascicularis	95.53	14.72
Macaca fuscata	310.00	22.10
Macaca mulatta	269.45	43.20
Macaca sylvanus	399.00	25.59
Mandrillus leucophaeus	410.20	68.89
Mandrillus sphinx	756.90	92.81
Microcebus murinus	7.48	1.96
Miopithecus talapoin	36.60	19.12
Nasalis larvatus	282.17	60.95
Nycticebus coucang	52.20	7.38
Pan paniscus	823.23	33.36
Pan troglodytes troglodytes	727.46	18.02
Perodicticus potto	61.18	14.72
Pithecia monachus	126.63	28.51
Pithecia pithecia	487.90	10.06
Pongo pygmaeus	974.94	127.35

Presbytis hosei	642.13	83.92
Presbytis rubicunda	529.05	87.73
Saguinus mystax	24.39	5.79
Saimiri sciureus	17.06	6.11
Sapajus apella	137.31	19.66
Symphalangus syndactylus	343.76	14.71
Theropithecus gelada	351.02	19.45
Trachypithecus cristata	139.09	34.23
Varecia variegata	59.06	15.53