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Brains, teeth and life histories in hominins: a review

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Summary - *The role of the brain in the somatic development, as well as in the establishment of the different variables of the life history pattern in vertebrates has been largely debated. Moreover, during the last thirty years, dental development has been used as a good proxy to infer different aspects of the life history in hominins, primarily due to the correlation that exists between age at first molar eruption and brain size in the order Primates. We review these questions using what is known about brain growth and maturation, dental development and life history pattern, mainly in* Homo sapiens *and* Pan troglodytes. *It has been assumed that the brain represents the pace-maker of our development. However, we consider that our particular phenotype is the result of a hierarchical genetic program modulated by epigenetic and environmental factors. The particular bauplan of any kind of organisms (e.g. primates) may explain the high correlation observed between different variables of its life history pattern, brain size or dental development. However, the correlation of these variables seems to be less reliable when dealing with low-rank taxonomical categories (i.e., species). We suggest that, while there is likely some relationship between the rate of somatic development and tooth development, our brain size and maturation (and, by extension, those of other species of the genus* Homo*) have derived towards a particular trajectory, with a unique pattern of prenatal and postnatal time and rate of growth and, particularly, with remarkable slow brain maturation. We suggest that extremely slow brain maturation could be a very recent acquisition of the last* H. sapiens *populations. Furthermore, our review of the literature suggests caution in drawing conclusions about aspects of the life history of the hominins from the information we can obtain from dental development in fossil specimens.*

Keywords - *Human evolution, Brain growth and maturation, Human life-history, Dental development.*

Introduction

Dental development, brain development and life history patterns are essential and possibly related aspects of the vertebrate biology, and they have been the subject of some primate evolutionary studies (e.g. Godfrey *et al.*, 2001). Life history theory explains how natural selection and other evolutionary forces shape organisms to optimize their survival and reproduction fitness in the face of the ecological challenges posed by the environment (e.g. Stearns, 1992, 2000; Roff, 1992). The assumed close relationship between different variables of the life history pattern and dental development in hominins has been investigated in many interesting reports (Bogin & Smith, 1996; Bogin, 1997; Bogin, 2010; Kelley & Schwartz, 2012; Lee, 2012; Smith, 1989, 1991, 1992; Smith & Tompkins, 1995; Thompson & Nelson, 2011, among others). However, some authors have seriously questioned the predictive utility of these variables, such as the permanent

gingival eruption of the first molar or the time of attainment of complete dentition, for inferring the life history pattern in primates and hominin fossil taxa (Dirks & Bowman, 2007; Robson & Wood, 2008; Guattelli-Steinberg, 2009; Smith, 2013). Certainly, while the study of the life history in living species is complex, it seems risky to make inferences in extinct species using only some data of their dental development. Furthermore, the role of the brain in life history also remains controversial (e.g. Kaplan *et al*., 2000; Deaner *et al*., 2003; Leigh & Blomquist, 2007; Robson & Wood, 2008).

As early as 1959, G.A. Sacher concluded in his comparative study of living primates that longevity is controlled by relative brain size. Further, Sacher argued that brain metabolism and energetics represent the pace-maker of vertebrate growth and ageing (Sacher, 1975, 1978). This statement was based on the physiological demands of fetal and postnatal brain growth and maturation (Sacher & Staffeldt, 1974; Martin, 1983). Furthermore, other researchers have assumed that a relatively larger brain (encephalization) was linked to a slow pace of development in modern humans (and by extension to other species of the genus *Homo*), since relative brain size seems to be highly correlated to many life history variables (Harvey & Clutton-Brock, 1985; Smith, 1989, 1992; Kaplan *et al*., 2000; Deaner *et al*., 2003). However, there is no consensus concerning this matter (Leigh & Blomquist, 2007). Harvey and Clutton Brock (1985) concluded that variation in most life history variables is highly correlated with variation in body size. However, these authors recognize that their analyses cannot distinguish whether size exerts a causal effect on life histories or whether other size related variables are involved. Thus, Harvey and Clutton-Brock (1985) discussed the effect of brain size at birth, the postnatal brain growth and the adult brain size, the former being highly correlated with other life history variables in most primate species. Later, Harvey *et al*. (1989) considered that the pace of life history is particularly determined by age-specific mortality rates. Species with low adult mortality show slower life histories, and *vice-versa.* Species with high rates of adult mortality and high probabilities of dying during the reproductive period tend to have faster life histories. In the same line, Hawkes (2006) has reported that shifts in adult survival or mortality risk modify the pace of linked life history events. The analyses carried out by Godfrey *et al*. (2001) on a large sample of living primate species, in which humans were not represented, are particularly interesting. These authors concluded that brain size is a better predictor of dental development than body size. However, cranial capacity alone is not able to account for variation in dental development. In contrast, these authors found that diet affects the absolute pace of dental development, independently, among others, from body and brain size. Furthermore, Kaplan *et al.* (2000) consider that the exceptionally long lifespan of humans, their extended period of juvenile dependence, their support of reproduction by older post-reproductive individuals, and their male support of reproduction through the provisioning of females and their offspring, are co-evolved responses to a dietary shift toward high-quality, nutrient-dense and difficult-to-acquire food resources. Konner (2010) also defend this hypothesis, but in a broader sense, assuming that we need an extra time to acquire all our complex cultural skills.

On the other hand and concerning dental development, it is also important to bear in mind whether the studies have been performed using longitudinal or cross-sectional samples. In this regard, longitudinal studies hardly match the large numbers that can be achieved in cross-sectional studies. Furthermore, longitudinal studies certainly result in a tendency in the direction of later estimations of time in teeth eruption (Dahlberg & Menegaz-Bock, 1958). Moreover, longitudinal data probably underestimate variance, whereas cross-sectional solutions should produce better estimates of variance (Smith *et al.*, 1994). A disadvantage of using cross-sectional data, at least in determining tooth emergence, lies in that the eruption time of early erupting teeth is estimated on the basis of later cohorts than the eruption time of later erupting

teeth (Parner *et al.*, 2001). Whenever possible in this review, it will be stated which sort of data sources are used in the cited references.

Summarizing, our aim is to review what we can know and what we can assume on brain growth and development in extinct hominin species. This information could help us to evaluate the nature of the relationship between dental development and brain size and maturation in hominins. Furthermore, this information may be useful in order to assess the feasibility of employing dental development as a predictor of life history variables in extinct hominin species. To address these questions it is necessary to establish a reference framework. Some information is available about brain growth in chimpanzees, our closest extant relatives (Sakai *et al.*, 2012), as well as about the neurobiological development and brain size trajectory in our own species, which seems to be unique within the primate order (Deacon, 2000; Vinicius, 2005; Somel *et al.*, 2009; Petanjek *et al.*, 2011; Miller *et al.*, 2012; Neubauer & Hublin, 2012; Rilling, 2014). Therefore, we will first present pertinent data about brain size and maturation in chimpanzees and modern humans. Later, we will explore dental development in chimpanzees, modern humans, and some extinct hominins, as well as with some aspects of the life history pattern. Finally, we will try to establish a possible relationship between brain, dental development, and life history parameters.

Features, growth and size of the brain

First of all, it is important to clarify that brain size is a term that can be expressed in mass or volume units. In palaeoanthropology, the most referred variable is endocranial volume (or its synonym, cranial capacity), which is measured in volume units (cubic centimeters, cm^3) (Neubauer & Hublin, 2012). On the other hand, brain and endocranial volumes are not equivalent terms. Endocranial volume measures the volume of the complete endocranium, including brain volume itself plus meninges volume, blood vessels and sinuses, cranial nerves and cerebrospinal fluid. Masses can be converted into volumes by dividing the mass by the specific density of the brain tissue (1.036 g/cm^3) (Blinkov & Glezer, 1968).

Among primates, humans belong to the most encephalized species. Although brain size in primates is primarily related with body size, the human brain is three times higher (Falk, 1980) and its neocortex is 3.4 times higher (Rilling & Insel, 1999) than what it would be expected for a primate of our body size. Throughout hominin evolution, a continuous increase in absolute and relative brain sizes has been observed (Neubauer & Hublin, 2012). Moreover, our brain presents about 100 billion of neurons (Cherniak, 1990), around 150,000 to 180,000 kilometres of myelinated nerve fibres and about 0.15 quadrillion synapses (Pakkenberg *et al.*, 2003). The human brain is not exceptional in its cellular composition, since it contains as many neuronal and non-neuronal cells as it would be expected for a primate brain of its size. Moreover, the human cerebral cortex holds only 19% of all brain neurons, as in other mammals (Herculano-Houzel, 2009). In spite of the large brain size of modern humans, it comprises only the 2% of total adult body weight, whereas brain consumes between 20% and 25% of its basal metabolism (e.g. Aiello & Wheeler, 1995).

It is interesting to remember some of the different hypotheses that have been proposed to explain why and how the increase in hominin brain in both absolute and relative terms occurred: 1) *the radiator hypothesis* (Falk, 1990) suggests that the expansion of the brain in *Homo* was possible by a reconfiguration of the cranial vascular supply in response to changes in hydrostatic pressure due to bipedalism; 2) *the brain growth length hypothesis* (Finlay & Darlington, 1995) proposed that brain components do not grow in mosaic, but they grow in uniform, concerted fashion, according to its own internal rules, and following a predictable allometric scaling of the different parts of the brain; 3) *the metabolic expense tissue hypothesis* (Aiello & Wheeler, 1995, 2002), maintains that brain size constrains the rate at which an individual can grow, because

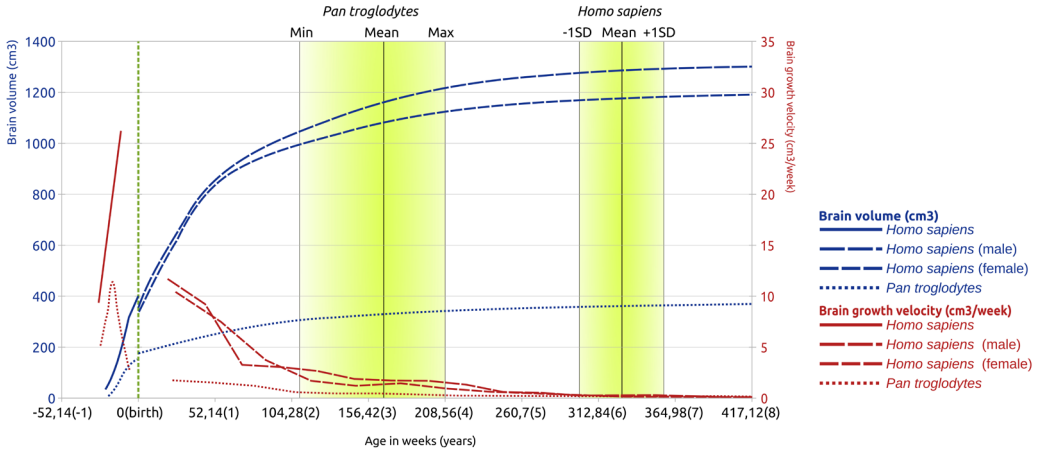

Fig. 1 - Brain volume trajectories (blue) (cm³) and brain growth velocities (red) (cm³/week) of* Homo sapiens *and* Pan troglodytes *from gestation period to the age of 8 years. Note that brain masses have been converted into centimeters of volume, dividing the grams by the typical density of brain tissue: 1.036 g/cm³ (Blinkov & Glezer, 1968). The average and variation of the age of mandibular M1 gingival emergence in* P. troglodytes *and* H. sapiens *is represented in yellow background. Data from the postnatal period were extracted from Leigh (2004), whereas data from the prenatal period were extracted from Sakai et al. (2012). The average of the mandibular M1 gingival emergence of* P. troglodytes *is from Smith* et al. *(2013) and that of* H. sapiens *is from Dean & Cole (2013). Birth is represented as a vertical dashed green line. The colour version of this figure is available at the JASs website.

it is expensive to develop the brain relative to the rest of the body in terms of the energy required; 4) *the maternal energy hypothesis* (Martin, 1996) proposes that the relative metabolic rate of the mothers during infants' gestation determines the neonatal brain mass of the neonate, while after birth, the maternal investment of the mothers during the growth of their offspring could be another factor implied in the brain development; 5) *the social brain hypothesis* (Dunbar & Shultz, 2007; Shultz & Dunbar, 2007) postulates that it was the computational demands of living in large, complex societies that selected for large brains. It is likely that a combination of some of these competing hypotheses, may explain the enlargement of the human brain throughout the last two million years of hominin evolution.

Gestation length in humans is slightly higher than in chimpanzees (38 *versus* 34 weeks respectively) (Kappeler & Pereira, 2003), so it is probable that the most important variable to set the difference in brain size at birth between humans and chimpanzees is the fetal growth rate (Neubauer & Hublin, 2012). Brain fetal rates in humans are between 9 cm³/week at the beginning of the brain growth to 26 cm³/week about two months before birth, which might represent the peak rate during fetal brain growth and posterior postnatal brain growth (Sakai, *et al.*, 2012, Fig. 1). Using three-dimensional ultrasound imaging, these authors showed that brain volume of chimpanzee fetuses at 16 weeks of gestation was only half of that in human fetuses at the same period (15.8 cm³ *versus* 33.6 cm³). Between 17 to 22 weeks, the chimpanzee and human fetal brain growth velocity increases. However, this velocity is still greater in modern humans (Sakai *et al.*, 2012). After 22 weeks, the chimpanzee fetal brain growth slows down and, at 32 weeks, the velocity of the brain in these primates would have reduced to about a 20% of the speed observed in modern humans (Sakai *et al.*, 2012). According to the estimation of these authors, the rate of chimpanzee brain growth was 4.1 cm³/week at

Tab. 1 - Mean adult and predicted neonatal endocranial volumes (cm^3) of different hominin groups and species. Predicted neonatal endocranial volume means are the result of applying a regression based on resampled data from seven catarrhine primate species published in DeSilva & Lesnik (2008) (see text for further details). This table is based on the Table 2 from DeSilva & Lesnik (2008) where readers can find the particular specimens included in each hominin group or species, as well as the references of adult endocranial volumes.

SPECIES	N	MEAN ENDOCRANIAL VOLUME ± SD (CM^3)	PREDICTED NEONATAL ENDROCRANIAL VOLUME MEAN ± SD (CM^3)	95% CI FOR MEAN NEONATAL BRAIN (CM^3)
Sahelanthropus tchadensis	1	365	148.3	130.8 - 168.2
Australopithecus afarensis	4	455.6 ± 79.4	173.8 ± 21.9	152.9 - 197.5
Australopithecus africanus	8	466.8 ± 46.4	177.1 ± 12.6	155.8 - 201.3
Australopithecus garhi	1	450	172.6	151.9 - 196.2
Paranthropus aethiopicus	1	410	161.4	142.1 - 183.2
Paranthropus robustus	2	530	194.4	170.7 - 221.3
Paranthropus boisei	9	499.6 ± 30.3	186.2 ± 7.1	163.6 - 211.8
Early *Homo*	8	651.6 ± 88.9	225.4 ± 22.4	197.5 - 257.2
Homo erectus	20	839.6 ± 138.6	270.5 ± 32.6	263.5 - 309.6
Later Asian *Homo erectus*	18	1056.7 ± 123.7	320.0 ± 27.2	279.1 - 367.1
Middle Pleistocene *Homo*	17	1218.9 ± 223.1	355.0 ± 28.7	309.1 - 407.8

about 32 weeks of gestation (just at the moment of birth), whereas in modern humans the value was 26.1 cm^3/week (at six weeks to the time of birth). That is, after 16 weeks, the human brain continued to accelerate its growth until around 32 weeks. However, it is likely that a slowdown of the brain growth rate in modern humans occurs before birth. This is because the average brain size in human newborns is 400 cm^3, and the only way to achieve this brain size is by changing the type of the growth function, from an exponential function to a logistic one. As a result, the brain of human newborns is about 2.7 times larger than that of chimpanzee newborns (about 400 cm^3 *versus* 150 cm^3) (Rilling, 2014). If we use brain weights, the average neonatal brain size in two modern human populations is about 367.7 and 381.8 grams and 150.9 grams in chimpanzees (see Table 1 in DeSilva & Lesnik, 2008).

Interestingly, and using a large data base of neonatal and mean adult brain masses of some catarrhine primates (including modern humans), DeSilva & Lesnik (2008) were able to make estimations for the neonatal brain size in hominins from the known data of adult brain size in adults (Tab. 1). From their data, DeSilva & Lesnik (2008) obtained the following regression equation: log (neonatal brain mass) = 0.7246 x log (adult brain mass + 0.3146; r^2=0.97). Considering the 95% confidence interval (CI), these authors estimated that neonatal brain-size of Lucy's children (*Australopithecus afarensis*) would have been 158.5 cm^3, with a range of 139.7 to 179.9 for the 95% CI. This is the same estimation as for *Pan*, since Lucy's brain was not larger than 400 cm^3. For other australopiths, DeSilva & Lesnik (2008) obtained values of 179.8±14.4 cm^3 (158.1-204.5 cm^3, 95% confidence interval).

In early *Homo* (e.g. *H. habilis*: about 650 cm³ for adults) the values are 225.4±22.4 cm³ (95% CI: 197.5-257.2 cm³). In early *H. ergaster/H. erectus* (about 840 cm³ in the adults) the values for the neonate brain are 270±32.6 cm³ (95% CI: 263.8-346.5 cm³). In late Asian *H. erectus* (about 1056 cm³ in adults), the values are 320±27.2 cm³ (95% CI: 279.1-367.1 cm³). Finally, for Middle Pleistocene hominins (about 1219 cm³ in adults), the values are 355.0±28.7 cm³ (95% CI: 309.1-407.8 cm³). Therefore, DeSilva & Lesnik (2008) observed that the brain size at birth in modern humans is the expected given our adult brain size. Thus, humans are not "special" for this feature, in contrast to Martin (1983)'s conclusion that humans have smaller brains at birth than expected given their adult brain size.

Apart from these estimations, and in relation to other living hominoids, we have at birth the largest absolute brain size and the smallest relative brain size compared to adult brain size (Neubauer & Hublin, 2012). Brain size at birth in humans cannot achieve larger sizes due to the energetic and obstetric constraints, meaning that a large portion of our brain growth must take place postnatally (Neubauer & Hublin, 2012). It is said that the human brain maintains a high fetal-like growth rate into the first or the two first postnatal years (Martin, 1983; Leigh, 2004). However, according to the estimations observed in Figure 1 we consider that the range of the fetal growth rates is only maintained until the end of the first postnatal life. After this time the rates of the brain growth are lower than those observed during prenatal life.

The percentage of adult brain size in modern humans is about 28% regarding the neonatal brain size, and 37% approximately in chimpanzees (data from Marchand, 1902: see Robson & Wood, 2008 and DeSilva & Lesnik, 2008). However, variation in these percentages ranges from 23.7% to 31.0% in modern humans, and from 30.0% to 40.0% in chimpanzees (see Vinicius, 2005 and references therein). For practical reasons, for this review we will use values of 28% and 37% for humans and chimpanzees, respectively.

On the other hand, and from a geometric morphometric perspective, endocranial ontogeny (i.e. changes in shape and size in time) in modern humans, chimpanzees and Neanderthals has been also studied. Some authors have noted significant differences among these hominins (Ponce de León & Zollikofer, 2001; Bruner *et al.*, 2003; Neubauer *et al.*, 2009, 2010; Gunz *et al.*, 2010). Modern humans exhibit a nonlinear endocranial ontogenetic trajectory, where three different shape-shift phases have been identified. Nevertheless, considering separately cranial vault and cranial base trajectories, modern humans display a nearly linear trajectory while the chimpanzees and Neanderthals exhibit a bounded trajectory (Neubauer *et al.*, 2009). These authors concluded that shape shifts driven exclusively by brain growth cannot be supported. Comparing modern humans' and chimpanzees' endocranial shapes at birth, the differences are evident, with no overlap throughout ontogeny, and displaying endocranial shape trajectories that significantly differ after birth (Neubauer *et al.*, 2010). In modern humans, parietal and cerebellar regions expand relatively in the so-called "globularization phase", which involves a flexion of the cranial base within the first postnatal year. This phase is uniquely present in humans, and does not occur either in chimpanzees (Neubauer *et al.*, 2010) or in Neanderthals (Gunz *et al.*, 2010).

During early infancy humans undergo a very fast increase of the white matter, in comparison to chimpanzees (Miller *et al.*, 2012). Human adult brains reach the 3.3-fold of neonate brain mass, whereas chimpanzee brains only reach a 2.5-fold. On average, modern humans achieve 90% of adult brain size by the age of five, whereas in chimpanzees this percentage is reached one year earlier. On the other hand, brain growth in chimpanzees may cease as late as 5 years of age, whereas in modern humans adult brain size is attained at about 5-6 years (Leigh, 2004). In this review we will consider that *H. sapiens* attains the adult size at about 6 years. Therefore, the idea that our larger brain is related to a longer development regarding great apes is not completely supported by the data. One year of difference

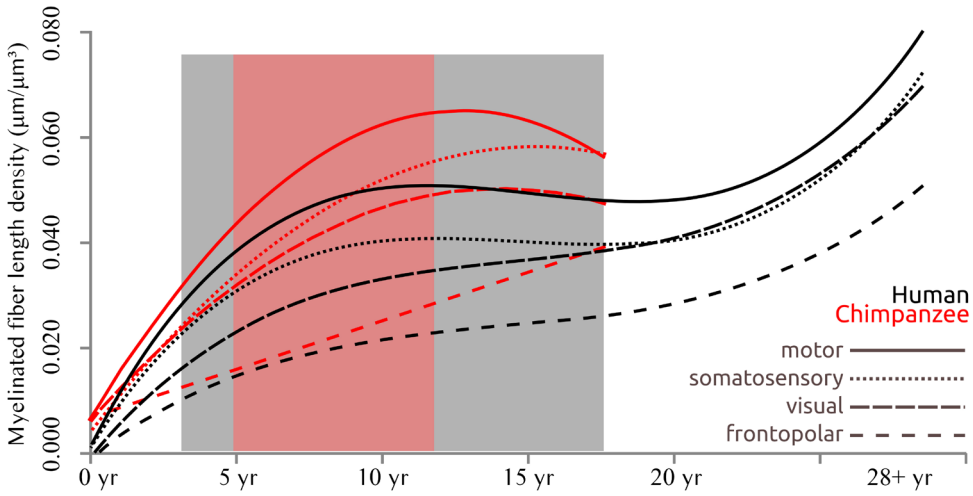

Fig. 2 - Myelinated fibers length density (μm/μm³) changes with age and from birth in four regions of the brain in* Homo sapiens *(black) and* Pan troglodytes *(gray): motor, somatosensory, visual and frontopolar areas. Dark gray and dark red areas in the background represent the period between weaning age and the acquisition of full sexual maturity in modern humans and chimpanzees respectively. Modified from Miller* et al. *(2012). The colour version of this figure is available at the JASs website.

between the ages at which both species achieve the adult brain size cannot completely explain such a brain size difference, particularly because they display almost the same brain growth velocity between 5 and 6 years old (Fig. 1). Thus, an important conclusion drawn from previous studies is that our larger brain is fundamentally a consequence of a higher rate of growth during gestation and the first months after birth, and probably not a matter of a longer growth period.

Brain development and maturation

The process of brain development implies different steps beginning with the birth of progenitor cells, followed by differentiation, migration of the cells to their final location, growth of axons and dendrites, dendritic branching and synapses, synaptic pruning, and myelination. This is a very complex process regulated by a developmental program that is still not fully understood in modern humans (Somel *et al.*, 2009; Cubelos *et al.*, 2010; Petanjek *et al.*, 2011; Miller *et al.*, 2012).

It is well known that the myelin sheath is essential to the development of the neurological function. Myelination has a crucial role in regulating the functional activity of axons (Wake *et al.*, 2011), and the density of myelinated axons allows to assess the relative maturity of the brain areas. Myelination responds to electrical excitation series of molecular-dependent-activity cascades, significantly increasing nerve impulse transmission speed (Wake *et al.*, 2011). Miller *et al.* (2012) compared the developmental trajectories of the myelinated fibers length density in four regions of the brain in chimpanzees and humans, namely the primary somatosensory area, primary motor area, most rostral part of the prefrontal cortex, and the prestriate visual cortex (Fig. 2). Their results revealed lack of myelination in the neocortex of human newborns, whereas about 20% of these areas were already myelinated at the time of birth in chimpanzees. In our closest relatives the density of myelinated axons reaches its maximum level at approximately the time of sexual maturity in most cortical regions, although Miller *et al.* (2012) have observed a gradual increase of the density of the myelinated

fibers length until about 17 years in the frontopolar area. Knickmeyer *et al*. (2010) obtained similar results in macaque monkeys, suggesting that the growth of cortical myelination is largely completed at puberty in primates. In contrast, Miller *et al*. (2012) observed that humans display lower myelination during childhood, implying a delayed period of maturation that extends beyond late adolescence and into the third decade of life. Therefore, our brain maturation is characterized by a particular (and probably unique) prolonged neocortical maturation. Much of our postnatal brain expansion results from the growth of white matter underlying the neocortex (Miller *et al*., 2012). Furthermore, these authors have found that neocortical dendritic development and synaptogenesis is not homogeneous, with the greatest delay in maturation characterizing the prefrontal cortex. Similarly, synaptic pruning in human prefrontal cortex is delayed until the age of 30 (Petanjek *et al*., 2011).

On the other hand, recent structural magnetic resonance imaging (MRI) studies have demonstrated that our species has relatively more association cortex than primary sensory and motor areas compared with other primates (Rilling, 2014). This means that a relatively larger surface of the human cortex is specialized in conceptual and other forms of higher-order cognitive processes in contrast to perceptive processes. Moreover, Gogtay *et al*. (2004) have suggested that brain subregions follow temporally distinct maturational trajectories in which higher-order association cortices mature after lower-order somatosensory and visual cortices, whilst brain areas which are phylogenetically older mature earlier than newer ones. This hypothesis might be of interest since time and order of the brain subregions maturations are two variables to keep in mind in our review. Interestingly, Brenna & Carlson (2014) have observed an elevated concentration of docosahexaenoic acid (DHA) in the brain of modern humans, particularly between about half of the gestation time and two years of age. DHA, which is an essential component required for lower and high order functions in humans, is provided by the dietary preformed DHA in the breast milk of modern human mothers. The presence of this molecule coincides with the periods when human brain growth velocities are higher. It is possible that a larger brain in modern humans requires a longer period of development for attaining adult skills (Deaner *et al.*, 2003).

An extremely delayed neocortical myelination in humans, as well as a long phase of developmental reorganization of cortical neuronal circuitry, can produce certain vulnerability to psychiatric disorders, particularly during adolescence (Andersen, 2003; Petanjek *et al.*, 2011; Miller *et al.*, 2012). However, this is at the same time an extremely effective process to increase our species-specific cognitive abilities. A remarkable delay in the developmental schedule of the human neocortex provides the opportunity for the increase of new connections, favored by social and environmental interactions, and assuming that this represents an adaptive advantage (Konner, 2010). Therefore, it is interesting to question whether other species of *Homo* had the same pattern as our species, or whether these species had a different genetic program for brain development. Unfortunately, the possible relationship between the age of neocortical myelination and dental developmental variables, either eruption ages or formation times of the enamel, remains unknown. In this context, we could speculate whether Neandertals had a brain developmental program similar to that of modern humans (see Bruner, 2004; Gunz *et al.*, 2010). It is possible that certain aspects, such as our strong ability to develop and use symbols, has a relationship with a dramatic delay in brain maturation. If so, our species would have had an additional advantage for its competitive success and subsequent population expansion as the only living hominin (but see Villa & Roebroeks, 2014).

Dental development

It is said that dental development is highly heritable, relatively resistant to starvation and other health problems (e.g. Turner *et al.*, 1991;

Bailey, 2008). Nevertheless, to what extent environmental factors influence dental development remains controversial. Heritability is defined as a measure of the degree of genetic control of a phenotype, ranging from 0% (which means no genetic effects) to 100% (which means no environmental factors) (Towne *et al.*, 2002). It has been published that the timing of tooth formation (calcification) and dental emergence were more highly correlated within monozygotic twin pairs than dizygotic twin pairs, suggesting a heritability of 0.85-0.90 (Garn *et al.*, 1960). Moreover, tooth size may be influenced to some extent by environmental factors (e.g. Perzigian, 1984). Thus, human tooth dimensions from Australian Aboriginals display about 64% of heritability, while a further 6% was due to common environment (Townsend & Brown, 1978). Concerning molar cusps, it seems that the number of cusps and the fissure pattern are under genetic control (Krogman, 1967; Towne *et al.*, 2002), while others suggest a low component of hereditary variability (Biggerstaff, 1975). Amongst environmental factors, socioeconomic status may be one of the factors responsible of its variation (Garn & Bailey, 1978). Therefore, depending on the dental traits studied, heritability ranges from 0.19 to 0.92 (Bailey, 2008). This means that although genetic effects are undoubtedly present throughout dental development, environmental factors cannot be neglected, and to what extent they participate is still under discussion.

In spite of these concerns, teeth are the most abundant and best-preserved elements of the fossil record, becoming a significant evidence to extract palaeobiological information. For several decades dental development has been considered as an excellent growth marker (e.g. Bermúdez de Castro *et al.*, 1999; Bromage & Dean, 1985; Bromage, 1987; Conroy & Vannier, 1987; Dean, 2007; Dean *et al.*, 2001; Dean & Vesey, 2008; Guatelli-Steinberg & Reid, 2008, 2010; Macho & Wood, 1995; Ramirez Rozzi & Bermúdez de Castro, 2004; Reid & Dean, 2006; Reid *et al.*, 2008; Smith, 1986) and the main tool we have to infer aspects of the life history pattern in hominins (e.g. Smith, 1989, 1991, 1992, 1993; Smith & Tompkins, 1995; Bogin & Smith, 1996; Bogin, 1997). According to Smith (1989), tooth development must be totally integrated into the plan of growth and development of a species. Therefore and according to this author, age of dental maturity ought to be used as a measure of primate life history (Smith, 1989).

First of all, it is important to remember that the anterior and posterior dentitions have distinct developmental trajectories (e.g. Smith, 1994; Bermúdez de Castro *et al.*, 1999). Both the developmental time and gingival emergence of the incisors are very similar in *H. sapiens* and *Pan* (Dean & Vesey, 2008; Dean & Cole 2013). The human canine is small and is integrated within the anterior dentition, whereas in *Pan* the so-called C/P3 honing complex (Haile-Selassie *et al.*, 2004) has a different function. In contrast, the total formation time of the three molars is greater in *H. sapiens* than in *Pan*, mainly due to slower rates of root extension in our species (Dean & Vesey, 2008). The average of the M1 gingival eruption in *H. sapiens* occurs at about 6.3 years, whereas this event occurs at about 3.2 years in *Pan* (Dean & Cole, 2013; Smith *et al.*, 2013). Nevertheless, it is important to consider the range of variability of this important process, since in modern humans there are records for M1 eruption between 4.8 to 7.0 years (Liversidge, 2003) and between 5.2 to 7.4 years (Dean & Cole 2013). In *Pan* (captive chimpanzees), the variability of this event ranges between 2.7 to 4.1 years (Smith *et al.*, 2007). In *H. sapiens* third molar (M3) root completion occurs about six years later than in *Pan*. However, and due to the fact that M3 eruption in our species is delayed nearly until the time of root apex closure, the M3 eruption in *Pan* occurs nine years earlier than in *H. sapiens* (Dean & Cole, 2013). Furthermore, the timing of the gingival emergence and the onset of the crown formation of the M2 and the M3 are remarkably delayed in *H. sapiens* with regard to *Pan*. Thus, the onset of the M2 in chimpanzees occurs at about 1.7-1.8 years, whereas this event occurs at about 3.0 years in modern humans. The onset of the M3 in chimpanzees occurs at about 3.7 years, whereas

this event is delayed until 8.0 years in modern humans (Reid *et al.*, 1998; Reid & Dean, 2006; Smith *et al.*, 2007; Dean, 2010). The extra-time that modern humans use in the development of the third molars coincides with our adolescence period (e.g. Bogin, 2010).

Life history theory

Life history theory tries to explain how natural selection and other evolutionary forces shape organisms to optimize their survival and reproduction in the face of the ecological challenges posed by the environment (e.g. Roff, 1992; Stearns, 1992, 2000). This theory also tries to find statistical correlations between different variables with a meaning of cause and effect, as well as to identify the judge that governs the pace of the life history pattern of mammals or specific groups of mammals. Thus, it has been shown that adult brain size is correlated with many life history variables (Harvey & Clutton-Brock, 1985; Sacher, 1975).

Natural selection acting on species with a common "bauplan" (e.g. primates) can produce a similar evolutionary response. This could be the reason for the high and significant correlations between variables of the life history pattern found in particular groups (Harvey & Clutton-Brock, 1985). It is evident that a slowdown of different physiological processes can produce a synchrony in the delay of the gestation length, age at weaning, age at sexual maturity, age at first breeding, age at complete dentition, age at complete somatic development or the lifespan.

Robson & Wood (2008) extensively reviewed the evidence we have about the evolution of hominin life history. These authors distinguish between life history variables (LHVs) and life history related variables (LHRVs). The former, includes variables such as gestation length, age at weaning, age at first reproduction, interbirth interval, mean life span and maximum life span; the latter includes variables such as body mass and brain mass in both adults and newborns, dental crown and root formation times and dental eruption times. According to Robson & Wood (2008), the LHVs reflect population vital rates and the timing of life history events. In contrast, LHRVs are variables that have been shown to be empirically constrained or correlated with LHVs.

An evolutionary life history model was presented by Charnov for mammals (1993). His model explains life history allometry by assuming that the age at maturity is primarily related to adult mortality rates. When these rates are high animals are expected to mature rapidly, thus maximising their lifetime reproductive success. This model also predicts that animals that mature late will have a larger body size, indeed optimizing their fecundity. Several predictions were also made by Charnov concerning the type of relationships that would be present among several life history variables, such as body weight and mortality and reproductive rates. However, there is not a consensus regarding Charnov's model which, according to some authors (Ross & Jones, 2004) it may only be appropriate in certain limited situations. Moreover, it is well known that primates (and great apes in particular) have slow life histories, with comparatively longer life stages, later ages at maturity, lower birth rates with smaller litter sizes, and longer adult life spans (Charnov, 1991, 1993; Charnov & Berrigan, 1993). These authors pointed out that primates are unusual among mammals, since they have an allometric exponent greater than 0.25 for alpha versus body weight, where alpha might be either female juvenile period length or age at first reproduction. Primates would be different from other mammals since they have very low production rates, which comes along with longer times to grow both females and infants, late maturation and slow breeding rates (Charnov, 1993).

Body size is correlated with most aspects of life history, where mammals (including primates) with greater body sizes tend to have longer gestation time and larger babies, a late age of weaning and first birth, and a longer lifespan (Harvey & Clutton-Brock, 1985; Purvis *et al.*, 2003). However, these correlations with body size may not be always entirely simple. Thus, Robson & Wood (2008) pointed out that gorilla

females, with a mean body mass of around 95 kilogrammes, reach maturity considerably faster than modern humans. When we apply these concepts to the fossil record other sources of uncertainty arise. Thus, sex estimation or body weight is very problematic when we have only a handful of fossils (Hillson, 2014).

Brain development and life history pattern

The relationship between a relatively large-brain with a slow life history, and the significant correlation between many life history variables with the size of the brain has led many scholars to assume a close physiological relationship between brain size and the pace of life history (e.g. Kaplan *et al.*, 2000). In this regard, the relationship between brain size and body size (encephalization) has been linked to the slow pace of development in our species. This concept generated the so-called "brain allometry extension" theory, which postulates a progressive extension of a conserved primate brain allometry into postnatal life of the human lineage. Vinicius (2005) does not agree with this theory, which was born from the evidence that in macaques (*Macaca mulatta*) the brain size of the neonates represents 70% of the adult brain size, and it is between 40% to 50% in chimpanzees, whereas in modern humans the value is about 23% (Schultz, 1941). As stated above, some authors have observed an overlap of these percentages in *Pan* and *H. sapiens* (see Vinicius, 2005 and references therein). Vinicius (2005) corroborates a moderate duration of brain growth in modern humans relative to our closest extant relatives. Furthermore, this author considers that encephalization of *H. sapiens* is related to a significant retardation in early postnatal body growth in comparison with other mammals, which affects the relationship between brain and body size. In fact, and according to Deacon (2000), human encephalization is a complex combination of changes in both brain and body growth. This combination seems to be exclusive of the hominin lineage. Although the number of neurons and glial cells in the human brain is proportional to its size and, therefore, our brain is a scaled-up primate brain in terms of the cell numbers (Azevedo *et al.*, 2009), its growth rate during the gestational period and after birth is remarkably greater than in other primates. Whereas neurogenesis can continue throughout our lives (e.g. Fallon *et al.*, 2000), brain growth after birth is largely due to the growth of dendrites. This process increases dramatically our brain size until the age of five, when we reach about a 90% of the total brain volume. This percentage is reached in chimpanzees at the age of four. Furthermore, the postnatal period would be responsible for about 65% to 70% of the total brain growth in both humans and chimpanzees (Vinicius, 2005). In consequence, most of the large human brain size is attained via rate changes during the gestation and after birth, particularly during the first 18 months of life. After this period, the *Homo* and *Pan* brain growth rates are not substantially different (Leigh, 2004).

The human brain is considerable larger than expected for a primate with our body mass. Since the brain tissue is metabolically very expensive (e.g. Foley & Lee, 1991; Leonard & Robertson, 1992; Fonseca-Azevedo & Herculano-Houzel, 2012), the increase in brain size would imply an increase of approximately 8% over the basic metabolic rate. However, the biological explanation of this increase is difficult to understand in terms of energy efficiency. The expensive tissue hypothesis seeks to provide a coherent explanation for this phenomenon. This hypothesis maintains that human guts were reduced in size in order to compensate for the energetic costs of the relatively large brain (Aiello & Wheeler, 1995; Aiello, 2007). The recent results of Kuzawa *et al.* (2014) support the hypothesis that unusually high costs of the brain development in our species is significantly related to a decrease in the rate of childhood body growth. There are also some implications related to life history when considering the expensive tissue hypothesis. Daily energy expenditure, which includes lactation period, is estimated to have been almost 66% higher (in average) in a *H. ergaster* female

than in the australopithecine or paranthropine females (Aiello & Key, 2002). This high-energy cost per offspring could have been compensated by decreasing the interbirth interval, which implies a reduction of the high-cost lactation period. Furthermore and due that weaned offspring in social species may come in competition with adults for food resources, a slowed growth would reduce the daily energy requirements of the immature individuals (Janson & van Schaik, 1993). Natural selection may have also favoured slowed growth in human children in order to protect maternal fitness, because they are partially dependent on the mother for food during the childhood and juvenile period (Wells, 2003).

Thus, we agree with Robson & Wood (2008) that the range of variation of the different life history variables, such as those related with dental development, would not be independent of the life history. We acknowledge the strong and significant correlation that exists among several life history variables, which suggest interdependence of population vital rates (Hawkes, 2006). But we also agree with Robson & Wood (2008) that the LHVs of a particular species may change or be altered in order to address a specific biological problem. Or, in other words, natural selection can shape ontogeny to face problems arisen in specific circumstances. It is reasonable to admit that the high brain growth rates during gestation and during the first months of postnatal life are closely linked to the energy requirements of the mother. This situation can be causally related to female body weight, gestational length, weaning age or other life history variables (Harvey & Clutton-Brock, 1985), as well as to the retardation in early postnatal body growth (Vinicius, 2005). However, this does not imply the need to accept the hypothesis of the general trend in mammals (or in primates, in particular), which has been popularly referred to as "live fast, die young" and "live slow and die old" (e.g. Promislow & Harvey, 1990; Smith & Tompkins, 1995; Bonduriansky *et al.*, 2008; Lahann & Dausmann, 2011). As seen above, our trajectory of brain growth is different from that of chimpanzees in both the rate and the time. It is obvious that regarding the increase in brain size, the strategy of present *H. sapiens* populations is different at least with respect to other primate groups and previous hominins.

Newborn and adult brain sizes in different primate species have been useful in order to compare brain growth patterns with some life history variables. In addition to brain size data, more recent research has revealed interesting and significant differences between *H. sapiens* and other primate species regarding the timing of the developmental events we have explained above, such as birth of progenitor cells, differentiation, migration of the cells, growth of axons and dendrites, dendritic branching and synapses, synaptic pruning, and myelination (e.g. Somel *et al.*, 2009; Miller *et al.*, 2012, Rilling, 2014). These differences offer an additional perspective that should be considered in the life history theory and to explain our modern human "uniqueness".

Concerning brain maturation, recent research points out that the time (not the timing) of events of the modern human brain follows a unique and very different pattern regarding other primates. Sexual maturity in primates is a life history variable which may have a relation with brain development. Thus, while synaptic pruning ends at about sexual maturity in chimpanzees, developmental remodeling of modern human brains, including a great elimination of synaptic spines, continues throughout the third decade (Petanjek *et al.*, 2011). Furthermore, it has been clearly demonstrated that chimpanzees and humans have dramatically diverged in the shape of the developmental trajectory of the myelinated fibers length density (Fig. 2) (Miller *et al.*, 2012). In our species the extension of this process goes far beyond sexual maturity and adolescence, and extends well into the period of somatic maturity, at least until the end of the third decade (Petanjek *et al.*, 2011). The molecular mechanisms responsible for this extreme delay in humans are not well understood yet (Miller *et al.*, 2012). Unfortunately, it is not possible to reconstruct the developmental trajectories of the myelinated fiber length density in extinct *Homo* species. Since myelination is crucial for

connectivity to facilitate rapid and synchronized information transfer, we can establish a relationship of the process with higher–order cognitive functions (Yakovlev & Lecours, 1967). Similarly, Somel *et al*. (2009) have reported a remarkable delay in the expression of genes related to the prefrontal cortex in modern humans compared to chimpanzees and rhesus macaques. This delay in the brain maturation exceeds the delay in other life history variables, thus implying that natural selection has produced a different shift of brain maturation in *H. sapiens* with respect to other primate species.

Dental development and brain development

Gingival emergence is defined as the gingival penetration of any tooth and thereby its visibility in the oral cavity (Oziegbe *et al*., 2014). Following this definition, we can never observe this event in fossils, because soft tissues are not preserved. Thus, different concepts of eruption in the global process of tooth emergence have been used when studying hominin fossils (Dean, 2007; Smith & Buschang, 2009; Bermúdez de Castro *et al*., 2010; Supplementary Information in Smith *et al*., 2010; Kelley & Schwartz, 2012). With this idea in mind and the appropriate caution, we can analyze the possible relationship between the emergence of the M1 and brain size, which is one of the most recurrent inferences in the study of dental development in hominins.

Smith (1989) published a database on the M1 age of eruption and brain weight in 21 primate species. She found a high and significant relationship between both variables (r=0.98), as well as between the age of M1 eruption and ten life history variables (see also Smith, 1992). Smith (1989) also found a high relationship between the same life history variables and the age of complete dentition (molars). However, it is important to note that this high correlation may be due to the study within a high order taxonomic category. It would be interesting to obtain comparative results in the study of the same aspect either within a more restricted taxonomic category (e.g. hominins) or within a determined species or population (e.g. *H. sapiens*). Thus, It is interesting to note that in the first report suggesting that early hominins had growth periods similar to the modern great apes (Bromage & Dean, 1985) *Australopithecus*, and early *Homo* specimens (KNM-ER 820 and SK-74b) cannot be distinguished in this important aspect of their biology. However, we know that the brain of early *Homo* was significantly greater than in *Australopithecus* (e.g. Aiello & Dean, 1990), that is, dental development alone seems to not be enough to estimate brain size and the life history pattern of the hominins.

Also interesting is to note that gingival emergence of the lower M1 in captive chimpanzees occurs, in average, at about 3.17 years with a range from 2.14 to 3,99 years, n=53, (Dean & Cole 2013; Smith *et al*., 2013). Since the values in both wild and captive chimpanzee populations overlap extensively (Smith & Boesch, 2011), we did not distinguish between the two (Smith *et al*. 2013). Independently of the group selected, gingival emergence of the lower M1 in *Pan* occurs before the attainment of 90% of the total brain size, and far from the age that is estimated when full brain volume is achieved (5 years according to Leigh, 2004) (Fig. 1). In modern humans, the ±2SD for the gingival emergence of the M1 is 5.2 to 7.4 years, with an average of 6.3 years (Dean & Cole, 2013). That is, the emergence of this tooth in modern humans occurs after the attainment of the 90% of the total brain size, and close to the attainment of the full brain volume, which is estimated in 6 years (Leigh, 2004).

It is interesting to note that gingival emergence of the M1 in modern humans occurs with a length of the root of about 8.0 mm, whereas in *Pan* the length of the root at the time of gingival emergence is about 4.0 mm (Dean & Vesey, 2008). However, the average of the timing of peak velocity in the root formation of M1 is 4.9 years in modern humans, whereas in chimpanzees this event occurs at about 3 years (see Fig. 7 in Dean & Cole, 2013). According to these authors, the advanced timing of peak root velocity in the root

of the M1 in chimpanzees could explain the advance of the gingival emergence of this tooth in these primates (and for extension to earlier hominins) in relation to *H. sapiens*.

Although the age at M1 eruption and the age at which complete dentition is attained in *H. sapiens* and clearly integrated with the somatic development, it is evident that these events have been compared only with the brain size. However, we are unable to know the maturation stage of a particular extinct species at the age of the M1 eruption or throughout the duration of the complete tooth development. Nevertheless, it is interesting to note that the onset and offset of the M2 and, particularly, of the M3 development is delayed in *H. sapiens*, as well as in some Early Pleistocene hominins (Bermúdez de Castro *et al.*, 1999, 2010). Modern humans complete their dentition beyond 18 years, whereas in chimpanzees this process ends at 12 years.

Dental development and life history pattern

The diagram of life history stages proposed by Schultz (1960) distinguishes five periods, where the dividing line between the infantile and juvenile periods is marked by the eruption of the first molar in the permanent dentition, and the line between juvenile and adulthood periods is defined by the emergence of the third permanent molar. As it has been previously stated, first molar eruption ages are highly correlated in primates with birth weight, age at weaning and female age at first birth, neonate brain weight and adult brain weight, whereas third molar emergence is correlated with the former ones, as well as with the body weight and the age of sexual maturity (e.g. Smith, 1991). Finding hominin fossils at exactly the moment when the M1s were emerging are rare. Some key specimens belonging to *Afropithecus*, *Sivapithecus*, *Dryopithecus*, *Australopithecus* and *Paranthropus* might have shown younger first molar emergence ages compared to modern humans, but within chimpanzee variation (Kelley, 2004; Hillson, 2014).

One of the most studied and complete early *Homo* specimens, in which it is possible to estimate the age-at-death by using dental histological variables is KNM-WT-15000 (Nariokotome boy). This African specimen dating back 1.53±0.05 million years (Brown & McDougall, 1993), died when he was between 7.6 and 8.8 years old (Dean & Smith, 2009). At this age, the Nariokotome boy had already emerged his second permanent molars, about 4 years earlier that the average in modern humans.

The length of the cheek tooth row increases when the first molars emerge in the oral cavity, suggesting that this enlargement of available dental area increases the ability to process solid food (e.g. Humphrey, 2010). Godfrey *et al.* (2003) defined dental precocity for any given age as the number of permanent and deciduous teeth, expressed as a ratio of the total number of postcanine teeth that could eventually erupt. These authors found that the most important predictor of dental precocity is the cranial capacity, which would suggest that primate species with small brains in relation to body size were likely to be dentally precocious. Furthermore, these authors also found that species with a longer gestation period tended to be more dentally precocious, while age at weaning was less important as a predictor of dental precocity.

Interestingly, the number of days between successive striae of Retzius (the so-called periodicity) has been included as a potential dental developmental variable to figure out some life history aspects in a diverse group of primates (Bromage *et al.*, 2009, 2012). Actually, these authors referred to the interval of Retzius periodicities as repeat intervals, finding statistically significant correlations in primates between RI and some life history traits on the one hand, and between RI (Retzius interval) and basal and specific metabolic rates on the other. The results of these authors suggest that RI correlates with body mass, birth weight, gestation length, lactation length, interbirth interval, age at sexual maturity, age at first breeding, lifespan, neonatal brain weight and adult endocranial volume. All of these correlations, excepting estrous cyclicity, share a dependence upon body

mass, suggesting that some aspect of metabolism may be responsible for periodic energy allocation at RI timescales. Moreover, this periodic rhythm of RI with body mass and other life history traits might be also aligned with hypothalamic-mediated neurosecretory anterior and posterior pituitary outputs, suggesting that these physiological and biochemical routes should be also taken into account when trying to elucidate life history variables from dental histological traits.

Depending on the hominid species we consider, the type of erupted teeth at the age of weaning and at the age when females give first birth is diverse, and thus, it is difficult to discern a pattern for these two variables (Hillson, 2014). Regarding age at weaning, modern humans display all the deciduous dentition in the oral cavity during this process, with no presence of permanent dentition. On the other hand, chimpanzees and gorillas present not only the whole deciduous dentition, but also the first permanent molars already erupted, whereas orangutans have much of their permanent dentition erupted or in process of eruption, with the exception of the canines and the third molars. As far as the first offspring birth in females is concerned, the eruption age for permanent third molars coincides reasonably well in modern humans, whereas in gorillas it takes place a little afterwards. In contrast, in both chimpanzees and orangutans the full permanent dentition occurs about two years, and even more, after the age when females give birth for the first time. Therefore, it is difficult to argue that dental eruption is a good proxy to establish age at weaning and age at first birth, at least in hominids (Robson & Wood, 2008).

Conclusions

From the information provided in the preceding paragraphs it seems reasonable to be cautious when using dental developmental variables to infer aspects of the life history pattern, especially when we deal with low-rank taxa (e.g. species). Much more information is needed in order to figure out the real variation of the whole set of variables involved in life history, including those ones from dental development. Longitudinal studies in a relative large sample of modern humans, chimpanzees and other primate species are needed to define the overall number of variables, which are related to the life-history pattern. Furthermore, it seems necessary to point out that multivariant analysis might elucidate the relationship among the life history variables and the life history related variables.

Regarding the discussion about the main pace-maker (e.g. the brain) of the course of development, it seems more reasonable to assume that the final phenotype of an organism (including morphological, physiological, or behavioural aspects) is the result of the development of a hierarchical genetic program. During the achievement of this program there is a wide range of epigenetic interactions between the different processes affecting all the aspects of growth and development. In spite of these interactions, it is possible to note a high correlation in some variables related to brain, dentition, life history, etc., especially when we deal with high-rank taxonomical categories. Natural selection may act producing changes in the genetic program, in order to face environmental challenges, thus altering the rate, time, timing, or the onset and offset of the different processes (heterochronies). Furthermore, environmental factors are responsible for the normal variation we observe in a particular species concerning all the variables we are able to examine. Thus, it is not realistic to expect a correlation of 1.0 or close to 1.0 between different variables, when we deal with the same species.

In this matter, our brain has dramatically changed its ontogenetic trajectory with respect to other primates to reach its large size. As concluded by Robson & Wood (2008), brain size represents a life history related variable, but not a life history trait itself. It is evident that brain metabolism and energetics could play an important role in the general physiology of organisms. Nevertheless, and given that chimpanzees and modern humans (and probably other hominins) reach similar brains size percentages in a short age

interval, it is difficult to infer a prolonged childhood in hominins using the size of the brain. Similarly, the great variability of the M1 gingival emergence, which is highly correlated with brain weight and has been used as a confident marker of a prolonged childhood, implies to be cautious when drawing conclusions on the prolongation of life stages in earlier hominins. In contrast, new research on brain maturation in present human populations and other primates are opening new perspectives for understanding the brain's role in the strategy of the life history pattern in hominins. A delayed brain maturation could be related to a retarded complete dentition in modern humans and possibly with the appearance of a prolonged adolescence. This delay in the dentition seems to be present in Early Pleistocene hominins, who could also have developed a post-juvenile period previous to the adult stage. However, it is not possible to make inferences about the brain maturation in these hominins.

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