RETHINKING PHYLOGENY AND ONTOGENY IN HOMININ BRAIN EVOLUTION

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Abstract

Theories of hominin and human cognitive evolution have traditionally focused on the phylogeny of the human brain, and on comparisons of human and primate brains in relation to social or ecological variables. Far less attention has been paid to ontogenetic processes, despite the recognition that experience has a profound influence on adult cognition. In this paper we discuss the interplay between phylogeny and ontogeny by examining relationships between human brain size, developmental scheduling and cognition.

The correlates of large brains include not only altered subsistence and life-history strategies to meet associated energetic costs, but also on macro- and micro-scale structural adaptations required to meet increased processing costs which mean that larger brains are of necessity more highly interconnected brains, with higher degrees of folding o the neocortex (gyrification) and higher ratios of myelinated connections between neurons (white matter) to neurons themselves (grey matter). Here we argue that the combination of these evolutionary trends underpins the complexity of human behaviour, as the neural circuits involved in cognitive mechanisms such as the mirror neuron system (the system governing motor emulation and imitation) and theory of mind (fundamental in social cognition) mature only slowly, and require considerable socially-scaffolded experience to develop to their full potential. These abilities are likely to be fundamental in characteristically human behaviours such as the cultural transmission of complex forms of tool manufacture and use attested to in the archaeological record, and their elaborated modern human forms, we argue, are possible only in the context of the evolution of relatively slower trajectories of brain growth and hence longer periods during which the growing brain can be influenced by experience among modern humans relative to other primates.

Here we review some of the differences in ontogenetic brain development between humans and other primates, and compare the rates and trajectories of neural development between ourselves and our closest living relatives the chimpanzees to

suggest that the human pattern of expanded periods of growth coupled with slower trajectories of neural development is likely to have been of huge significance during hominin evolution. In addition, we discuss fossil and archaeological proxies which might allow the reconstruction of evolutionary patterns of development, suggesting that it is only post-*Homo erectus* and specifically among *Homo heidelbergensis* and *Homo neanderthalensis* populations that developmental patterns approximate those of modern humans, arguing for a similar – but not identical – role for socially-scaffolded learning of complex technical skills as among modern groups in these species.

Introduction

Theories of hominin and human cognitive evolution have traditionally focused on the phylogeny of the human brain in relation to socio-ecological variables. A prominent example of such research is the Social Brain Hypothesis, the central tenet of which suggests that the size of the neocortex places constraints on social cognition and hence the size of the social group (Dunbar 1992, 9). Far less attention has been paid to ontogenetic, developmental processes such as the effects of infant socialization within these larger and/or more complex social groups, despite the fact that many lines of evidence now suggest that length and intensity of development and socialization have a profound influence on adult cognition and particularly on social performance.

By arguing for a renewed focus on ontogeny we are not suggesting that phylogeny is not important. Experiments in raising chimpanzee infants in human households did not produce simply unusually hairy humans (Hayes 1952) – our genetic heritage is of course fundamental to the structure and function of our brains, and to our development more generally. Indeed, our argument below will be based on the premise that it is the phylogenetic evolution of crucial life history parameters that makes the role of ontogeny, development and environmental so important. Adult cognition and indeed brain configuration is the result of the *interplay* between phylogeny and genetics on the one hand and ontogeny and an individual's interactions with the physical and the social environment on the other. Even fully 'modern' *Homo sapiens* do not automatically become fully-functioning members of their societies, any more than other animals denied environmental input at critical periods of development acquire many of their own species-typical traits – even those often considered genetically 'hardwired' such as

birdsong require experience and exposure to environmental stimuli to develop (e.g. Brainard and Doupe 2002). One of the aims of this paper is thus to argue that both phylogeny and ontogeny must be considered in any account of hominin evolution.

We will argue here that it is in fact the strong relationships between brain size and life history (Robson and Wood 2008; Barrickman *et al.* 2007; Smith and Tompkins 1995; Harvey *et al.* 1986) that are key to investigating hominin brains. We will examine the general ontogenetic trajectory of brain growth in humans relative to that in chimpanzees, and the relevance of these trajectories for social cognition, focusing particularly on two specific aspects of brain structure – gyrification and the ratios of grey and white matter in the brain – that are of particular importance during brain development. In the final section of the paper we consider the implications of a difference in trajectories between humans and chimpanzees for an increasing (phylo)genetic role for ontogenetic developmental processes of socialization during hominin evolution.

Ontogeny and phylogeny of human life history and growth

Modern humans have a larger than expected adult brain for our body size, relative to the ratio in other primates (Isler *et al.* 2008). In theory, encephalization could be achieved by either extending the period or increasing the rate of brain growth (or through some combination of the two; Robson and Wood 2008, 401). Either of these paths to encephalization will necessarily have significant implications for wider life history strategies.

Human life histories have much in common with those of the great apes. All great apes have relatively slow life histories, with long lifespans and slow growth (Charnov and Berrigan 1993). Slower life histories also correlate with larger adult body size (as energy can be invested in growth over a longer period), as well as with a constellation of other traits including larger babies (because larger mothers can invest in larger offspring), longer gestations and later age at first reproduction (Zollikofer and Ponce de Léon 2010; Robson and Wood 2008). Most elements of the human life history 'package' – with the interesting exceptions of age at first weaning and interbirth interval – see discussion in Robson & Wood (2008) are thus predictable from general primate trends, but are at the extreme end of the spectrum (Robson and Wood 2008). Our large body

and brain size mean that we develop extremely slowly, with an extended period of juvenile dependence, late puberty and age at first reproduction, and years (even decades) of prolonged and intensive parental effort.

Pre-reproductive phases of human life are therefore absolutely and relatively longer than observed among other large-bodied apes with similar gestation lengths (Crews and Gerber 2003) and may even include evolutionarily novel stages of development such as adolescence (e.g. del Giudice *et al.* 2009; Locke and Bogin 2006; Bogin 1999; Schultz 1969). This unique human combination of life history traits has most frequently been explained in terms of selection for extended periods of development as an adaptation for the acquisition of complex ecological and/or technological foraging skills (e.g. del Giudice *et al.* 2009; MacDonald 2007), social skills (Joffe 1997), or indeed both (Walker *et al.* 2006).

A related possibility is that the human life history strategy may have been adaptive because it mitigates ecological risk or reduces mortality. Under this hypothesis juvenile growth rates are slow because energetic resources are directed towards brains and immune systems to reduce the risk of starvation, and because slower rates of growth free up resources that can be used to feed younger siblings (Crews and Gerber 2003). The resulting reduction in adult mortality among humans compared to other great apes (Robson and Wood 2008) may have reduced constraints on prolongation of growth, development and longer life spans in general, perhaps aided by cultural factors such as 'material culture, language, and socio-culturally elaborated life ways, including long-term care of family members and late-life reproduction by men' (Crews and Gerber 2003).

Although in this argument

> '… juvenility did not evolve primarily for skill-learning, … it would nonetheless *permit* extensive learning … once extended skills/social learning became possible thanks to a long juvenility, a self-reinforcing cycle could have ensued, in which the advantages of learning generated an evolutionary pressure to increase juvenility even further and promote the growth of even bigger brains' (del Giudice *et al.* 2009, 9)

As well as their derived life history strategies modern humans are characterized by a distinctive pattern of growth. While human gestations are not significantly longer

than those of other anthropoids (Crews and Gerber 2003), they do demonstrate several differences in the allocation of energy to their foetuses, particularly in the last trimester when human foetal neurological development is fast-tracked at the expense of other tissues (Crews and Gerber 2003), contributing greatly to humans' secondary altriciality. This prioritization of brain growth continues throughout the first few years of life in humans: while brain growth continues at its rapid early pace for some time after infancy, bodily growth rates decline until the pubertal growth spurt corrects the imbalance (Bogin 1999).

Regardless of this rapid peri- and post-natal brain growth, at birth human infants have achieved a smaller proportion of their brain growth than other great apes, and this difference is maintained throughout their growth., as demonstrated clearly in Figure 1. Humans therefore reach their adult brain size more slowly than other primates. However, the differences have been exaggerated; the allometric exponent of neonatal brain size relative to adult brain size is negative (i.e., larger-brained species typically have smaller relative neonatal brain sizes), and human neonates have more or less the size of brain expected for an anthropoid primate of our brain size (DeSilva and Lesnik 2008), at ~29.9% of adult size compared to ~40.1% for chimpanzees (see DeSilva and Lesnik 2006 for a comprehensive review). Human infants also reach adult brain size earlier than usually claimed – on average 90% of adult brain size is achieved by around 5 years, only 1 year later than in chimpanzees (Robson and Wood 2008). While human mothers give birth to unusually large infants, then, those infants' brains are only slightly smaller than we would expect based on primate trends.

In addition, the overall trajectories of relative brain growth for both humans and chimpanzees are remarkably similar (Figure 1), suggesting that if humans were simply born later we would not deviate markedly from general great apes' gestational strategies – at least, in terms of brain development. As discussed above, when dental and somatic or bodily growth are also taken into account, human ontogenetic patterns deviate much more markedly from those of chimpanzees, being significantly slower; Zollikofer and Ponce de Léon 2010, 443. Nevertheless, in terms of the degree of neural development of our offspring relative to those of other primates, humans *do* appear to have an anomalously short gestation period. One potential selective pressure for this probably relates to the constraints imposed by the size of the female pelvic canal, itself reduced relative to that of other primates by the bipedal posture of humans (Franciscus 2009).

However, pelvic capacity is unlikely to be the only cause of these difference between humans' and other primates' brain development. Postnatal development is not linear, and while comparisons of neonatal and adult brain size are informative, consideration of patterns of growth at a finer scale reveal more differences between humans and other great apes that may have significant implications for adult cognition and behaviour.

While among precocial primates high gestational brain growth rates slow rapidly after birth relative to overall bodily growth, among (secondarily) altricial primates these rapid gestational rates of brain growth continue (Martin 1990). In humans these rapid growth trajectories continue for a full year after birth, compensating somewhat for our highly altricial offspring's small neonatal brain size. By a year after birth, human infants are pursuing a brain to body growth trajectory much like that of other primates – clearly demonstrated in Figure 2, in which we have 'shifted' a chimpanzee trajectory to be as altricial (i.e. small-brained) at birth as a human neonate.

Thus while trajectories of growth relative to adult brain size are not radically different among humans compared to other primates, growth *rates* do display some interesting differences. Figure 2 compares the growth rates of chimpanzees and humans over the first five years of life, and demonstrates that while rates of brain growth in chimpanzees peak no more than two months after birth, the human peak again occurs a full year later, at approximately 14 months. Note also that the peak growth rate in humans is somewhat lower than that in chimpanzees – meaning that despite our extreme altriciality, our brains never grow as fast postnatally (relative to their size) as do those of chimpanzees. Finally, it is also clear from this graph that the relative amount of brain growth occurring postnatally is substantially greater in humans than it is in chimpanzees (i.e. the area beneath the postnatal section of human curve is substantially greater).

Socialization, mirroring and Theory of Mind

Explanations of the specific advantages of extensive relative post-natal brain growth focus on the adaptiveness of a 'critical period' in which the brain can be 'tuned' to its environment during a relatively plastic growth phase. Some degree of neural plasticity continues well into adulthood (e.g. Merzenich 1987; Greenfield 1997, 115-118). However, the brain is particularly plastic early in life as the synapses develop between neurons

('synaptogensis'; Fig 3). Many of these developing synapses will be lost as the brain matures, due to competition for limited synaptic space and neural apoptosis ('programmed cell death'; Fig 3). Thus the synaptic capacity of immature neurons is almost 50% greater than that of adult cells (Lenroot and Giedd 2006, 720). In the prefrontal cortex, peak synaptic density occurs at 3-4 years of age and declines as brains mature, particularly after puberty (Höistad *et al.* 2009, 6; Bear *et al.* 2007, 709), as those synapses that are reinforced by frequent use out-compete those that are under-utilized, and grow stronger to enable more efficient transmission of information between neurons that are frequently associated in particular recurrent tasks (Deacon 1997). 'Slower' trajectories of brain growth thus allow for a much longer period of 'experience-expectant information storage' (Greenough *et al.* 1987) over which these processes of synaptic proliferation, competition and pruning and neural apoptosis can occur (Figure 3), and during which they can be influenced by environmental stimuli (Bear *et al.* 2007; Westermann *et al.* 2006; see also Grove and Coward 2008 for further discussion). This long period of extreme plasticity in human children coincides with the development of 'higher-order' neurobehavioural cognitive functions, including highly developed motor and social skills (Courchesne *et al.* 2003: 343).

The mirror neuron system

Mirror neurons, which are activated by both performance and observation of specific, goal-directed actions (Rizzolatti and Craighero 2004), were first identified in macaques and only later in humans. Until very recently, most work has thus assumed an evolutionarily primitive heritage among primates for the basic mechanisms involved in motor emulation that were elaborated later in the hominin and human lines. However, more recent work has suggested that the information necessary to match observed with executed actions may not be (completely) genetically specified, but that sensorimotor learning during development may also have a vital role to play. Experimental work has demonstrated that the functioning of the mirror neuron system (MNS) in both monkeys and humans is strongly affected by training and experience (Catmur *et al.* 2008; Iriki and Sakura 2008; Catmur *et al.* 2007; Keysers and Gazzola 2006; Ferrari *et al.* 2005), suggesting that the 'mirroring' properties of the system are not completely innate. Instead, many are acquired through simple Hebbeian or associative learning processes, in which the temporal correlation of observation and motor performance activates both neural circuitries simultaneously, entraining the circuitries associated with both

observation of others' actions (in multiple sensory modes) and kinaesthetic and sensory feedback from one's own actions and links them into a shared 'mirroring system', The Hebbeian maxim is thus, 'what fires together wires together' (Catmur *et al.* 2007; Keysers and Perrett 2004).

Neurological imaging and kinematic studies have demonstrated the vital role of sensorimotor functions and bodily 'know-how' in tool manufacture and use (Stout *et al.* 2008; Bril and Roux 2005; papers in Roux and Bril 2005) alongside - if not primary to - the 'higher-level' prefrontal and executive functions presumably involved in the broader contexts of action from the sourcing of raw materials to schemata of use. Among all tool-using primates, tool use is socially acquired and therefore likely to be reliant on MNS-mediated motor imitation – suggesting that it is the evolution and/or development of the MNS that underpins the appearance and elaboration of stone tools in the archaeological record. While the monkey MNS has now been demonstrated to respond to actions performed with tools (previously thought to be a human specialism), this has been demonstrated only after a long period of experimentation and familiarization of the monkeys to the tools and their use (Ferrari *et al.* 2005, 213, 221). In contrast, recent studies have suggested that in humans mere observation, or even simply *thinking about* motor actions may be almost equivalent to actual motor practice in improving motor learning (Heyes 2001, 256). While trained individuals do demonstrate stronger activations in response to others' actions (for example, trained pianists report finding it difficult to keep their fingers still while listening to piano music), even naïve individuals show some degree of neural activation when they observe others' actions (Keysers and Gazzola 2006, 389). This probably reflects the flexibility inherent in the varying selectivity of different neurons in the MNS, with some responding only to very specific motor actions and others more broadly, so that even novel actions can be extrapolated from the wide variety of motor skills that *are* within the observer's motor vocabulary (Keysers and Gazzola 2006, 389).

It is also notable that in monkeys, an MNS response to tool-actions was not sufficient for them to actually imitate the behaviour – given the opportunity to use a stick used in an experiment to access food left out of reach, the monkeys never attempted to do so (though at least one did pick up the stick and bite it; Ferrari *et al.* 2005). In fact, primates generally, while good *emulators* (being able to reproduce the physical results of actions in often very creative ways), are usually considered rather poor at *imitation*. In Horner and Whiten's 'puzzle box' experiments (2005), for

example, juvenile chimpanzees shown how to access food inside the box performed only those actions relevant to retrieve the prize, while human children performed even the unnecessary actions they had observed. However, results of similar experiments designed to test apes' imitation skills have been mixed, and Heyes' review of the evidence suggest that chimpanzees 'can imitate to the extent that they have had prior experience of interacting with humans and/or explicit training to imitate' (Heyes 2001, 253; see also Iacoboni 2005). Animals deliberately exposed to particular forms of stimuli not frequently encountered in the wild routinely develop skills and behaviours not practiced by their wild conspecifics – hence the mismatch in a wide range of cognitive skills demonstrated by human-enculturated and wild chimpanzees (e.g. Ferrari *et al.* 2005; Heyes 2001, 253), suggesting that development of both the MNS and imitation are heavily scaffolded by experience .

A key observation here is that motor 'mirroring' among humans is also very closely linked to social skills. Humans tend to (non-consciously) imitate one another's facial expressions, gestures and mannerisms during social interactions (Frith 2008), with the degree of mirroring related to high scores on paper tests for empathy. Such mirroring inclines the 'imitated' party to perceive the interaction (and his/her interlocutor) positively (Heyes 2001, 256). Significantly, many of the motor actions involved in such social mirroring involve 'perceptually opaque' movements. For 'transparent' motor behaviours such as hand movements, others' and one's own actions can be perceived simultaneously and the neural pathways are thus amenable to simple associative learning processes. However, the movements of the face and trunk that are so crucial to social interaction are typically only visible using cultural artefacts such as mirrors, or through interaction with others (Catmur *et al.* 2007; Heyes 2001). Co-activation and entrainment of the motor and sensory neural circuits involved in these actions requires that we, 'watch others as they do what we are doing – whether they are deliberately imitating our movements, as adults imitate infants, or simply reacting in the same way to ongoing events, like fellow spectators at a sports match' (Catmur *et al.* 2007, 1529; see also Keysers and Gazzola 2006, 396; Heyes 2001).

The key to how the human MNS functions to imitate skilled behaviours such as tool manufacture and use is therefore to be found not only in phylogeny but also in ontogeny, as social skills and behaviours are critical to imitation and the acquisition of skilled motor actions. Chimpanzee tool use is of course highly skilled and socially acquired, particularly in the context of the mother-infant bond where infants have

strong intrinsic motivation to copy behaviour and mothers to facilitate such copying (e.g. Matsuzawa 2007). However, this facilitation stops short of formal teaching (Tomasello 1999), and it has been argued that chimpanzee learning is based on a dyadic subject-object framework (mother-infant; mother-object; infant-object; object-object) focused on the emulation of actions on objects, rather than on socially-referenced triadic relationships among mothers-and-infants-and-objects (Matsuzawa 2007, 10; see also Sherwood *et al.* 2008, 435). In human children, such triadic relations commence from around 9 months of age (Sherwood *et al.* 2008, 435), and are strongly associated with other social skills, notably the capacities for joint attention and intentionality.

Individuals of many species may act together, either because their actions mutually affect one another and become coordinated or because of 'simultaneous affordances' in the environment that stimulate similar behaviours, for example a fresh carcass or indeed a buffet table (Knoblich and Sebanz 2008). However, higher levels of joint action may occur when individuals are able not only to perceive behavioural cues such as direction of gaze or bodily orientation but also to interpret them in the light of their own motor repertoire (via the MNS) and to attend to the same object(s) *together* - for example a parent pointing out things of interest (Knoblich and Sebanz 2008). Gaze following in particular has been widely studied in a variety of animal species, but studies on chimpanzees have produced mixed results (Sherwood *et al.* 2008, 430, 434 for review) while both dogs (e.g. Hare and Tomasello 2005) and goats (Kaminski *et al.* 2005) are consistently capable of following humans' gazes. This would suggest that domestication/socialization to (human) social systems in which joint attention is common may again be a key stimulus for the development of these abilities.

Once individuals are able to determine what someone is attending to, they may also be able to compare their own perceptions with those of the other to determine whether they are shared (Knoblich and Sebanz 2008). This level of joint attention clearly requires complex social cognitive skills, notably Theory of Mind (see below), and is likely to be the essential prerequisite for teaching, allowing the instructor to determine whether the learner has all the necessary perceptual attention or whether attention-guiding gestures such as pointing may be necessary (Frith 2008). While, as noted above, chimpanzee mothers *facilitate* infants' learning by making all the necessary equipment available, they do not direct attention to parts of the task they get wrong or correct their mistakes (Matsuzawa 2007; Tomasello 1999), and chimpanzees do not appear to use 'ostensive' gestures that would indicate the signal to follow will be a

deliberate communication about something of relevance to the receiver. In contrast, human infants are very sensitive to these behavioural cues; for example, eye contact prior to demonstration of a novel action or the naming of objects dramatically improves a child's imitation of that action or recall of the name (Frith 2008).

Among humans, individuals are also usually able to move beyond this stage of behavioural cueing to model the *intentions* behind actions and to engage in complementary action to aid (or to hinder) others' actions – *joint intentionality*. In order to achieve this it is necessary to represent both their own and others' contributions to the final goal (Knoblich and Sebanz 2008, 2025). Among humans, of course, there is a questionmark over the extent to which the evolution of human language scaffolds the development of higher stages of joint attention during childhood, or indeed the evolution of the underlying cognitive mechanisms among our hominin ancestors. However, many of these behaviours have precursors in other primates and thus were probably inherited from a common ancestor, and language learning itself is hugely reliant on social interaction and cognition in general, and ToM (see below), joint attention and perhaps also the MNS more specifically, suggesting that these forms of fundamentally social cognition are primary to language, not results of it.

In short, far from being innate, genetically specified mechanisms for acquiring skilled behaviour, the MNS and skilled motor imitation more generally are hugely influenced by experience acquired during development, which is provided as much by the social as the physical environment. The acquisition of these skills, utilising genetically inherited basal capacities, is thus strongly associated with - and probably scaffolded by - fundamental mechanisms of social cognition such as Theory of Mind (ToM).

Theory of Mind

Theory of Mind (ToM) is perhaps most usefully defined as the ability to understand that not only do others *think* in much the same way that you do, but also that *what* they think may differ. This appreciation that others have a different perspective from yourself, and to model that alternative perspective, underpins human social interaction, and sets us apart from most other primates in kind, and from all other primates in degree (see e.g. review in Emery and Clayton 2009). In modern human infants ToM emerges fully in infants by 4-5 years of age (Emery and Clayton 2009; Grove and Coward 2008 and references therein; Brüne and Brüne-Cohrs 2006, 440), i.e. during

the period of extreme plasticity occurring during the key phase of ontogenetic brain growth discussed previously.

However, in some individuals ToM does not develop in the usual manner; autistic spectrum disorders (ASD), whose severe forms are sometimes known as Asperger's Syndrome, are characterized by deficits in social cognition and interaction and the avoidance of novel situations and behaviours of any kind. Although the specific nature of both the deficits and the neurological mechanism(s) involved in ASD remain the subject of considerable debate, one significant line of enquiry suggests that the condition can be defined, at least in part, by a lack of ToM abilities (Baron-Cohen *et al.* 1985) while many other cognitive capacities and 'non-social' forms of intelligence are preserved (Brüne and Brüne-Cohrs 2006, 446).

In addition, individuals with ASD often demonstrate difficulties with imitation that seem to represent a failure of the ToM capacities that would normally 'scaffold' the development of the MNS

> '…resulting from early inattention to social stimuli (including adults imitating the autistic infant), and deficits in joint attention reducing the frequency of synchronous movement in response to a common stimulus' (Heyes 2001, 259,

such that Hebbeian processes responsible for the necessary neural connections do not occur, or occur to a lesser extent, in these individuals.

A growing number of researchers have also implicated abnormal neurological developmental processes in ASD. Current evidence suggests that the autistic brain grows substantially faster than that of normally developing individuals (Courchesne *et al.* 2003, 2004, 2007; Redcay and Courchesne 2005), reaching adult weight considerably earlier as a result. Yet the adult brain size of autistic individuals does not differ significantly from that of neurotypical individuals, implying that it is the *pattern* of growth that is the crucial factor.

In figure 4 we plot rates of brain growth of both neurotypical children and those with ASD for comparison; the important point to note here is that the ASD phenotype involves a period of neural 'overgrowth' relative to neurotypical controls (Redcay and Courchesne 2005). As a result, children with ASD have substantially bigger brains than neurotypical controls between the ages of approximately 2 and 5, 'at the beginning of an important period of developmental neuroplasticity and learning' (Courchesne *et al.*

2003:343). Courchesne and colleagues argue that this more rapid growth of the brain in individuals with ASD shortens the critical period during which experience of the physical and social environments may influence synaptic proliferation and pruning.

One argument is that this reduction of the timeframe in which the selective effects of experience may 'shape' patterns of neural development means that ASD synaptic proliferation is explosive and random, resulting in early fixation of potentially anomalous connections and the subsequent inability of the apoptosis mechanism to achieve targeted pruning of maladaptive synaptic connections (Casanova *et al.* 2008; see below).

Further support for this position may be found in recent arguments that ASD individuals often experience sensory hypersensitivity. Individuals with ASD have much better eyesight (≈2.79x better than average) and more sensitive olfactory, haptic and auditory systems than neurotypical controls; furthermore, the degree of hypersensitivity correlates with scores on measures of ASD severity (Baron-Cohen *et al.* 2009). As Baron-Cohen *et al.* point out, such hypersensitivity could be the result of processing differences at multiple levels: sensory receptors could be denser or more sensitive, neural processing could be faster and/or top-down inhibition systems could be affected. This last might mean that the process of forming higher-level 'holistic concepts and meaningful labels' (Snyder 2009) that usually help structure perception of sensory information and inhibit the costly processing of lower-level details does not occur (or occurs to a lesser extent) among individuals with ASD. These individuals would then routinely experience sensory 'overload', with reduced top-down processing constraints resulting in a low signal-to-noise ratio.

Neuroimaging of the brains of individuals with ASD does indeed appear to show reduced coordination of activity between association areas and those mediating perceptual and emotional processing. Brains are more connected between local regions, and differences in gyrification and in grey and white matter distribution (see below) suggest an increase in short-range relative to long-range connections (Casanova *et al.* 2008). One argument is that among children with ASD, short-range neural connections proliferate at the expense of long-range circuits and systems relating to top-down control and coordination during early development.Such a pattern of development might result from a failure of synaptic pruning mechanisms following early over-production and/or a disturbance in white matter production, such as in processes of

myelination that would make long-range connections more efficient and competitive. (Casanova *et al.* 2008).

It therefore seems likely that the development and refinement of higher-order and particularly social cognition is intimately related to the evolution of slower trajectories of neural developmental trajectory in normally developing humans, relative to that in other primates and in our hominin ancestors, and that a focus on the interplay between ontogenetic and phylogenetic factors is therefore fundamental to understanding hominin brain evolution. Most studies of hominin brain evolution have focused primarily on gross brain size and/or the relative sizes of different brain and particularly neocortical structures. However, larger brains are associated not only with the energetic costs offset by changing life-history strategies, but also with significaitn processing costs, as increased brain (and body) size is associated with increased transmission times for nerve impulses. In addition, as the number of neurons increases, the number of connections between them increases exponentially, and thus, given already extremely high levels of connectivity between cortical neurons, larger brains are potentially highly costly and inefficient. The ways in which these costs are offset, and the ways in which these interrelate with solutions to large brains' energetic costs, have significant repercussions for large-brained species. Two of these adaptations to the increased processing costs of large brains – gyrification and the ratio of white to grey matter in the brain - are examined briefly in the following sections.

Gyrification

Perhaps the most obvious feature of the brain's gross anatomy is the wrinkled and folded surface of the neocortex. Viewed in section, it is clear that some parts of the neocortex bulge outward (gyri) while some are folded inward (sulci). The ratio of total cortical surface (i.e. *including* the surface area of cortex hidden in cerebral sulci) to exposed cortical surface (i.e. *excluding* the surface area of cortex within sulci; Rilling 2006) yields a 'gyrification index' (GI) which varies both phylogenetically and ontogenetically (Figures 5 and 6).

The traditional explanation for increased levels of gyrification in large-brained primates has centred on the need to fit a larger brain (or, more specifically, neocortex) into a semi-spherical skull. This explains why gyrification of the expanded surface of the

neocortex is more pronounced in larger-brained species (White *et al.* 2009; Rilling 2006; Zilles *et al.* 1988) - figure 5 demonstrates the existence of distinct evolutionary relationships between GI and brain weight in prosimians and anthropoids: the latter have substantially higher GIs than the former across much of the range of empirically documented brain weights, and GI increases with brain weight at a substantially higher rate in anthropoid primates (though it should be noted that the abscissa in Figure 5 represents a logarithmic scale, indicating that, although GI is *absolutely* higher in larger brained animals, GI as a ratio to brain weight is *relatively* higher in smaller-brained primates).

Among humans the degree of gyrification in some areas (notably temporal/parietal association regions and prefrontal cortex) is even greater than predicted from our larger brain size (White *et al.* 2009; Sherwood *et al.* 2008; see also Rilling 2006). This observation is better explained by newer theories of gyrification which argue that sulci and gyri develop as strongly connected regions are drawn together by the many axons linking them, reducing transit time for action potentials and enhancing the efficiency of specific circuits (White *et al.* 2009; Lenroot and Giedd 2006, 720). Such a mechanism would explain the general link between brain size and GI (as transit time and efficiency become increasingly significant costs as brains become absolutely larger), and the human deviation from general mammalian trends in this regard is of particular interest here, suggesting adaptations for processing efficiency over and above those required by encephalization *per se*.

The argument that gyrification relates to the development and elaboration of neural circuits also explains the ontogenetic changes in gyrification. Early stages of gyrification occur in the foetus only 10-15 weeks after conception (White *et al.* 2009, see Figure 5), but it is during the third trimester (when maternal resources are increasing directed towards foetal brain growth; see above) that GI increases dramatically and the brain begins to develop its adult morphology (White *et al.* 2009, see below; Lenroot and Giedd 2006). Although it was traditionally thought that gyrification plateaus after birth (i.e. matches the threefold postnatal volumetric growth of the brain), 3D techniques of assessing GI are now beginning to document changes in GI occurring throughout childhood and adolescence (White *et al.* 2009). In particular, gyrification appears to increase significantly in later-maturing regions such as prefrontal cortex between 6 and 16 years and declines thereafter, perhaps especially at adolescence (White *et al.* 2009).

Although individuals' patterns of gyrification do appear to be strongly heritable, there is considerable individual variation and monozygotic twins also show considerable differences. Deeper and earlier-developing sulci such as the Sylvian fissure (the two very deep sulci lateral sulci that are one of the most prominent landmarks of the brain) are more similar between twins (and thus likely to be more highly constrained genetically) than superficial sulci which develop postnatally and which may thus be more plastic in response to stimuli from the physical and social environments (Sherwood *et al.* 2006; White *et al.* 2009).

In short, then, the phylogenetic and ontogentic development of gyrification indices in the human brain suggest adaptations for greater connectivity to offset the potential inefficiency of larger brains – and, indeed, the evolution of gyrification indices greater even than this requirement in some parts of the human brain. Ontogenetically, the gradual development of gyrification among late-maturing parts of the brain may suggest a role for developmental experience in literally shaping the adult brain.

Grey and White Matter Ratios

The relative balance of 'grey' and 'white' matter in the brain provides an alternative perspective on these processes. Grey matter (actually a blood-suffused rosy colour in the living brain) is comprised of neural cell bodies, while 'white' matter is mainly comprised of supporting glial tissue such as astrocytes (which play a role in regulating neuronal energy uptake), oligodendrocytes and myelin (the former synthesizes the latter, which sheathes axons to facilitate long-range propagation of action potentials; Barton 2006).

The ratio of neurons to glia has long been known to vary phylogenetically (see Figure 7), with larger-brained (and bodied) species having lower neuronal densities (Barton 2006; Sherwood *et al.* 2006). However, in larger-brained species those neurons are larger and have longer and thicker axons (improving conduction velocity) which are increasingly myelinated (sheathed in fatty myelin), helping to insulate them and speeding up synaptic transmission), thus conserving processing speed in the face of greater transmission distances (Barton 2006). As brains grow larger across speciesthe volume of white matter thus rises disproportionately (Sherwood *et al.* 2006), and the ratio of grey to white matter in human brains is as expected for a primate of our brain size (Smaers *et al.* 2010; Schoenemann *et al.* 2005), making them *relatively* more

connected than those of smaller nonhuman primates - perhaps especially in prefrontal areas (Höistad *et al.* 2009, 5).

However, ratios of white to grey matter also vary ontogenetically. Most of the neurons we will ever have are present by birth, and therefore volumes of grey matter do not change significantly post-natally. The rapid postnatal growth of the brain is instead due mainly to proliferation of synapses, maturation of the glial cells and myelination of axons (Höistad *et al.* 2009, 5), and white matter volume thus increases dramatically between birth and adolescence (see Figure 8), when considerable amounts of synaptic pruning occur. Myelination of cortical axons begins before birth. First to myelinate are the spinal cord and brainstem; the fibres linking the cerebellum to the cerebral cortex and which are necessary to the fine control of voluntary movement only begin to myelinate after birth, and do not mature until about 4 years of age (Höistad *et al.* 2009, 5; Grove and Coward 2008; Lenroot and Giedd 2006), while intra-cortical connections, particularly in prefrontal regions, continue to myelinate well into the third decade of life, and do not decline here until after 50 years (see Figure 3 and refs in Höistad *et al.* 2009, 401). In contrast, in humans grey matter growth declines after the age of 5, with volumes peaking at 10-12 years in frontal and parietal and 16-18 years in temporal regions (Höistad *et al.* 2009).

Both gyrification and white:grey matter ratios undergo significant changes during adolescence, when a variety of gross psychological and behavioural changes also occur and also when a number of psychiatric disorders such as schizophrenia first manifest. This may thus be another critical period for brain development, as growth patterns change and brains enter the later, less plastic stages of maturation. While ASD seems to relate to atypical early brain development trajectories (see above), schizophrenia has been argued to represent an exaggeration of 'normal' brain maturation mechanisms occurring during adolescence, such as reductions in grey matter volume, although myelin deficiencies and changes in white matter volume also often occur (Paus 2001; Paus *et al.* 2008, cited Höistad *et al.* 2009, 1, 6). While arguments continue to rage over the relative contributions of genetic inheritance and environment to conditions such as schizophrenia and ASD, genetic components do appear to be substantial (Picchioni and Murray 2007; Freitag 2006), suggesting high heritability of such developmental disturbances, which may be an unwelcome negative result of the extreme scaling of large brains. As discussed above, large brains – or, rather, enlarged neocortices – necessarily entail several functional correlates in order to maintain efficiency. They are increasingly

dominated by disproportionately large late-maturing neocortices, which are increasingly closely inter-connected, with greater ratios of white to grey matter and larger gyrification indices. All of these features of large brains require concomitantly slower maturational schedules and thus longer 'critical periods' of plasticity during which they are influenced by social and physical environmental stimuli. The downside would seem to be that the complexity and prolongation of the process of 'wiring' the brain renders larger brains more vulnerable to a variety of developmental abnormalities such as ASD or schizophrenia, as well as to degenerative conditions such as Alzheimer's or multiple sclerosis (Sherwood *et al.* 2006), suggesting that humans' large brains may be near the functional limits of encephalization (see e.g. Hofman 2001 for discussion)

Discussion: the Evolution of Ontogeny

The complex relationship between the phylogenetical evolution of the human brain and its ontogenetic development merits serious consideration of the extent to which the two processes may have interacted throughout hominin evolution (see e.g. Zollikofer & Ponce de Léon 2010 for discussion). Relatively small variations in developmental patterns can have large effects both overall brain size and the relative sizes of brain components. One hypothesis is that encephalization may have been achieved via relatively simple single-gene mutations affecting the number of cycles of symmetric division precursor cells for neurons undergo before each cell begins to increase exponentially (Rakic 2009, 726): the more precursor cells that can be formed, the larger the structure that results, and as brain size increases, late-maturing structures such as the neocortex grow disproportionately larger via the same mechanism (Finlay and Darlington 2005; Finlay *et al.* 2001). Many of the genes thought to have been under selection in recent human evolution are believed to be regulatory genes governing the timing of developmental processes, and indeed regulatory genes may be fundamental to evolution more generally (Vaquerizas *et al.* 2009, 260).

Large brains are associated with many costs, including reduced efficiency and high energetic demands. Nevertheless, encephalization has clearly been adaptive among primates generally, and the hominin lineage in particular, indicating that these costs are adequately balanced on an evolutionary timescale by benefits. One obvious possibility is that large brains are adaptive because of a net cognitive gain of some kind, although the

nature of the relationships between brain size, cognitive prowess and behavioural sophistication remains frustratingly unclear. Another possibility is that the wider constellation of adaptations surrounding large brains themselves are also adaptive.

As we have seen, comparison of human brains with those of other primates demonstrate clearly that the energetic and efficiency costs of larger brains have been met by evolutionary changes to the structure of the adult brain - for example through increasing gyrification and greater proportions of white to grey matter – as well as to broader life histories strategies such as longer, slower developmental schedules (Isler & van Schaik 2009). However, it is also highly possible that these 'side-effects' of larger brains were also adaptive in and of themselves, and contributed to a positive feedback loop during hominin evolution in which the ontogenetically selective effects of extended 'critical periods' of development, via which the structure of the brain itself can be at least partly fine-tuned to be optimal for the required functions, was also evolutionarily adaptive and therefore selected for in and of itself,

Larger brains are of necessity relatively more interconnected brains to maintain efficiency of signalling; however, the complexity of human (and indeed ape) behaviour and of the neural 'wiring' involved is such that our brains require extremely significant environmental input from both the physical and social environments if the individual is to function sufficiently well to survive and to negotiate a complex social world in order to reproduce. Thus, an increasing reliance on physical and especially social interaction to structure hominins' slower-growing brains is likely to have been adaptive not only as a means of off-setting the energetic and processing costs of larger brains, but also because it allowed the development of elaborated forms of hgher-order and social cognition possible *only* in the context of extended periods of growth and slower trajectories of neural development. Modern human patterns of brain growth and development trajectories may thus represent an extreme state of such a positive feedback loop, maximising the length of time during which environmental input can significantly influence the brain and allow the development of complex forms of cognition, to the extent that only small deviations are associated with conditions such as ASD and schizophrenia, which significantly impact on particularly social cognition, and reduce the likelihood of reproduction and thus evolutionary fitness (Avila *et al.* 2001; Walsh *et al.* 2008).

Clearly, such fine-grained neurological developmental processes as gyrification and white:grey matter ratios cannot be studied directly in fossil remains. Work is

needed to establish the extent to which they may be estimated from proxies such as gross brain sizes ascertained from endocranial volumes or from the, many elements of the broaderlife-histories of extinct hominins which can be accessed in the fossil record, and allow tentative estimates as to the nature and timing of possible inflection points in the evolution of human developmental scheduling.

In a previous paper (Grove and Coward 2008) the authors argued for *Homo erectus* as a possible break-point in hominin developmental scheduling. More recent work, including a comprehensive review by Robson and Wood (2008) and work by Zollikofer and Ponce de Léon (2010) have since provided further data, and allow a more detailed consideration of the evidence.

Certainly in terms of overall brain size *Homo erectus* would seem to be a highly plausible candidate. 'Archaic' hominins (the pre-erectines, in Robson & Wood's terminology; 2008) remained relatively small-brained (with the larger brains of the robust australopithecines apparently a specialized adaptation related to their derived dental and jaw morphology; DeSilva and Lesnik 2008). DeSilva and Lesnik calculated that the brains of australopithecine neonates would have been around 38.1% of adult size at birth and those of early *Homo* 35.2%, compared to values of ~40% for chimpanzees and only 29% for humans (see refs in Franciscus 2009), suggesting a general lack of selection for secondary altriciality for both the australopithecines and the earlier *Sahelanthropus* and *Ardipithecus* (refs in Zollikofer and Léon 2010, 447; Robson and Wood 2008, 412-415). The rejection of derived life-history scheduling for these early hominins is also supported by dental data documenting a more rapid trajectory of growth (Robson and Wood 2008, 411), although dental analyses of the robust australopithecines underline the mosaic nature of life history among different hominins by suggesting a unique 'package' of dental ontogenetic scheduling (Zollikofer and Ponce de Léon 2010, 447).

Only among *Homo erectus* specimens (*sensu lato*) do brain sizes increase to nearer modern than chimpanzee values (DeSilva and Lesnik 2008; Leigh 2006; Walker and Ruff 1993), and a number of studies have suggested that *Homo erectus* brain sizes were consistent with modern human brain-growth, with only ~33.1% of adult brain size achieved by birth (DeSilva and Lesnik 2008; Robson and Wood 2008). Other anthropological and archaeological developments associated with late *erectus* have also been used to suggest a significant change in lifeways including increased body size (Robson and Wood 2008), a greater focus on dietary meat and longer limbs suggesting

adaptations for more efficient bipedalism (O'Connell *et al.* 1999) as well as expansion into northern latitudes (see refs in Grove and Coward 2008, 396) that might relate to both dietary and social innovations in meeting the different energetic demands of human developmental schedules. The association of *erectus* with the handaxe in particular has been argued to suggest an increased role for cognitive mechanisms permitting the faithful imitation of skilled motor behaviours. Acquiring the skills of Oldowan core-and-flake technologies may require only a relatively straightforward extension of action repertoires and social skills (Knoblich and Sebanz 2008). However, more complex tool behaviours involving 'roughing-out' stages intended not to produce useful flakes but to prepare for later stages of manufacture may require higher levels of joint attention and intentionality to learn - the imitation, rather than emulation, of goal-directed rather than simply sequential motor sequences. Thus ToM is also likely to be significant here (Knoblich and Sebanz 2008) in order to translate between one's own and others' perceptions – from 'what *I* see' to 'what *s/he* sees' and vice versa) – even before considering the significance or otherwise of handaxe symmetry, symbolism and/or 'sexiness' (Hodgson 2009a, 2009b; Kohn and Mithen 1999 and comments thereafter; Wynn 1995; see also McNabb this volume for further discussion).

However, other research has suggested that *Homo erectus* may not have been quite so modern after all. In particular, interpretations based on two of the major fossil specimens for examining life history scheduling in *Homo erectus* - the Mojokerto and Nariokotome juveniles – remain controversial. A mismatch in age at death as calculated using dental and skeletal methods for the Nariokotome juvenile (Dean and Smith 2009; Walker and Leakey 1993) has been used to argue that the derived modern human pattern of delayed juvenile growth and catch-up adolescent growth-spurt had not yet become established (Smith and Tompkins 1995), and that the Nariokotome boy had already undergone an early growth spurt more similar to that known among chimpanzees (Zollikofer & Ponce de Léon 2010, 448). Several more recent analyses of the material have also argued for a primate-style growth trajectory (Dean and Smith 2009; DeSilva and Lesnik 2006; Leigh 2006).

Age estimates of the Mojokerto child vary much more widely, ranging from 0.1-1.5yrs to 4-6 years of age at death, and make it difficult to determine how much brain growth had occurred during gestation. If the Mojokerto child does fall at the younger end of this proposed age-range this would suggest a fast trajectory of growth more akin to that of modern non-human primates. However, if older a slower, more derived

'human' pattern is more likely (DeSilva and Lesnik 2006; Coqueugniot *et al.* 2004). Meanwhile, while the the subadult specimen from Dmanisi apparently developed faster than modern humans based on its degree of skeletal maturation, it nevertheless fell within the 95% range of modern human variation (Zollikofer & Ponce de Léon 2010, 446). Zollikofer and Ponce de Léon's recent review concluded that early brain growth in *Homo erectus* was likely to have been fast (i.e., more 'modern'), but that these rates were not sustained for long (i.e., more 'primitive'; 2010, 446).

In short, the *Homo erectus* material does not provide unambiguous evidence of a shift towards derived human life history and developmental scheduling. Perhaps this is not surprising given the wide geographical and temporal distribution and variability of specimens.

It is therefore worth expanding on the arguments put forward previously (Grove and Coward 2008) to consider the later pre-modern *Homo* species more thoroughly. Although *antecessor*, *heidelbergensis* and *neanderthalensis* share considerable similarities with modern *Homo sapiens* in both postcranial and cranial morphology, insofar as it is fair to judge from the archaeological record they appear to have demonstrated several differences in behaviour and perhaps also cognition. Body masses and brain sizes among these species are statistically indistinguishable from those of modern *Homo sapiens*, and DeSilva and Lesnik (2008) calculate that ~29.5% of brain growth would have been completed prenatally among Middle Pleistocene *Homo*, compared to a figure of 29.9% for modern humans. Zollikofer and Ponce de Léon (2010) suggest that while postnatal brain growth rates were higher among Neanderthals than modern humans, their larger adult brain sizes meant they took the same amount of time to develop as in modern humans.

However, a variety of other lines of evidence have also been used to investigate patterns of gestation and development in these mid- Pleistocene species. Studies on dental development, including crown and root formation and eruption times, have given mixed results (perhaps not surprising given the small sample sizes of many of the studies and the variability of modern human dental developmental schedules). Studies have variously argued that Neanderthals:

1. Developed on faster and more rapid trajectories than *Homo sapiens* (e.g. Smith *et al.* 2007);

2. Had shifted towards the derived slow growth rate characteristic of humans relative to the shorter, more rapid periods of growth of *Homo antecessor* and *Homo heidelbergensis* (e.g. Bermúdez de Castro *et al.* 1999);

3. Developed on a trajectory almost indistinguishable from modern humans (Machiarelli *et al.* 2006; Guatelli-Steinberg *et al.* 2005; Ramírez Rossi and Bermudez de Castro 2004; Dean *et al.* 2001), and

4. Shared with *all* pre-modern *Homo*, including *erectus* (s.l.) a similar pattern of dental development, in contrast to non-human primates and archaic hominins, with *Homo ergaster* representing the evolutionary link between the two (e.g. Bermúdez de Castro *et al.* 2003; see review in Robson and Wood 2008, 414).

The much better preserved fossil record of the Neanderthals (including many finds of juveniles and two neonates (Holloway *et al.* 2004) also allows consideration of other skeletal traits that might inform on life history strategies, notably the dimensions and shape of the pelvis. Trinkaus (1984) had suggested that Neanderthal pubic morphology was consistent with a longer period of gestation in this species, but this suggestion was strongly refuted by Rak and Arensburg (1987) and Rosenburg (1988). More recently, Weaver and Hublin (2009), based on the pelves of the Tabun female and Kebara male, concluded that Neanderthals retained a more primitive birth mechanism than modern humans, but that obstetric difficulty would have been about the same in both species.

In addition, the stage of skeletal growth attained by the adolescent Neanderthal skeleton Le Moustier 1 by 10.5-13yrs of age would locate it in the lower part of the modern human bodily growth trajectory - although its height was only slightly less than that of modern humans, suggesting that Neanderthal adolescents probably underwent a similar growth spurt to modern humans (see e.g. Zollikofer & Ponce de Léon 2010, 448 for refs).

This admittedly brief survey of work on life history evolution in later *Homo* indicates a number of variable adaptations with no simple dichotomy between 'fast'/primate and 'slow'/human strategies (DeSilva and Lesnik 2008; Robson and Wood 2008, 417; Crews and Gerber 2003, 13), but nevertheless a gradual development of the characteristic modern human condition of expanded juvenile period of development, , with *Homo erectus* (s.l.) perhaps pushing the boundaries of non-human primates strategies, and *Homo heidelbergensis* and *neanderthalensis* approaching, if

not quite matching, modern human developmental schedules (cf. Hodgson this volume).

The significance of this slower developmental scheduling lies in its association with extended periods of brain growth, and hence the greater degree of environmental influence the growing brain was subject to during the critical periods of synaptogenesis and synaptic competition.

Conclusions

Investigation into the evolution of modern human cognition has focused primarily on insights from phylogenetic comparison of gross brain size and structure of human brain and those of other primates. The role of ontogenetic and developmental factors has not been accorded the significance it deserves in studying the evolution of cognition, and particularly of technological and social behaviours.

The implications for archaeology are significant. Two of the most significant elements of modern human cognition, the Mirror Neuron System and Theory of Mind, are both strongly reliant on social and kinaesthetic experience scaffolded by social interaction for full realization in human infants, and their elaboration in modern humans may be related to our much longer periods of development and particularly dependent childhood relative to other primates. During this time brains continue to develop and mature in the context of social and physical environments which impact on the processes of synaptic competition and pruning and myelination, as documented by changing patterns of gyrification and ratios of grey to white matter. These 'slower' trajectories of growth (relative to those of other primates) are of course part and parcel of wider life-history strategies related to the re-structuring of energetic budgets across the whole lifespan necessitated by encephalization. At the same time, adaptations to the processing costs of larger brains - in particular, the phylogenetic patterns of gyrification and the ratio of white to grey matter – led to increasing interconnectivity of hominin and human neocortices, and these processes also had significant effects on cognition. It is the scale and complexity of those connections, and the structuring role of environmental input in their development - that both allows the elaborations of the MNS and ToM seen among modern humans, and their interaction, and that renders

humans more vulnerable to developmental and/or degenerative disruption of normal processing.

It is possible that this role of ontogenetic experience in shaping the brain was a fortuitous by-product of encephalization adaptive for other reasons. However, the potential adaptiveness of the neural and cognitive plasticity that results may also have been adaptive in and of itself, and an alternative possibility may be that large brains are a by-product of selection for increasing neural plasticity achieved through delayed maturation of the brain. The modern human brain and cognition is likely to be the result of a complex constellation of selective pressures and releases linking encephalization, long,slower life histories and delayed maturation of the brain, larger and more complex social groups and subsistence practices etc. (see e.g. Coward & Grove submitted figure 1), and there is no reason why selective pressures should have remained constant or equal throughout hominin evolution, but it does seem clear that ontogenetic processeses of neural development, and the structuring experience of and interaction with the social and physical world are likely to have been extremely significant throughout hominin evolution.

Early developments in the hominin line, notably the habitual use of stone tools in the extraction of animal protein (only appearing themselves at 2.6mya (Semaw *et al.* 1997) but attested to by cutmarks on bones from 3.3mya (McPherron *et al.* 2010)), would seem to represent significant behavioural changes from panin lifeways. Not only are these stone tools used in rather different ways, but they also seem to demonstrate enhanced levels of motor skill relative to those known among even enculturated and trained chimpanzees (Delagnes & Roche 2005) – possibly related to the adoption of bipedalism and the release of locomotive selective pressures on wrist and hand anatomy (Ambrose 2001, 1750; Hodgson this volume). However, these early tools do not seem to be accompanied by any obvious fossil indicators of changed life histories (Robson & Wood 2008), and appear to be explicable in terms of more skilled forms of motor emulation, rather than goal-level (socially-scaffolded) imitation, and it is not until the appearance of Mode 2 and subsequent technologies that it is clear that at least precursors of the cognitive skills involved in fine-grained imitation and social interaction had become established at a level distinguishing hominins from other primates. However, these early changes in lifeway may represent the earliest elaborations on a basic primate theme, establishing the selective environments which made the later elements of the modern human cognitive suite adaptive.

Acknowledgements

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Figures

Figure 1. The trajectories of human and chimpanzee brain growth compared. The three human trajectories are from autopsy samples; the lines shown are the best-fit lines calculated by Kretschmann *et al*. (1979) from the raw data in each case. The chimpanzee line was calculated by fitting the growth equation of Kretschmann and colleagues, Brain Weight $= \frac{P_1}{1+\exp(P_2+P_3 \cdot \text{Age})}$, to data published by Herndon *et al*. (1999).

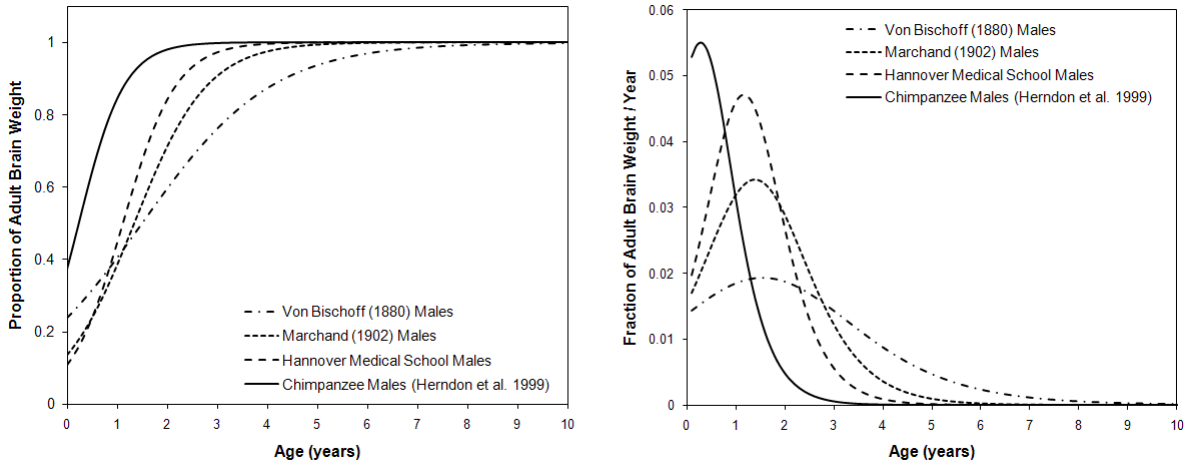


Figure 2. The similarity between the growth curves of chimpanzees and humans; the 'shifted chimpanzee' is born as altricial as a human, and follows a very similar trajectory. The curves show human data from the Hannover Medical School sample (Kretschmann et al. 1979) and chimpanzee data from the Herndon et al. (1999) database. Both curves show male growth trajectories.

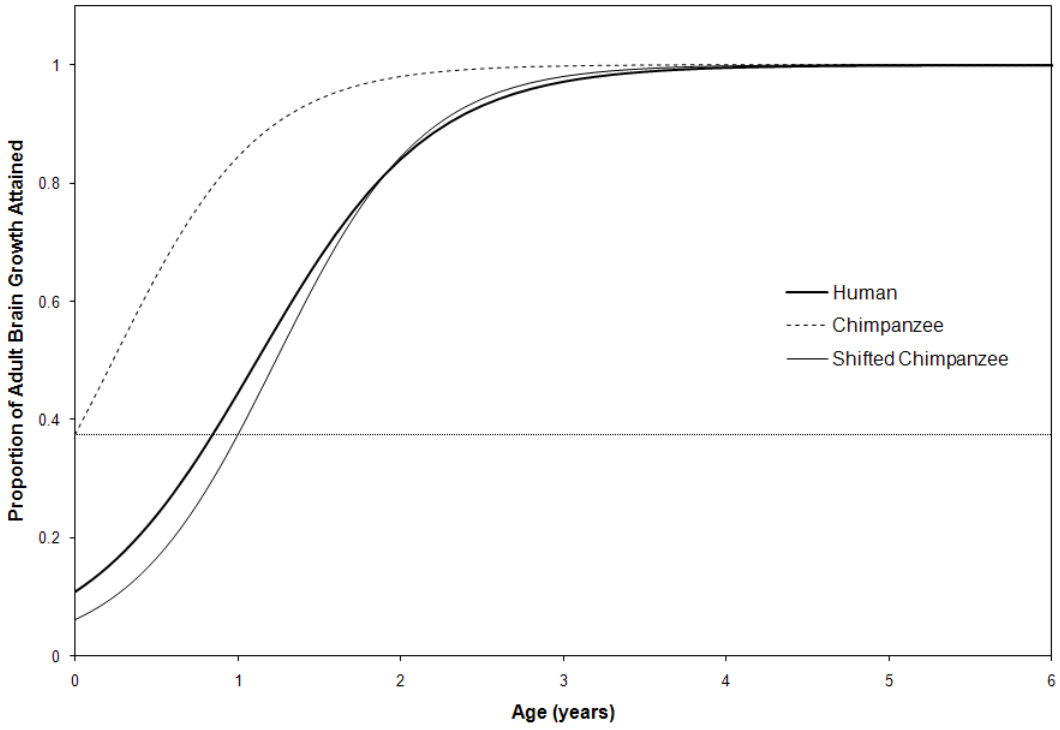


Figure 3. Sequence of events in brain maturation (redrawn from Lenroot and Giedd 2006 figure 1). Neurulation, the initial development of neurons, occurs first and is followed by multiple cycles of the production of new neurons (neurogenesis). New connections begin to be established between neurons (synaptogenesis), and the axons of different neurons 'compete' for space to synapse on the dendrites of recipient neurons and thereby establish a connection between those neurons (synaptic competition). Programmed cell death (apoptosis) prunes under-utilized neurons throughout these processes. Axons connecting neurons are ensheathed in fatty myelin to insulate and speed up action potentials travelling between those neurons, while further development and multiple branching (arborisation) of dendrites and axons and denrities continues throughout life.

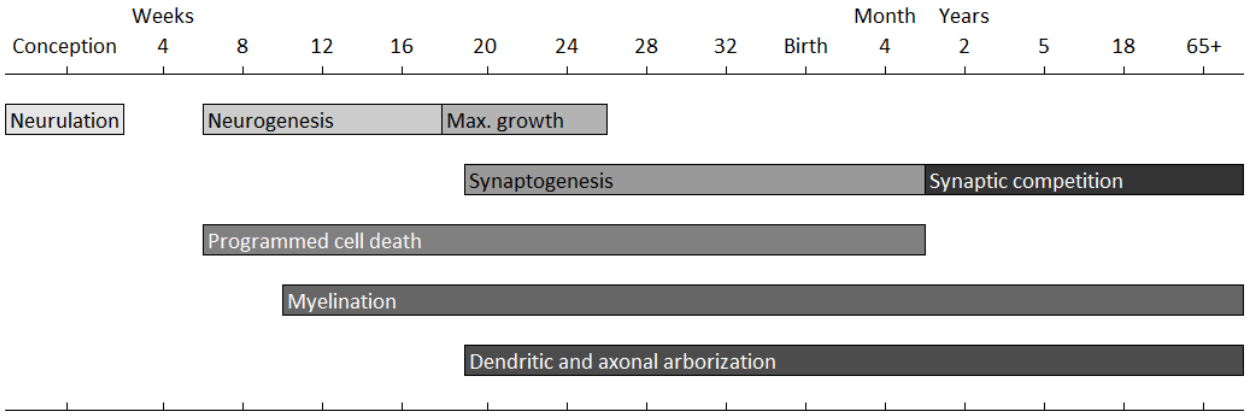


Figure 4. Chimpanzee, non-autistic, and autistic human growth rates over the first five years of life. Note all lines are scaled to proportion of adult brain weight; there is of course a vast difference between the sizes of chimpanzee and human brains, with ASD and non-ASD brains being virtually identical in size by adulthood. The human curve is calculated from Hannover Medical School data (Kretschmann et al. 1979), and the chimpanzee curve from the Herndon et al. (1999) database. The autistic overgrowth curve is calculated by multiplying the Hannover Medical School curve by age-specific values of the Redcay and Courchesne (2005) autistic overgrowth equation.

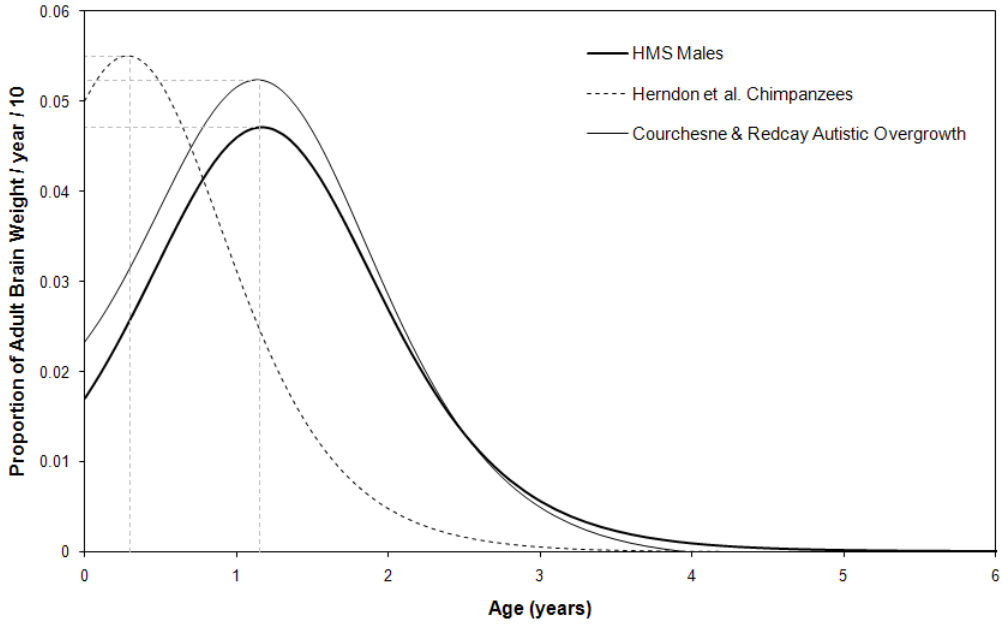


Figure 5. The phylogeny of the gyrification index in prosimians and anthropoids (data from Zilles *et al*. 1989).

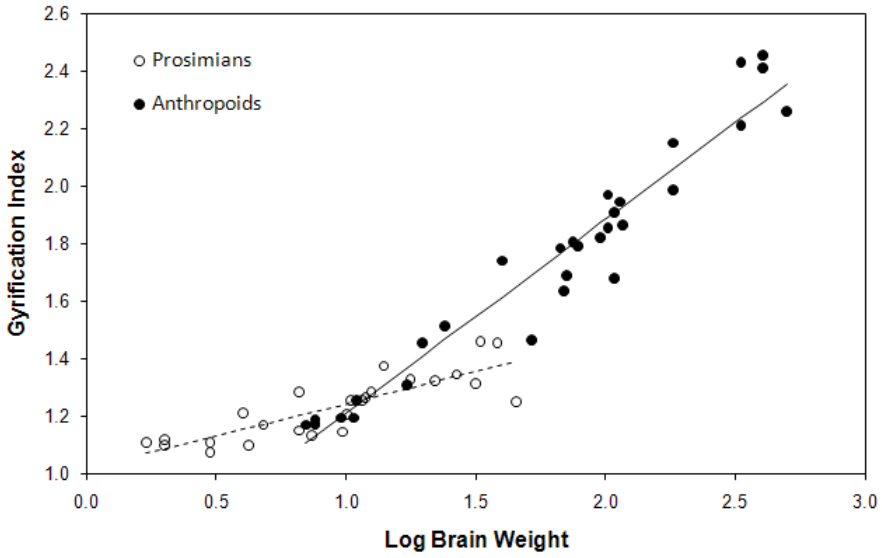


Figure 6. The ontogeny of the gyrification index in humans (data from Zilles et al. 1988; curve fit as per the Kretschmann *et al*. 1979 procedure - see caption to Figure 1).

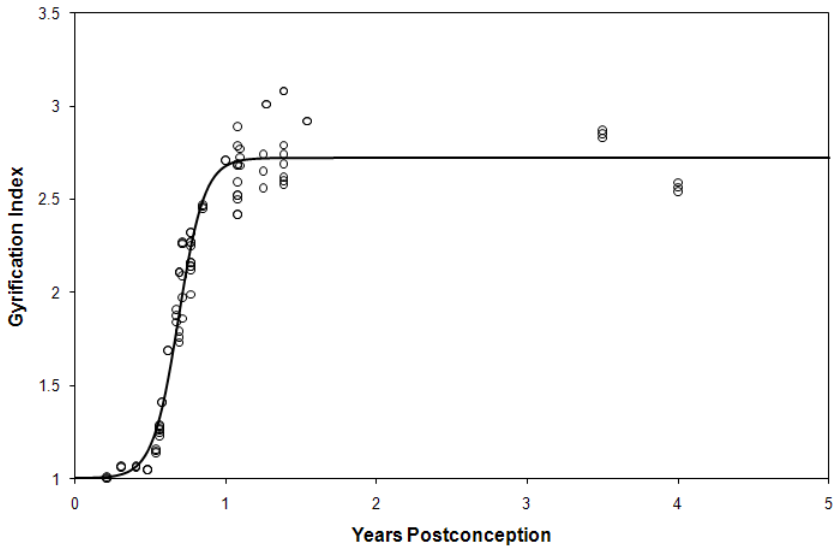


Figure 7. The phylogeny of grey and white matter ratios (data from Frahm *et al.* 1982).

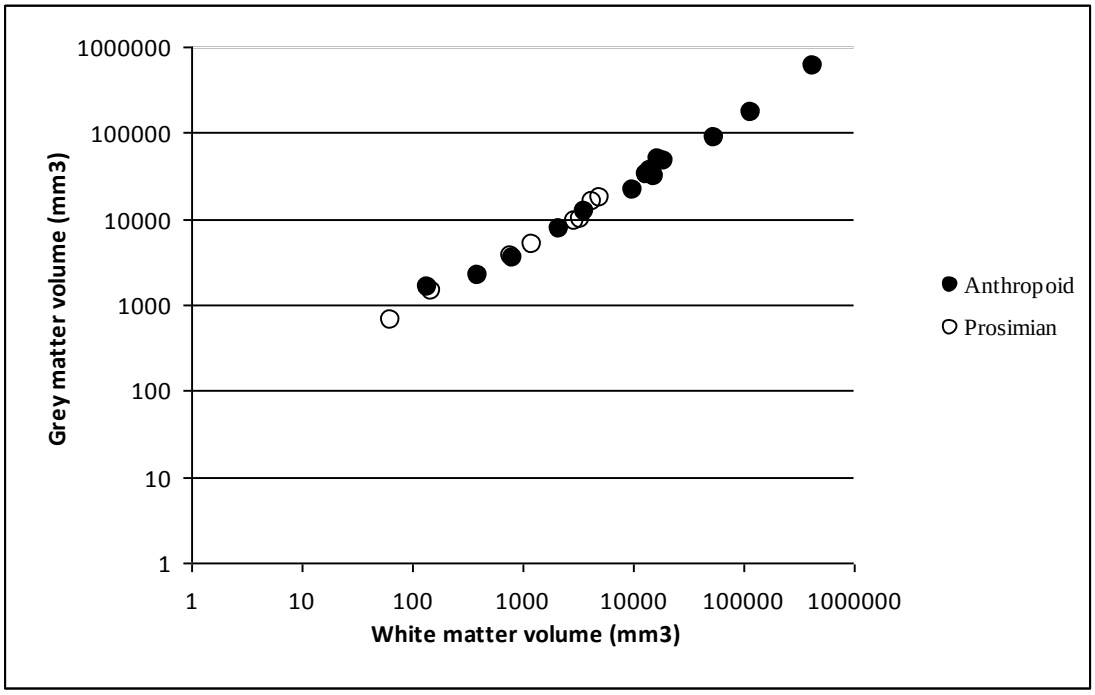
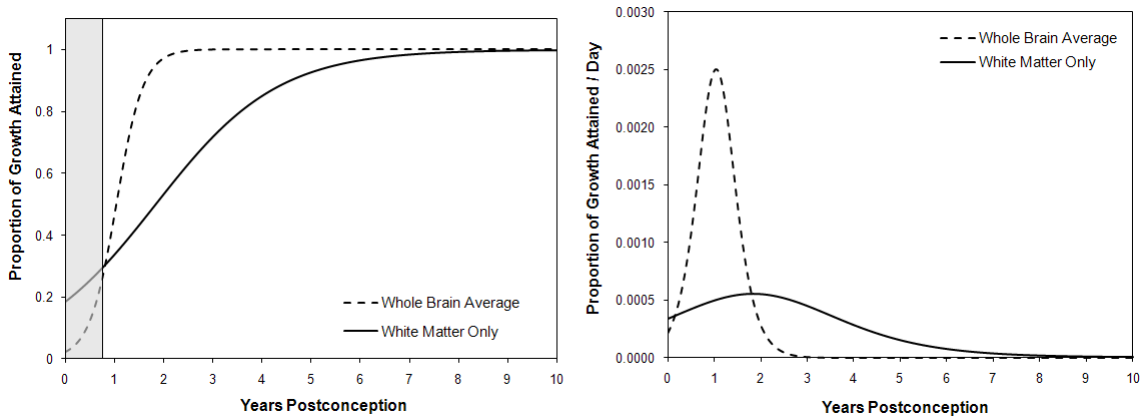


Figure 8. The white matter growth trajectory compared to the average growth trajectory for all brain elements in humans (equations from Klekamp *et al*. 1989).



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