

The paleoanthropological implications of neural plasticity

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Due to neural plasticity, cortical areas can support “ectopic cognitions”. These are cognitions that exist in spite of no specific evolutionary preparation (sight on auditory cortex neural circuits; language upon visual cortex ones). First, their existence is inconsistent with evolution having selected genes to construct cognitions in the same manner that they construct complex body organs such as the eye. Second, their existence suggests that neural plasticity might itself have played an important – but so far – uninvestigated role in human origins. Three interrelated processes can be identified. (1) Human evolution by expanding brain size would have provided extra cortical space for neural plasticity to underlie nonevolved cognitions. (2) Human evolution by changing the body’s input and output capacities (articulate hands, bipedalism, vocal tract and breathing modifications) would have provided new types of information and control for the development of such cognitions. (3) Cultural processes, increased in complexity by these new cognitions, would then through their material, content (symbols), and motivational effects have led to the development of further nonevolved cognitive capacities. Thus, neural plasticity, rather than cognition constructive genes, could have provided the means by which evolution created complex human cognitions.

Natural selection has strongly shaped our species. Our feet are optimal for strident bipedalism (Gebo and Schwartz 2006), our hands are adapted for dexterity (Napier 1976, Susman 1994), and our vocal apparatus and our breathing are well suited for speech and song (Fitch 2000, MacLarnon and Hewitt 1999). However, our brains and their large volume relative to our body size would seem particularly significant given our unique capacity for language, articulate thought and symbolic culture (Deacon 1997, Klein 1999, Klein 2002). The central place of the human brain has also been established by genomic research: not only are roughly half of our genes activated here (Caceres et al. 2003, Gitton et al. 2002, Uddin et al. 2004), but microarray studies of gene activation show that they are (in comparison to nonhuman primates) specifically up regulated in the cerebral cortex (Caceres et al. 2003, Preuss et al. 2004, Uddin et al. 2004), the brain component that has most expanded during human evolution (Deacon 1990, Passingham 1982, Rilling and Insel 1999). Furthermore, genomic studies of nervous system developmental genes, including the brain size determinant genes *ASPM* and *Microcephalin*, and the conjectured speech related transcription factor *FOXP2*, demonstrate that such genes have been under recent evolutionary selective pressure (Dediu and Ladd 2007, Dorus et al. 2004, Enard et al. 2002, Sikela 2006, Zhang, Webb, and Podlaha 2002). Consistent with these links between genes, brain and human cognition, genetic influences have been found upon cerebral cortex regions such as the Broca's and Wernicke's areas that process language (Thompson et al. 2001). These findings, however, by themselves, provide no information as to the nature of the links that exist between human evolution, the human brain, and human cognition.

In this article I argue that genes do not as widely believed dedicate the neural circuits of the brain to carry out the different kinds of information processing that underlie human cognition. Instead, I show that evolution created the human brain with neural circuits that at a basic level are equipotential in nature (though I allow that certain kinds of cortical area-related efficiency might have been selected for). Thus, different cortical areas are formed open in regard to the possible cognitions that they might process, with instead of genes determining their specialization, this being established upon them during their development. Human evolution, I further suggest, exploited in various ways the opportunity offered by such equipotentiality during development to create complex cognitions. These various ways included evolutionary changes to the capabilities of the human body, and changes to the brain (such as brain expansion and prolonged immaturity) that increased the power of the learning environments that humans make culturally amongst themselves to impart cognitions.

This argument is organized in two halves. In the first half, I review the evidence as to whether genes are, in fact, responsible or not for determining the existence of specialized cognitions -- in particular, the human specific ones that characterize the different cortical areas upon the brains of anatomically modern humans. I argue that neural plasticity, above all, as expressed in the phenomena of ectopic cognitions, provides strong evidence against genes having this particular role. This is because ectopic cognitions demonstrate that specialized cognitions can be induced in neural circuits in cortical areas of the brain -- such as sight in the auditory cortex -- that could not have been genetically prepared for them by evolution. This establishes that, in spite of cortical areas normally engaging in different kinds of apparently dedicated information processing, that such areas actually start off, in fact, with the potential to underlie diverse cognitions, including nonevolved ones, at least at a *basic* level.

In the second part of the article, I detail how human evolution and culture might have exploited the equipotentiality offered by neural plasticity to provide humans with their varied cognitive faculties. Human evolution did this, I suggest, in three ways: first, it expanded the brain and so provided additional neural circuits that were free to take on new tasks (prior to humans, nonhuman brains lacked sufficient "house room" for neural plasticity to underlie

neural circuits that carried out nonevolved cognitions). Second, human evolution radically changed the input and output capabilities of the human body, particularly those linked to bipedality, dexterity, and vocalization. This had the effect of providing the human brain with novel sensory inputs and motor outputs that offered new types of activities and associated competences that could be mastered and learnt such as dance, tools, song, and language. Moreover, these new cognitions themselves further facilitated innovation by increasing the developmental stimulation and richness of the learning environments that humans linked to culture, intentionally and incidentally, provide for the cognitive development of children and adolescents. Human culture, I suggest, particularly does this in three respects. First, it provides material resources -- the cognitive ability to read, for example, requires educational support and writing materials if it is to exist. Second, it provides content -- symbols and symbolic creations -- for example, the writing systems and texts needed for literacy. And third, it provides the goals and motivation that encourages people to learn -- literacy, for example, must be useful (texts to read, reasons to write) if people are to devote time to teach, learn, and practice its skills<sup>1</sup>.

### Two Paradigms

There are two theoretical manners in which the connections between human cognition, the human brain and its genes, and their evolution can be explained. First, such explanations can offer a *fons et origo* type of account that in a reductionist manner seeks to combine together in one model both an explanation of the nature of a human cognition as information processing phenomena, and also an explanation as to why that cognitive capacity happens to exist -- its origin. In the context of human evolution, the research program seeking to do this is gene-constructivism. In this, genes both explain how cognitions work (they construct their functionality), and also (due to the past selection of the neural circuit construction that underlies their functionality), why such cognitions arose. Such an approach does not attribute an important role in the creation of human cognitions to culture.

Alternatively, the nature of human cognition, and why such cognitions exist can be separated and theorized as two different, though related, scientific issues. There is no evolutionary reason, after all, why the genetic factors *necessary* for the information processing that underlies human cognitions have also to be *sufficient* ones. A noncognitive example explains why. It is a necessary condition for our ability to read these words that we possess the optical faculties in our eyes (such as the focusing power of their lenses) that allow our brains to see letters, but the evolution of the eye and its lens was not a sufficient requirement for such literacy. Many other enabling factors have also to exist: such as language, a script, materials with which to write, and opportunities and purposes that make it worthwhile to learn to read and write. The genes that act to construct our eye lenses, after all, markedly predate the existence of our capacity to use them for reading (they are thus not sufficient), but without our eye lenses and their constructive genes, we cannot read (they are still however very necessary). In a similar manner, it is possible that the genes constructing the neural circuits necessary for reading written words, might -- and almost certainly did -- preexist their use in enabling us to read. As a result, these genes while important do not suffice by themselves to explain the existence of this skill. Such an approach therefore is not reductionist as it identifies that nongenetic and nonneurological enabling factors are critical, as much as those directly involving the brain and its genes, to the creation of human cognition. Culture, moreover, is identified in such an approach, as playing a particularly key role due to its providing many of the enabling factors that underlie the existence of complex cognitions. Literacy, for instance, as a cognitive ability exists only due to the existence of cultural or cultural processes such as writing symbols, textual content, and educational institutions and communicative practices.

### Cognitive constructive genes

According to the gene-constructivist view, human specific cognitions arise from neural circuits that have specialized information processing competences (Barkow, Cosmides, and Tooby 1992, Cosmides and Tooby 1994, Pinker 2002, Pinker and Bloom 1990). These neural capacities, in turn originated in the genetic selection that shaped the functionality of cognition in the human ancestral environment of the Pleistocene. Thus, the nature of human cognition -- its construction by genes -- reflects, or at least is highly informed, about the selective origin by which these constructive genes create in various cortical areas different specialized cognitive operations.

The analogy here is with genes and how their selection results in the construction of functionally complex body organs. Genes orchestrate, for example, the patterned construction of the eye lens during embryo growth under transcription factor gene regulation (Ogino and Yasuda 2000). The eye lens forms initially, for instance, when an outpocketing of the neural tube called the optic vesicle expands and touches the epidermis so causing the activation of regulatory genes that induce it to thicken, and so produce an intermediary tissue, the lens placode, out of which the future lens will then develop -- a process that critically depends upon the expression of the *Otx2* gene in the epidermis cells at the time when it is touched by the neural tube (Ogino and Yasuda 2000). Genes, as a result of controlling this cellular "patterning" of tissues, build biological organs with specific physical functionalities such as image focusing optics. Therefore, when the various parts of the eyes -- lens, retinal pigments, cornea -- that enable the eye to work as an optical organ are studied, such study investigates functional mechanisms of a physical kind that only exist due to the past selection upon such organ function constructing genes.

Gene-constructivism claims that the same gene selection that determines the construction of functionally working body organs also applies to the development of neural circuits in the brain, and their associated human specific cognitions (Cosmides and Tooby 1994, Pinker and Bloom 1990). It is a powerful idea since if it is correct then it can explain the origins of human cognition. The eye is a adaptation that through its developmental regulatory genes was selected because it provides a key survival advantage. Cognitions also have adaptive utility, and so according to this argument, were chosen in the same way in regards their function. "An organism's phenotypic structure can be thought of as a collection of 'design features' -- micro-machines, such as the functional components of the eye or liver ... Natural selection is a feedback process that 'chooses' among alternative designs on the basis of how well they function" (Cosmides and Tooby 1994: pp. 44-45).

This link made by gene-constructivism between evolution and cognition, can at the outset be questioned in two respects.

First, physical entities (such as light) require highly "engineered" gadget-like organ structures (such as eye lenses functioning as optical devices), so it could be expected that genes for body organs will be selected that pattern them to build organs that in specific ways functionally exploit physical processes. But it is not so obviously the case that genes have also in a parallel manner been selected to construct neural circuits for specialized rather than general types of information processing capacities. The processing of information is notably not constrained in the same way as that of physical entities -- differently designed machines are needed to focus photons and pump fluids, for example, but we are all familiar with computer chips that process without modification the varied kinds of informational operations that underlie the functionality of word processors, spreadsheets, and computer games. Similarly, it might be that neural circuits are constructed by "generalist genes" (Kovas and Plomin 2006), and then only acquire their mature specializations during their development.

Second, gene-constructivism is itself only one of five potential links that might exist between genes and cognition that need to be considered. Genes, in addition, to the possibility of constructing specific competences in neural circuits, can also have effects upon the enhancement of their equipotentiality, their input output fating, their efficiency, and the biasing/snowballing processes that direct their development. In addition, genetic effects can arise from nonadaptive processes such as neutral drift, and these processes might also effect the brain and its capacity for cognitive processing.

*Enhanced equipotentiality.* It could be that evolution selects for genes that create neural circuits that have powerful abilities, (depending upon learning circumstances), to acquire, diverse kinds of cognitive functions. Further on in this article, some of the ways are reviewed in which human evolution might have acted to enhance the ability of neural circuits in the human brain to developmentally acquire novel complex cognitions.

*Input output fating.* Instead of specialization existing in cortical area neural circuits, such specialization might derive indirectly from how genes determine the layout between sensory organs, the musculoskeletal system and the brain, and thus the subcortical inputs and outputs to such circuits. Genes that determine differences in such connections would act to fate neural circuits in different areas to specialize in different particular cognitions, and would do so, even if such neural circuits were initially created as equipotential (Creutzfeldt 1977). Retinal input, for example, is setup to go to the visual cortex, and by this would determine that its neural circuits process sight, even if its neural circuits were equipotential and able to potentially process other cognitions. Indeed, this seems to be the case for neural circuits in the different areas of the cerebellum (a part of the brain adjacent to the brainstem that like the cerebral cortex has expanded in human evolution and has many zones of specialization). Cerebellar neural circuits are each similar in their dendritic, synaptic, axon and neuronal cytoarchitectonic features (Bloedel 1992). However, while such different zones are near identical in their circuitry, they have markedly different and genetically fated connections with the rest of the brain, and this links to them developing different specializations (Bloedel 1992, Ito 2006). A similar process could be at work, at least in part, also in the cerebral cortex.

*Neural circuit efficiency.* Pure equipotentiality in the cerebral cortex would seem unlikely given that its neural circuits (unlike those of the cerebellum) vary considerably in their dendritic, synaptic, axon and neuronal cytoarchitectonics. However, such information processing differences need not necessarily link to innate functional specialization since they could also link to innate efficiency fine-tuning of otherwise equipotential circuits. This possibility is credible. Cognitions -- those in sensory and motor areas in particular -- are selected for reliability, speed, and to be in all ways efficacious to survival. Evolution is not concerned to create, for instance, neural circuits with a mere capacity for basic sight but visual faculties that in their efficiency, reliability and quickness maximally aid an animal's survival by detecting camouflaged prey and predators. As a result, success or not in survival, will strongly act to select for genes that adapt the cytoarchitecture of neural circuits that are fated due to retinal input to usually become the visual cortex so that they are innately optimized for sight, even though the basic visual skills they improve upon might be done potentially due to equipotentiality at a less efficient level in other cortical areas (and vice versa, these "visual" circuits must also potentially do the skills of other areas, albeit less efficiently) . Natural selection, therefore, could be expected to map even an equipotential brain with genetic effects and associated cytoarchitectural differences.

*Biasing/snowballing.* Genes can fate cognitions upon particular areas by creating small initial biases that "snowball" neural circuits into particular specializations. If an equipotential area of the cortex has an initial bias that involves it in a task, this will feedback upon itself, so once it is involved in processing it, that involvement will increase further

through development feedback leading to specialization. The biases precipitating this “snowball effect” could be the initial layout of connections, or some slight advantage in their efficiency. Interactions are another possibility since cognitions develop in the context of each other’s development (Elman et al. 1996, Mareschal et al. 2007). A small bias that channels the development in one of the brain’s areas into one specialization can effect not how other areas in turn acquire their cognitive specializations (Elman et al. 1996, Mareschal et al. 2007, Quartz and Sejnowski 1997). For example, they are unlikely to compete and develop the same specialization, a snowballing related factor has been suggested to be why the equipotential homologous left and right areas in the two cerebral hemispheres develop different functions (Kosslyn 1987). Such biasing/snowballing does not necessarily lead to effective cognitions: some individuals with reading problems initially get trapped into using less efficient cortical areas for word recognition with their problems only resolving when reading interventions shifts them to employ the area used by successful readers (Simos et al. 2007).

*Non-adaptive “neutral drift” genetic changes.* The five links suggested so far between genes and cognition -- construction, equipotentiality, efficiency, connection fating and biasing/snowballing -- all allow that such links are shaped, or at least influenced in some way, potentially by adaptive advantage and so by natural selection. But, natural selection is not the only factor shaping inherited genetic material since this is also effected by nonadaptive processes such as random mutation, recombination and genetic drift (Lynch 2007). The relative importance of adaptive and nonadaptive processes upon changes that happen to DNA has recently become investigable through analyzing directly the genome DNA sequences of different species and individuals. Such genomic research has found, perhaps somewhat surprisingly, that natural selection at the DNA level could have been a less critical factor in shaping the genome than nonadaptive processes (Lynch 2007). Statistical comparison of phenotypic differences also argues that nonadaptive factors such as genetic drift, rather than natural selection, could have been responsible for important physical trait changes, including ones effecting humans, such as the cranial differences that exist between Neanderthals and anatomically modern humans (Weaver, Roseman, and Stringer 2007). This raises the possibility that some of the gene expression differences in the human brain, and their effects upon cognition, might also be unrelated to past adaptation.

### Evolution and openness

The alternatives noted above, compared to gene-constructivism, imply a radically different way of understanding the link between human evolution and human cognition since they predict that nongene factors such as culture might have played a key role in shaping human cognition. This is because, if genes dedicate the function of neural circuits, in much the way that they dedicate the functionality of body organs, then they are set up to carry only out only that genetically determined function. But if genes act in indirect ways, in the context of a general circuit equipotentiality, such as fating neural circuit specialization through their inputs and outputs, or if they increase their efficiency for particular tasks, then in different developmental circumstances, such areas might be able to underlie other kinds of cognition. For example, connections in the brain, though genetically determined, are also initially very exuberant, and only then get refined to those found in adults by extensive elimination followed by myelination -- the thickening of the myelin covering of those connections that remain that acts to improve their ability to conduct neural information (Innocenti and Price 2005). Thus, in those born blind, while the retinal inputs to the visual cortex wither (Breitenseher et al. 1998, Uggetti et al. 1997), their white matter connections in the cerebral cortex mature in the absence of retinal input in a considerably different manner to that found in those with sight (Lui et al. 2007, Noppeney et al. 2005). As a result, neural circuits in their

visual cortices can develop nonvisual capacities through their expanded links with other cortical areas (reviewed below). A similar situation exists if such neural circuits are equipotential but fine-tuned to be efficient for a particular cognition, as this allows that they remain open to process in novel learning circumstances other functions such as those, for example, that underlie written word recognition. However, if cognitions depend upon genes constructing specialized neural circuits in the manner in which they create complex body organs, then such neural circuits will lack this openness to develop other capacities -- much as the eye can only work as optical device, and not (as with the differently selected organ of the cochlea inner ear) as a microphone.

Thus, knowing how genes link to neural circuits is crucial to how the origins of human cognition are understood. This issue is particularly significant since adult humans as a consequence of engaging in language and culture act to create a particularly rich and complex learning environment for the acquisition of cognition in human children and adolescents. Does this have any effect upon the cognitions that humans can acquire, or are human cognitions already set in what they do by the past selection of their genes?

Evidence supporting gene-constructivism.

The gene-constructivist approach, receives seemingly strong support from recent discoveries about the cerebral cortex. Regulatory genes control the development of the brain, including the cerebral cortex, as strongly as they do any other body organ (Molyneaux et al. 2007). Moreover, this development links to making specific changes to different cortical areas. Neurons, for example, in the visual cortex develop from an area-specific gene expression in an embryonic “protomap” (Donoghue and Rakic 1999, Muzio et al. 2002) with cytoarchitectonic areas being developmentally determined by a rich variety of transcription regulators such as *Emx2* and *Pax6* (Lukaszewicz et al. 2006). 106 such genes, indeed, have so far been identified in regard to anterior posterior patterning of such areas (Kudo et al. 2007) -- and this is far less than the total number for the human cerebral cortex since it is patterned in other ways, and this number only concerns the patterning of the less complex mouse brain. This genetically determined embryonic development of the human brain results in various areas having different neuronal features: neuron types exist in the visual cortex that are found nowhere else in the cerebral cortex (Gray and McCormick 1996, Sherwood et al. 2003), the morphology of its cells are different (Elston 2002), and its neurons have double the number of synapses compared to neurons elsewhere in the cerebral cortex (Huttenlocher 1994). The branching complexity and spine density of pyramidal neurons projecting to the visual cortex also varies in an area specific manner (Elson and Rosa 2006). Further, retinal input goes to eye-specific columns that arise even before retinal axons reach the cortex (Crowley and Katz 2002): this helps them build neural networks in the visual cortex that have a peculiar and unique circuitry (Kaas and Collins 2003), moreover, one with features that are genetically inherited (Kaschube et al. 2002). This visual circuitry varies widely across species with, for example, ocular dominance columns being absent in some monkeys (Tigges, Tigges, and Perachio 1977). Interestingly, in spite of our close biological relationship to other apes, even the human visual cortex when compared to them shows distinctive circuitry (Preuss and Coleman 2002, Preuss, Qi, and Kaas 1999).

However, much of the above evidence for genes shaping cortical area specialization, rather than reflecting genes acting to create basic innate area linked operations, could equally well be due to them enhancing cortical area linked efficiencies. One reason for suspecting this is that even when the cytoarchitecture of the visual cortex is developmentally disrupted with disordered layering and abnormal gyration, it can still engage in sight (Zesiger et al. 2002). This suggests that many cytoarchitecture features of the neural circuits underlying the normal visual cortex are not critical for its basic ability of enabling sight. Another argument

is that although some cytoarchitectonic properties such as ocular dominance columns preexist retinal input (Crowley and Katz 2000, Crowley and Katz 2002), and therefore are innate, they nonetheless seem to be optional: in different squirrel monkeys, ocular dominance columns can form elaborate mosaics or be totally absent without obvious effects on the monkey's visual capacities (Adams and Horton 2003).

Gene-constructivism faces two other substantial problems. First, it is silent upon how it is that human cognition could have altered so radically since anatomically modern humans first evolved as simple hunter-gatherers (Amati and Shallice 2007, D'Errico 2003, Klein 2002, Mellars 2006). Contemporary humans possess a range of cognitions that could not have been part of the cognitive phenotype of the earliest anatomically modern human. One example concerns the neural capacities related to literacy which include not only skills directly tied to reading and writing (Cohen et al. 2003), but also its effects upon oral speech, language awareness and reasoning (Morais 1987, Morais et al. 1979, Olson 1996). Mathematics is another area: while even nonhumans possess number processing abilities (Brannon and Terrace 1998), the cognitions of post-agrarian people goes far beyond that which could have existed prior to the Upper Paleolithic<sup>2</sup>. This is particularly so in the abilities of those that specialize professionally as mathematicians which includes not only their nonevolved mathematical faculties but also their capacity to discover new ways in which to think mathematically. For example, the brains of people such as Gauss, Euler and Riemann had the competence to discover entirely new kinds of mathematical thought such as complex numbers, graph theory and non-Euclidean geometry. However, this capacity of their brains could not have been one that was selected for when the genes for their brains -- together with that of all other anatomical modern humans -- were originally selected. There is also the reported nonuniversality of what had previously been taken to be human universal cognitive capacities such as the concepts for counting, quantification, syntactic subordination and perfect tense (Everett 2005). The cognitive styles of people in different cultures in regard to attention, use of categories, rules and logic to explain and predict behavior are also reported to be different (Nisbett 2004). Other differences related to abstract and decontextualized cognition also exist and are discussed below.

But perhaps most critically, the gene-constructivist approach does not explain why amongst all biological species, humans should be the only one that has fundamentally changed its way of life, from that of simple hunter-gatherers coexisting together in small bands in which it evolved, to that which is mostly now of school-educated citizens that coexist in large nation states. To attribute the construction of human information processing to human genes fits poorly in with the extraordinary capacity for flexible and complex cognition in anatomically modern humans that has made this possible -- a flexibility and complexity that, furthermore, has biologically existed and arisen only in this one species.

### Neural plasticity

Neuroscience, moreover, has discovered specific evidence that differences at the basic level of functionality of neural circuits are not constructively predetermined by genes. The phenomenon which shows this goes by the name of "neural plasticity". This phenomena refers to the capacity of neural circuits to shift from one information processing operation to another to which it may, or may not, be related but to which it is distinct (Buonomano and Merzenich 1998, Kujala, Alho, and Naatanen 2000, Pascual-Leone et al. 2005, Yuste and Sur 1999). Neural plasticity can, for example, concern information processing that is of a broadly similar kind such as switching from processing input from the hand or toes to the face as after the loss of limbs (Elbert et al. 1994, Yu et al. 2006), while other forms concern that of an apparently radically different nature such as "visual circuits" in the blind processing linguistic syntax (see below). In the context of the argument of this paper, neural plasticity is used to

refer to neural plasticity in the narrow sense whereby neural circuits take on basic abilities that are radically different (rather than being merely related) to those that could have been given them by evolution (for example, vision instead of hearing, language instead of vision). Such examples of neural plasticity demonstrates that diverse kinds of basic information processing -- including those that are specific to humans -- can arise upon cerebral cortex areas that could not have been prepared for them by evolution (Amedi et al. 2004, Amedi et al. 2005, Burton et al. 2002, Röder et al. 2002a). Evolutionists call such nonevolved functions “exaptations” (Gould 1991, Hampton 2004).

The importance of neural plasticity has only become fully appreciated in recent decades, though it was first identified as a phenomena as early as the end of the nineteenth century by Ramon Cajal, the discover of the neuronal basis of the brain (DeFelipe 2006).. Neural plasticity, it should be noted only describes the general property whereby neural circuits are flexible in their information processing. In its origins, this flexibility can be as complex as the diverse processes that underlie neural circuits and the circumstances that affect them such as their development, or their compensating responses to brain damage. The developmental plasticity that occurs after brain formation includes such physical changes such as axon and synapse elimination after their initial proliferation (Innocenti and Price 2005), the thickening of the myelin surrounding the remaining axons, and the restructuring of the extracellular matrix in which neurons, dendrites, axons, and synapses which make up neural circuits are embedded (Hensch 2005). Injury-associated neural plasticity involves processes that vary considerably depending whether it is in response to acute damage (strokes, bullet wounds), slow neuronal death (such as with slow growing brain tumors (Desmurget, Bonnetblanc, and Duffau 2007)), and whether it happens or not to brains that are also still being shaped by developmental plasticity (Johnston 2004). Neural plasticity usually involves changes down at the level of dendritic spines (Hickmott and Ethell 2006), but sometimes, it can involve the reorganization of the whole brain. For example, in the early stages of Parkinson’s disease there are few cognitive impairments because the brain successfully reorganizes itself in response to basal ganglia dysfunction with compensating changes in the cerebral cortex and cerebellum (Mentis et al. 2003). These developmental and injury related changes to neural circuits are controlled depending upon circumstances by a varied range of transcription factors, growth factors, neurotrophins, and neurotransmitters (Hughes et al. 1999).

Not only can neural circuits themselves change but also critically, as noted above, so can their connections (Lui et al. 2007). This is particularly shown during neural circuit development after the brain is formed as it is initially created with an exuberant excess of axons, axonal branches and synapses that then get pruned down as neural circuits acquire their adult functions (Innocenti and Price 2005). This physically active refinement of neural circuits during development has a major effect in that it considerably increases the energy needs of young brains, and in particular, those of humans. For example, the part of the human brain that most expanded in human evolution, cerebral hemispheres, has double the adult metabolic rate in its cortical gray matter component, and due to its prolonged maturation, this doubling peaks and continues between 4 and 10, and only declines to adult levels around 16-18 (Chugani 1998). As a result, for example, a male child of 7 years of age devotes three times its resting energy to its brain -- over 60% compared to that of an adult at around 18%<sup>3</sup>. The resulting high capacity of the brain during this hyperactive period of brain change for neural flexibility not only allows extensive learning, but also reflecting the flexibility that underlies such learning, an ability to suffer major brain injuries with no effect that if they occurred in adults would cause severe impairment (Johnston 2004).  
What neural plasticity is not

Several theoretical issues need to be clarified about neural plasticity. First, its flexibility is not akin to the plasticity attributed in the past by philosophers to the mind being a “tabula rasa” or “blank slate”. This philosophical idea concerned the passive taking upon the mind of associations imprinted by the environment (or “experience” as argued for by British Empiricists such as John Locke (1987/1690)). In contrast, neural plasticity is an active adaptive flexibility inherent in the information processing of neural circuits that makes them equipotential and so open (depending upon learning circumstances) to underlie potentially very diverse information processing tasks. Such flexibility happens developmentally to neural circuits as a function of the different kinds of informational content, either contained in their inputs, or contained in the feedback that happens to their outputs (such as imparted upon them, for example, by dopaminergic prediction error signals (McClure, Daw, and Montague 2003)). The ability of cortical networks to carry out different forms of information processing, moreover, does not entail that they can do any type of information processing task, only a relatively large variety that have not been specifically prepared for by natural selection. Neural plasticity, further, need not necessarily be independent of gene effects upon different cortical areas, such as those noted above, in regard to input/output layout and efficiency optimizations (Majewska and Sur 2006). Such factors might constrain neural plasticity, or even give it particular opportunities for learning novel functions.

In the place of tabula rasa and its passive taking on of external forms, neural plasticity is better seen as analogous (at least in terms of the contingencies that enable and underlie the flexibilities of its information processing) to programming languages such as C++ or Fortran, and their unrestricted “equipotential” information processing. Programming languages can construct, depending upon how their computational procedures are coded, very distinct forms of information processing abilities, as exemplified in the very different operations done by word processes, spreadsheets, flight simulators and other kinds of software. These capacities are, moreover, often ones that were never initially imagined as possible uses of programming language by the original creators of the programming languages. Here the analogy is not with programming languages as a means of programming but their capacity to create diverse kinds of software, and the fact that the creation of such software diversity is contingent, not upon what the programming language were initially originally planned to create, but upon the circumstances that afterwards determine software development such as the abilities and imagination of those involved in writing its code.

In a parallel manner, the neural circuits in any cortical area have the potential capacity (in terms of such things as their synaptic thresholds, dendrite organization, and communication with other neurons) to build the information operations that underlie a diverse variety of cognitions that are unrelated to the uses to which they were put when they were originally selected as neural circuits. Moreover, like with programming languages, this open potential of neural circuits is contingent --but in its case upon the circumstances that support and shape the acquisition of cognitions. Programming languages and neural plasticity therefore offer respectively to software and cognition, open information processing abilities of enormous potential -- but these potentials can only be realized if there are also appropriate enabling circumstances for their development.

Historically, the neural plasticity trait of equipotentiality was first discussed by Lashley (1929). Lashley, however, held a somewhat different notion to the one argued here: notably, he excluded from equipotentiality many aspects of information processing such as discrimination learning, and even suggested that it, “holds only for the association areas and for functions more complex than simple sensitivity or motor co-ordination” (Lashley 1929: p. 25). But more significantly, in equipotentially, he was concerned only with already developed cognitive capacities, whereas instead the equipotentiality discussed in this article, concerns

primarily that of neural circuits in children and adolescents, and the open potential they have during their development to acquire new cognitions, particularly nonevolved ones.

#### Cortical areas and neural plasticity

Neural plasticity occurs throughout the nervous system (Wall, Xu, and Wang 2002), but it has been studied predominately in the neocortical area neural circuits spread across the cerebral cortex. This complex structure -- six layers -- covers in humans an area of over 1600cm<sup>2</sup> (Barta and Dazzan 2003) upon which were estimated, even in the pre-fMRI days, to be nearly 200 functionally different areas (Roland 1993). With increasing brain size, there is an increasing parcellation of its surface into more cortical areas that increases at roughly the square root of the number of its neurons (Changizi and Shimojo 2005). It is known from functional imaging and the effects of strokes and brain injuries, that neural circuits in different cortical areas normally associate with particular cognitive and affective capacities (Marshall and Fink 2003). This link between function and area is reflected in many of their names: Brodmann 17 (also known as striate cortex, or V1) is familiarly called the primary visual cortex; Brodmann 41 (or A1), the primary auditory cortex; and Brodmann 4, as the primary motor cortex. A major unknown in neuroscience and neurogenomics is how, and in what ways, these area specializations link to neural plasticity and the cortical area-associated expression of developmental genes.

At the cellular level, neural plasticity, exists due to the way in which the information processing abilities of neural circuits can be created and modified by changes to dendrite and related aspects of neuronal cytoarchitecture (Hickmott and Ethell 2006), and their circuit organization (Kalisman, Silberberg, and Markram 2005). Such neural circuit flexibility has been studied in computer simulations (Elman et al. 1996, Quartz and Sejnowski 1997, Rumelhart and McClelland 1986a, Rumelhart and McClelland 1986b)<sup>5</sup>. Research upon artificial neural networks shows it is the information provided to such simulations in their inputs, and the information provided by error signal feedback to their outputs, that allows them, (depending upon that learning information, to acquire different cognitive capabilities, including such diverse ones as motor control, music memory, and syntax (Tong et al. 2007). The same neural network circuitry can thus acquire in an equipotential manner radically different information processing competences. This diversity includes many cognitions that have been argued by gene-constructivists to require prior evolution such as the learning of the syntactic capacity to deeply embed clauses (Hadley and Cardei 1999). Moreover, simulations of neural networks also demonstrate that aspects of cortical areas that had been previously attributed to genes such as the hierarchical specialization of the ventral visual system can in fact also be generated nongenetically (Wyss, Konig, and Verschure 2006).

One question raised by such computer neural networks is whether the networks in the brain that they mimic actually possess such equipotentiality, or whether natural ones have instead been “hardwired” by evolution with specific operations. Neural plasticity suggests that networks should be functionally open, while gene-constructivism suggests they should not.

Mammalian sight and the visual cortex is a good test case for whether or not evolution has constructed basic cognitions into cortical area circuits. (a) Sight is highly selected given its key importance to survival in mammals, and (b) that this selection has been going on continuously for nearly 200 million years (Luo, Crompton, and Sun 2001). As such, the cortical information processing of sight provides a critical test to decide whether the origin of basic cognitive functions is in genes or in neural circuit equipotentiality plus their development. The mammalian visual cortex, for instance, if the gene-constructivist approach is correct, should consist of specially evolved neural circuit ‘sight’ modules. These modules, moreover, should have circuitry (like the physical structure of the eye) that is

developmentally built of dedicated processes -- except that in their case that they will functionally analyze retinal input instead of focusing photons. Further, neural circuits capable of processing sight should not be inducible elsewhere in the cerebral cortex -- as much as evolution created the ability to focus photons to be done only by the eye and not by other body organs such as the heart or inner ear.

Neural flexibility, in contrast, would predict that the basic functionality of sight can occur in areas of the brain other than in the visual cortex. Such evolutionary mispositioned cognition if discovered would be ectopic in that it would be located in areas of the brain for which its functionality could not have been specifically prepared for by evolution as an adaptive specialization. Thus, finding sight in nonvisual cortex areas would provide strong evidence that area expressed genes do not dedicate neural circuits with specialized cognitive operations. Instead, it would argue that genes initially act to create neural circuit with an initially basic functionality -- equipotentiality. Genes still can shape this equipotentially since they can impose inputs and outputs upon different areas, so causing through this area related specialization. Further, upon this area fating, yet more genes add specialized task efficiencies. But such specialization is secondary and allows the possibility of cognitions arising upon them of a different nature in different developmental circumstances. The existence or not of ectopic cognitions, therefore, is theoretically important to many fields such as neural network modeling and neurogenomics since this provides critical information about the role and limits of genes compared to developmental circumstances upon the functioning of neural circuits.

It is also of great consequence to the sciences of paleoanthropology and evolutionary psychology since the existence of ectopic cognitions would call into question whether the "great leap forward" in modern human behavior after the biological origin of the modern humans as a species necessarily required (as has been suggested) advantageous mutations that changed the human brain's capacity for modern human cognitive faculties (Corballis 2004, Klein 2002). If genes do not specialize brain areas to have cognitions, then such gene evolution would not have been necessary for the new cognitions that have dominated humans since the upper Paleolithic. Rather, to understand human innovations would require theoretically appreciating such issues as the subtle but powerful ways in which humans came to shape their cognitive capacities through changing the cultural world that they as humans create amongst themselves. Thus, how we approach the brain, its genes, human evolution, human cognition, and even culture, will critically depend upon whether or not ectopic cognitions exist.

Ectopic cognitions

Mriganka Sur's group at MIT has shown that the auditory cortex of ferrets (*Mustela putorius fero*) possesses the capacity to see (Sharma, Angelucci, and Sur 2000, von Melchner, Pallas, and Sur 2000). Other groups (Frost 1999) have also carried similar work upon hamsters (*Mesocricetus aurast*).

In brief, Sur and his colleagues surgically redirected retinal input in newborn ferrets by lesioning, (1) the lateral geniculate nucleus, and lesioning, (2) the auditory input from the cochleae of their inner ears. The effect of this double lesioning was that retinal axons that would have grown into the lateral geniculate nuclei (an intermediate part of the brain between the retina and the cerebral cortex) cannot go there (effects of lesion one) and grow instead into the nearby medial geniculate nucleus (effects of lesion two is that it no longer receives competing cochleae input). As a result, axons from this thalamic nucleus reproject visual input (not the now absent auditory input) into the auditory cortex. Such redirection results in functional sight (Sharma, Angelucci, and Sur 2000, von Melchner, Pallas, and Sur 2000). Sur's research thus demonstrates that the auditory cortex is capable of acquiring many of the same basic informational operations that are done in the visual cortex in regard to retinal input.

This finding of ectopic visual cognition upon the auditory cortex is remarkable since in the evolutionary lineages that led up to ferrets or hamsters, the cochlea was always subcortically fated to project to the auditory cortex area (via the medial geniculate nucleus) to enable hearing, and that likewise, the retina (via the lateral geniculate nucleus) was always fated to project to the visual cortex to enable sight. Natural selection cannot as result be responsible for the capacity of auditory circuits to process retinal input.

However, it should be noted that Sur's research also questions the relative efficiency with which the visual processing in such auditory circuits is done compared to that in the real visual cortex (Majewska and Sur 2006). Certain cytoarchitectonic aspects of the visual cortex exist that do not appear in exactly the same way as they do on the auditory one when it processes sight. For example, such cortex has less periodic orientation maps with larger orientation domains, and, in spite of processing vision, retains certain cytoarchitecture features of the auditory cortex such as the structure of its thalamocortical arbors, intracortical connections, and at the synaptic level, its spine dynamics (Majewska and Sur 2006, Sharma, Angelucci, and Sur 2000). This suggests that the auditory cortex can acquire the basic operations of sight that are done in the visual cortex but not the degree of their efficiency since this links to cytoarchitectonic features that are innately wired by natural selection into the visual cortex. To use a metaphor noted by Pinker (2002, p 94), it is one thing to turn water into wine -- the auditory cortex into a visual one, but quite another to turn water into the Darwinian equivalent of Château Lafite-Rothschild.

The importance of such evolutionarily ectopic cognitions in understanding human evolution has been doubted as they do not concern the higher cognitions such as language that are human specific (Pinker 2002). This is questionable. First, the basic function of vision in spite of its (i) importance to survival, (ii) circuit specialization, and (iii) nearly 200 million years of natural selection, fails to show such evolved area specialization. Why should human cognition be different with nearly less than a hundredth (and probably much less) of its time of evolution? Second, human specific cognitions unlike vision have the advantage of incorporating culturally transmitted learning, and so they would be expected to be less, not more, dependent upon genes (Kirby, Downman, and Griffiths 2007). Third, no human specific cognition has in its selection faced the intense life or death selective pressure for extreme efficiency such as occurs with sight and the need to detect food and predators.

Moreover and decisively, ectopic cognitions concerning syntax and semantics have been found in the visual cortex in those who have lost their sight early in life or were born blind (Amedi et al. 2004, Amedi et al. 2003, Burton et al. 2002, Röder et al. 2002a). This argues that human specific cognitions like general sensory and motor ones (such as sight) can arise in cortex that has not been evolutionarily prepared for them (Gould 1991).

To appreciate this research, first some background information about congenital and early blindness. One of the anomalies of development in those born blind or whom go blind when young is that while their eye muscles (Kompf and Piper 1987, Fig. 7), optical nerves, (Breitenseher et al. 1998) and lateral geniculate nuclei (Uggetti et al. 1997) weaken or wither away, their visual cortex remains, apart from some local gray matter reduction and white matter increase (Noppeney et al. 2005), indistinguishable from those with sight (Breitenseher et al. 1998). (There are, however, extensive connection changes, as noted earlier and discussed later, with the rest of the cerebral cortex (Lui et al. 2007).) From the late 1980s reports based upon functional imaging (Sadato et al. 1996), brain injuries (Hamilton et al. 2000) and TMS (Cohen et al. 1997) suggested a role for the visual cortex in Braille reading. However, a correlation between activity in the visual cortex and Braille reading is consistent with two explanations: that (1) this is due to processing solely the tactile aspects of Braille, or (2), processing in addition its linguistic aspects such as syntax and word meaning. Three

research groups have studied whether the visual cortex is involved in the processing of *spoken* words by those with blindness.

Brigitte Röder and colleagues (2002) used a method that exploited a peculiarity of German case markers (*der, den and dem*) that allow the word order of noun phrases to be permuted without change in literal sentence meaning. Comprehension time, however, is quicker when the sentence is in the familiar canonical subject, indirect object, direct object word order rather than the rarer indirect object, direct object, subject one (Röder et al. 2002b). Contrasting sentences with easy compared to less easy syntax provides a measure of syntax processing. Since sentence like word strings can also be made with pronounceable pseudo-words, a comparison can also be made between meaningful and nonmeaningful sentences thus allowing the detection of semantic processing. Previous research on this task in sighted adults has shown that it activates the Broca's and Wernicke's language areas in the inferior frontal cortex and the perisylvian region of the left hemisphere (Röder et al. 2002a). Röder explored whether similar patterns might occur in individuals that had been born blind from eye defects. The language areas that lit up in blind-folded but sighted individuals were present, so were also areas in the visual cortex -- this happened both when they processed syntax and sentence meaning. Significantly, this activation varied with syntactical difficulty which limits other possible explanations.

Harold Burton's group (2002) used a verb generation task. Amongst the individuals taking part were eight that had gone blind before the age of three, and six that lost sight after the age of seven. As with the Röder study, blind individuals activated broadly the areas activated by the sighted but with the addition of ones in the visual cortex including the primary visual cortex. While both of the blind groups had activation in the primary visual cortex, the early group had more extensive activation in the secondary visual ones such as the fusiform gyrus. Such visual cortex activations to spoken words have also been found in blind people that cannot read Braille (Burton and McLaren 2006) so it does not link to some peculiarity of learning Braille. It might be objected that such activation might be due to normal mental imagery being more extensively used by individuals with blindness. But the younger individuals had gone blind, the stronger they used the visual parts of their brain. As Burton notes, "it seems illogical that only early blind subjects "mentally visualized" when doing verb generation as these people have the least knowledge of objects" (Burton et al. 2002, p. 3368).

Amir Amedi and colleagues (2004, 2003) used TMS (Transcranial magnetic stimulation -- a noninvasive process by which rapidly changing magnetic fields applied above the skull transiently disrupt the adjacent brain) to produce temporary lesions in the visual cortex. In sighted individuals, this had no effect upon their generation of associated words in a verb generation task but in those born blind (or who had gone blind very early in life) this caused them to make semantic errors. Blind subjects reported that they had problems "coming up with the right word" (Amedi et al. 2004, p. 1268). This suggests the visual cortex was playing a critical role in their capacity for language.

Three studies from three independent research groups (Amedi et al. 2004, Amedi et al. 2003, Burton and McLaren 2006, Burton et al. 2002, Röder et al. 2002a) have thus found using different methods the existence of spoken language associated activation in the visual cortices. Such radical changes in the competences of visual cortex neural circuits is consistent with the white matter connections changes that occur in those born blind with the rest of the cerebral cortex (Lui et al. 2007) that act to allow the equipotentiality of the neural circuits of the visual cortex to take on nonvisual tasks.

Further phenomena exist that supports, or is consistent, with the capacity of the brain to develop ectopic cognitions for which it is not evolutionarily prepared.

*Reorganization*, Ectopic type cognitions would seem to occur during the recovery after brain injury that depends upon cortical reorganization (Taub, Uswatte, and Elbert 2002), as speech and motor control move into areas not specifically evolved for them (Meinzer et al. 2004, Rijntjes and Weiller 2002, Thiel et al. 2001). Brain malformations (Jech, Zemankova, and Krasensky 2004, Lazar et al. 1997), and slow growing brain tumors, also known as low-grade gliomas (LGG) (Desmurget, Bonnetblanc, and Duffau 2007), can also shift language abilities into nonevolved areas. Desmurget and colleagues (2007, p. 909) in their review of low-grade gliomas and cerebral plasticity note that “functional specializations are not the inevitable byproduct of local architectonic differences. The ability of ‘non-canonical speech areas’ (BA 46 and 47) to compensate for LGG invasion located in Broca’s area provides an especially convincing support for this idea”.

*Physical changes in the brain*. The cerebral cortex shows nongenetic physical changes that parallels the presence of neural plasticity in task specialization. Learning to play the violin thickens the connections between the two cerebral hemispheres ( Schlaug et al. 1995), and acts to increase nearly threefold the area devoted to the left hand (Elbert et al. 1995). In keyboard players, there is an enlargement of the motor cortex that is proportional to the number of years that they have been practicing their instrument (Amunts et al. 1997). Hippocampal gray matter volume of taxi drivers increases with how long they have been navigating their routes (Maguire et al. 2003). Musicians also have larger cerebellums -- a difference that increases with the number of years that they have been practicing (Hutchinson et al. 2003). Learning a second language increases the density of gray matter in the left inferior parietal cortex (Mechelli et al. 2004). Literacy has been found to change the organization and the structure of the corpus callosum (Castro-Caldas et al. 1999, Petersson et al. 2007), inferior parietal/ parietotemporal regions (Petersson et al. 2007), and the communication between speech areas (Petersson et al. 2000). At a cellular level, education effects the quantitative measures of the dendrite branching of human cerebral cortex neurons (Jacobs, Schall, and Scheibel 1993). (One could also add here the above mentioned language of the visual cortex in those born blind that links to white gray matter changes (Lui et al. 2007, Noppeney et al. 2005)). The functional changes made possible by neural plasticity would thus appear to lead to the kind of substantial and area-related “hardware” modification of the brain that previously would have been attributed only to the constructive effects of genes.

*Neural pluripotentiality*. Another source of evidence is neural pluripotentiality; that is the profound lack found by brain functional imaging of a one-to-one mapping between any particular cognition and any particular cerebral cortex area (Anderson 2007, Lloyd 2000, Manjaly et al. 2005). The Broca’s area among other functions, processes the recognition of body actions (Hamzei et al. 2003), syntax (Caplan et al. 2000), musical syntax and harmonic sequence analysis (Maess et al. 2001), working memory (Chein et al. 2002), phoneme-to-grapheme (Omura et al. 2004), inner speech (Hinke et al. 1993) and detecting embedded figures (Manjaly et al. 2005). Stefan Koelsch and colleagues have reported that each area in the brain specialized for language also has an involvement in the processing of musical chord sequences (Koelsch et al. 2002). Anderson (2007) after reviewing neuroimaging done upon the human brain and different cognitive tasks found that individual brain areas are activated statistically by an average of over 9 types of task. Moreover, that these tasks usually are not closely correlated as two thirds of the areas are activated by tasks in at least three kinds of task category (out of a possible four). This information processing promiscuity is difficult to explain if the human brain is an evolved collection of dedicated information processing areas as predicted by the gene-constructivist approach.

Ectopic cognitions and human evolution

The existence of ectopic cognitions and neural plasticity reviewed above argues that human evolution did not specialize the circuitry of cortical areas in a gene-constructivist manner (Pinker 2002). This fits in with the research noted above involving artificial neural networks simulations in which diverse types of functionality can be acquired by such circuits (Elman et al. 1996, Quartz and Sejnowski 1997, Rumelhart and McClelland 1986a, Rumelhart and McClelland 1986b, Tong et al. 2007). However, the pressures of natural selection such as spotting food and predators argue that this equipotentially is likely to be combined with genes that lead to the connection fating of particular areas for specialized tasks and that create efficiencies in them linked to survival. Thus, the evolution of the brain rather than being shaped by genes setting up of different cortical area with dedicated cortical area linked functions (as implied by gene-constructivism), is based instead upon the setting up of equipotential neural circuits upon which genes influence area specialization (through determining their inputs and outputs), and upon which genes determine cytoarchitectonic features that give such areas task related efficiencies.

The idea that brain evolution created cortical circuits with a basic level of equipotentiality has radical implications for paleoanthropology. This is because it implies that neural circuits will be functionally open to acquire novel operations and so cognitions lacking any biological and so evolutionary precedence. After all, for the auditory cortex, the function of sight is equally as much an unbiological function as any cognition of cultural origin (the auditory cortex was never in its neural circuits prepared by evolution to do either kind of information processing task). This raises the possibility that human cognitions owe their existence not to constructive genes but to neural plasticity working indirectly with other changes created by human evolution, particularly those that enhance the capacities of the human body, and through such capacities, the uniquely rich cultural based learning environment that humans create amongst themselves for developing children and adolescents.

Neural plasticity and human evolution

The potential of neural plasticity to underlie human cognitions is not the same, as humans actually exploiting neural plasticity to develop them. To give a metallurgical example, aluminum can be potentially be turned into a jet aircraft engine but the hypothetically potential of that metal to be engineered in this way is far from ensuring that actually happens. In addition to the properties of aluminum that make it suitable to be engineered into engines, engineers and a range of other factors must also be in existence (the desire for flight, an industrial economy, knowledge of thermodynamics, machine tools, engine designs). Potentials because of their dependence upon such enabling conditions can therefore hypothetically exist (as with aluminum and making engines) without necessarily ever been realized (as they were not until recently with the potential of aluminum to form engines). Thus, to avoid the mystery of why human cognitions exist, this paper must begin to detail these neural circuit competence enabling factors. Identifying neural plasticity is important, but not enough, since we need to also understand how in humans the potential offered by neural plasticity to underlie new cognitions could have, in fact, come to be realized, as it has, in actual human cognitive capabilities.

Neural plasticity, after all, has existed (like the optics of our eyes) long before the human brain itself evolved. Part of the evidence, for ectopic cognitions, indeed, comes from research on ferrets, a species whose lineage separated from that of primates, and so our own species around 100 million years ago (Cao et al. 2000). Moreover, since mammalian brains have a broadly similar neurocircuitry that goes back to their evolutionary origins (Aboitiz, Morales, and Montiel 2003), neural plasticity must date back at least roughly two hundred million years to the early Triassic era (Aboitiz, Morales, and Montiel 2003, Luo, Crompton, and Sun 2001). Even this date might be an underestimate since the neural circuits of the mammalian cortex are closely related to those in earlier evolved vertebrates (Aboitiz,

Morales, and Montiel 2003). There is thus no reason to presume that neural plasticity is specific to mammalian neural circuits, and might in fact be inherent to neural circuits in general (Kalisman, Silberberg, and Markram 2005).

We must therefore ask why a phenomena present in the brains of *all* animals is the basis for novel cognitions *only* in one? This issue, it should be noted, does not affect gene-constructivism, since, its theory in explaining the nature of human cognition, also explains its origin. Evolution by its nature works upon phenotypes constructed by genes, and these have to be expressed if they are to be selected. Thus, if genes prepared cortical neural circuits in a constructive way (as they do the eye), understanding how these cognitions work will at the same time also tell us much about the reasons for their existence. Any paleoanthropology based around neural plasticity, however, faces the higher bar of having to explain why neural plasticity (a property possessed by even early primitive brains) stopped being merely a biological potential, and became the actual basis, only in humans, for evolutionary *de novo* cognitions. As a result, such a neural plasticity based approach must go outside neurology, and explore the largely unknown and diverse nonneurological factors, including culture, that, though their interactions with neural plasticity, enable the existence of human cognition.

Here, I shall conjecture that these enabling paleoanthropological interactions occurred in three broad areas: encephalization, body capability changes, and culture (though note, I shall give a very peculiar definition of what constitutes “culture” linked to my concern to describe how it acts as an enablement factor upon information processing). While two of these factors concern genes and natural selection, they do so by how they indirectly act upon the brain’s faculty for cognition, and as such, leave the brain open to acquire culturally enabled capabilities. My discussion of these factors is done not to specifically review them, but to establish that genes do not have to directly construct cognitions as there are neural plasticity based alternatives. As a result, it seeks to show the existence of such alternatives, even if their discussion is far from complete. I am not aiming here to provide a new roadmap of human evolution, just to illustrate the nature of some of the neural plasticity related paths that it might have taken.

#### Encephalization

As noted, a key problem facing the neural plasticity account of human cognition is to explain why neural plasticity leads to novel cognitions only in humans but not in other animals. That human brains are different, I suggest, arises from them having the neural room that allows neural plasticity to be used to acquire new cognitions. Here three factors come together. First, brains are energetically expensive, particularly during development, so a strong selection pressure exists to keep them as small as possible within the allometric range allowed for by body size. Second, extra neural circuits allow expanded abilities, for example, in humans, the size of an individual’s oral vocabulary is proportional to gray matter density in their bilateral posterior supramarginal gyri (Lee et al. 2007). In nonhuman animals, likewise brain size links to the cognitive flexibility needed to avoid predators, acquiring food, survive in novel environments and success in social coexistence (Aiello and Dunbar 1993, Ratcliffe, Fenton, and Shettleworth 2005, Sol et al. 2005). Third, cognitive tasks can crowd out each other in the brain because neural circuits have a limit upon how far they can take on multiple information processing tasks. This happens in human brains when they are injured and so have reduced numbers of neural circuits. For example, following left hemisphere brain lesions, language skills can develop in the right hemisphere but only at the expense of crowding out preexisting right hemisphere visuospatial functions (Lidzba et al. 2006); a similar crowding out has been suggested for nonverbal skills in the right hemisphere following left sided epilepsy (Strauss, Satz, and Wada 1990). These three factors result in the existence of an evolutionary squeeze upon what brains can do with the consequence that evolution, in general, does not allow neural circuits superfluous to essential sensory and

motor needs. However, without neural circuits free of such competing demands, neural plasticity cannot underlie the creation of new cognitive capacities.

Human brains solved this problem by being large, and more importantly large for a primate of human body size -- about three times as large as would be expected (Deacon 1990, Deacon 1997, Passingham 1982, Rilling and Insel 1999). Thus, humans have a processing area of neural circuits greatly in excess of that needed for basic sensory and motor faculties (which are roughly proportional to body size). This as a result leaves substantial areas of cortex with neural circuits that are free, at least potentially, to acquire through their neural plasticity novel nonevolved cognitions.

The unique use of neural plasticity for nonevolved cognitions by humans does not imply that neural plasticity lacks importance in nonhuman animals. It is, for example, useful for fine-tuning cognitions during brain development, or in recovery after brain impairment. However, neural plasticity in nonhuman brains is restricted by the above noted limited brain size. The neural plasticity present in animal brains, as a result, might be capable of underlying nonevolved cognitions (as shown in the above ferrets) but as a developmental potential this was biologically, "blocked", until humans arose with their massive (for their body size) brain expansion.

Extra cortical space provided by brain expansion is not, however, the only factor that increases the capacity of the human brain for novel cognitions. As brain size increases, so does the proportion of the brain taken up by connections -- white matter -- to the power of  $4/3$  (Zhang and Sejnowski 2000). Further, the number of its interarea interconnections increases by the square of their number (Changizi and Shimojo 2005), and the total number of cortical areas by the square root of its number of their neurons (Changizi and Shimojo 2005). This situation creates the possibility of more complex attentional processing during cognition as it leads to the potential existence of brain organization that is increasingly highly segregated (short-range) and integrated (long-range) (Fair et al. 2007). Indeed, greater refinement of intracortical integration has been found to link to quicker action selection choices (Boorman et al. 2007). In this context, it is also interesting to note that one theory of autism -- a condition in which individuals can be normally intelligent by measures such as IQ tests but subtly impaired particularly in social cognitions -- is that it results from impairment in the development of cognitive integration caused by reduced refinement of intracortical connections (Minshew and Williams 2007).

Bigger brains also have deeper and increasingly convoluted cortical folding and so more areas, fundi, deep within the folded cortex that are surrounded by a wide "rim" of white matter connections. This white matter rim provides an opportunity of extra communication that seems to act to enhance their processing capacities: 47% of functional imaging peaks locate to them, while such cortex makes up less than 8% of the cerebral cortex (Markowitsch and Tulving 1994).

Another factor linking human brain evolution to enhanced neural plasticity are genetic changes in how synapses function. Genes that are up regulated in the human cortex include those that effect synaptic plasticity (Caceres et al. in press, Uddin et al. 2004). More interestingly, such up regulation also happens to genes that either support a high metabolic rate, or protect the brain from its effects (Caceres et al. 2003, Uddin et al. 2004). This is intriguing because human brain expansion in contrast with that which happens with other animal brains, does not link to a decreased metabolic rate<sup>5</sup> as it is as high as that of smaller primates such as macaques (Uddin et al. 2004, p. 13034). As noted earlier, human brain metabolism has a prolonged period peaking at 4-10 years of age that then tails off to adult levels around 16-18 that reflects learning associated changes to neural circuits. It is possible that the higher than expected level of adult human brain metabolism reflects a continued capacity to acquire new cognitive skills after adolescence that is due to retaining some of the

energy demanding processes associated with such developmental neural plasticity. This retention could have been responsible for the observed up regulation in human brains of metabolism related genes (Caceres et al. 2003, Uddin et al. 2004).

An additional link between brain expansion and neural plasticity is the prolonged nonadult immaturity of humans (Leigh 2004). During this period in humans, there is extensive reshaping of cortical grey and white matter thickness (initially relative thickening of the former, and then of the later) that directly correlates to development of intellectual abilities (Shaw et al. 2006). Such prolonged maturation crucially depends upon the continued availability of stimulating learning experiences that provide the developmental information for acquiring cognitions. While comparative measures of the richness of learning environments are not available, it is reasonable to suppose that those of human are considerably much richer and better adjusted to the learning needs of nonadult brains than that of nonhumans due to language and the active tuition of skills and knowledge that is unique to humans (Csiba 2007). Consistent with such stimulation having positive effects, the cognitive abilities of children at 11 links to the degree to which they as three year old sought out novel experiences and so give themselves rich learning experiences (Raine et al. 2002). Further, such early stimulation might also directly enhance neural plasticity since it has been found that environmental enrichment enhances the speed of development of neural processes such as vision by up regulating neurotropic factors involved in neural plasticity such as BDNF (Sale et al. 2004). Thus, human culture and its superrich and supportive learning environment matches the developmental needs of larger brains and the extended period in which they acquiring cognitions.

Novel peripheral inputs and outputs

Even if the human brain has the opportunity to use neural plasticity to create novel cognitions, this does not explain why that opportunity, in fact, came to be exploited. One factor is the evolution that happened outside the brain of changes to the capacities of the human body.

The brain and its functions are strongly influenced by the nature of the information and external control supplied by its sensory organs and its musculoskeletal system. For example, if an eye from a large species of salamander is grafted into a small one, the number of axons in the optic nerve is increased and with this occurs an expansion in the number of cells in the visual part of its brain (Twitty 1966). Individuals with brains adapted for three retinal pigments that have four in their retinas have increased color discrimination (Jameson, Highnote, and Wasserman 2001), while other evidence argues that the brain's capacity for trichromacy preceded the evolution of the third photopigment (Jameson, Highnote, and Wasserman 2001). In those born without arms and hands, the somatosensory cortex that had previously would have represented the hand gets used as part of the areas that represent their still existing feet and lips (Kamping, Lütkenhöner, and Knecht 2004). Neurons in the motor cortex that control breathing can shift to controlling arm muscles when surgeons repair brachial plexus avulsion by reinnervating disconnected nerves to the spinal motoneurons (to which they link) that previously controlled respiration (Carlstedt et al. 2004).

Innovations in the human body's capacity for sensory experiences and doing things thus could have allowed human evolution to exploit neural plasticity to reshape human cognitions without it also selecting genes that directly change the brain's neural circuits. There have been major changes to the capabilities of the human body in which this might have occurred. Notably, these have happened to the vocal apparatus and breathing, hand dexterity, and the ability of human to remain stably bipedally upright while running and engaging in skilled upper body actions (such as accurate throwing)<sup>6</sup>. Even the appearance of the human eye is different with an expanded white sclera either side of the dark iris that contrasts strongly with it that makes human gaze direction easily identifiable (Kobayashi and

Kohshima 1997). Related to these body innovations are human specific cognitions such as those that underlie speech, song, tool making dexterity, rhythmic dance and projectile aiming. The human eye by aiding people identify where a person is looking and so attending, for instance, has been suggested to promote the development of the ability to read other's intentions (Ricciardelli, Baylis, and Driver 2000).

To detail the links between the evolution of human body and how new cognitions arise from their novel inputs and outputs would involve extensive discussions. However, detailing all of them is not central to my argument, which instead is the nature of the links between the human body and cognition as a means by which genes can create new cognitions by acting indirectly through neural plasticity. Therefore, for illustrative purposes, I will restrict myself to a brief example that shows the general principle: the link between how our physical ability for producing sequences of vocalizations provides one of the enablements for spoken language.

#### Multiple vocalizations and language

Humans evolved a new manner of breathing, thoracic breathing, for use in vocalization, that is distinct from diaphragm respiratory breathing (MacLarnon and Hewitt 1999)<sup>7</sup>. Normal diaphragm breathing has the respiratory function of clearing carbon dioxide from the lungs and refreshing the body with oxygen. In this, it is usually slow and equal in duration between both in- and out-breaths. It is used for this purpose by both humans and nonhuman mammals. Thoracic breathing, unique to humans, in contrast, usually involves muscles other than the diaphragm such as the intercostals, (though in those with spinal injuries, it can also involve only the diaphragm (Meyer 2003)). Its out-breath is substantially longer than its in-breath as this allows humans to make on the out-breath multiple strings of song, spoken and other vocalizations. Such human vocalizations include laughter vocalizations, an affective response, that unlike song and speech, that is shared with nonhuman primates such as chimpanzees. However, though chimpanzees and humans both laugh, they do so in noticeably different ways reflecting the lack of thoracic breathing in the former: chimpanzees make a laugh on each in- and out-breath, Ha. Ha. Ha. Humans, in contrast, string them along on a single out-breath spliced up with vocalizations -- ha, ha, ha, ha .. (Provine 2000). The inability of chimpanzees to breath in a manner that allows them to pronounce on each out-breath continuously varying strings of vocalizations is why attempts to teach them language have only been successful with manual gestural-signs or sign boards.

Thoracic breathing, a human specific respiratory adaptation, has the crucial consequence of making it possible for the human brain to perceive and produce communications of a particularly complex kind. For example, the expressive sophistication of human language as a form of communication is closely linked to its use of a large vocabulary of many thousands of lexical tokens (personal, place, object and animal names, descriptive verbs, nouns and so on). Many of these words are syntactically modifiable by internal and affix changes upon lexical pronunciation. Without such a vocabulary of syntactically modifiable words, the lexical complexity and richness that gives human language its communicative power could not exist. The thousands of lexicals that underlie human language, however, depend for their existence upon the human ability to sequentially recombine (Studdert-Kennedy 1998) and imitate (Skoyles 1998), a small subset of elementary phonetic pronunciation units vocally into larger and syntactically modifiable word pronunciations. The vocal concatenation that facilitates this --and so the enormous vocabularies of human languages and indeed syntax itself (see Nowak, Plotkin, and Jansen 2000), however, in turn, can only exist due to thoracic breathing. This is because without thoracic breathing, there would be no respiratory means to concatenate elementary vocalizations units needed to make spoken words on each prolonged out-breath. Thus,

thoracic breathing, directly enables human to have spoken language with its vast spoken word vocabulary, together with syntactic and multi-word sentences.

Here evolution by making a change in the spinal and supraspinal networks needed for the muscular control that underlies thoracic breathing (MacLarnon and Hewitt 1999), created the opportunity to exploit a potential that the nonhuman brain could do already, but only if it was provided with the right kind of vocal productive capability. For example, it is only because we can produce multiple combinational vocalizations upon each out-breath that the developing brain gains the background experience of constantly hearing a rich vocabulary of many thousands of spoken words daily in syntactic and complex descriptive sentences (the average adult says around 16,000 such words a day (Mehl et al. 2007)). The evidence from Kanzi is that the nonhuman brain if given this continual experience of spoken sentences can learn to recognize and comprehend words and their meaning (Savage-Rumbaugh and Lewin 1994). But while the nonhuman primate brain, as evidenced by Kanzi, has the receptive ability to comprehend human-type language, this potential, due to the lack of suitable respiratory control in nonhuman primates, is necessarily latent as such vocal communication in nonhumans is never made. In this way, it may be that many of the key elements enabling language are already present in the nonhuman brain (Aboitiz et al. 2006), but without the body capability innovations, such as thoracic breathing, the biological opportunity for them to be adapted for linguistic use does not exist.

Thus, changes in the inputs and outputs available to the brain can have by themselves the potential to radically change the cognitions that arise in the human brain. As a result, human evolution by enhancing the capabilities of the human body, could (when working with neural plasticity) at least, have in part, innovated human specific cognitions, rather than such cognition being the direct result of evolution having selected specialized neural circuits.

Neural plasticity and culture

Changes in the capacities of the body such as thoracic breathing cannot explain the new cognitions that arose after the shift from simple hunter-gathering -- the form of life in which humans evolved -- to the more complex social and technological life found in agrarian, industrial and now postindustrial societies. This suggests the existence of additional enabling factors that humans create amongst themselves that derive from culture and the cultural transmission of cognitions (Cavalli-Sforza and Feldman 1981, Henrich and Boyd 2002). These factors are present also to some extent in nonhuman animals (Bonner 1980), particularly in closely related ones such as chimpanzees (Whiten et al. 2007). However, they are limited in their ability to create new cognitive abilities since nonhuman animals lack the cognitions enabled by expanded brains, and the enhanced body capabilities discussed above, that allow humans to produce a particularly rich, vibrant and propagating cultural world -- language, symbols, kinship systems, myths and everyday knowledge -- between each other.

To explore the interactions between culture and neural plasticity, I will again use an analogy with programming languages such as Fortran and C++, since the development of their ability to underlie the information processing of diverse forms of software, is also, like neural plasticity and cognition, closely linked, I suggest, to culture-like enabling factors.

First, the existence of different kinds of software depends upon material support factors. Skilled programmers must exist, and their existence, in turn depends upon other enabling preconditions such as the ability to type and read ASCII code, manuals, programming know how, teaching and computer courses. Beyond them an economy must exist that pays programmers or gives them free time. There must be the technological know-how and the industrial and financial environment to build and design computers and supply a reliable source of electricity. This vast complex web of material factors creates a support "culture" without which programming languages (as an isolated potential) would never result in any software.

Second, software needs informational content if it is to work. Word processors need dictionaries, spreadsheets need mathematical formula, and software in general depends upon libraries of modules, add-ons and computer standards to expand their functionality. This type of enablement factor once acquired can be shared between programs, accumulate and generally expands what they can achieve.

Motivations are a third enablement factor. Before people invest time and effort in creating software, they need to appreciate that it can achieve worthwhile tasks. Goals and aspirations are thus potent factors in software development. For instance, we are familiar that computers are useful in storing and manipulating financial data but it took a Harvard Business School student, Dan Bricklin, in the late 1970s to foresee that usefulness and then use a programming language, Integer Basic, to create, VisiCalc, the first spreadsheet. Motivation must also exist to teach, learn and master proficient programming skills.

Three types of cultural enablement

These three kinds of factors (material, content, and motivational) that when combined with programming languages support the existence of software development, I suggest, apply also to neural plasticity and the cultural enablement of human cognition.

*Material culture.* Modern human cognition closely links to diverse forms of material, educational and other kinds of support. All contemporary industrial societies require the fulltime schooling of children and adolescents, and manufacture or supply the technologies that aid or facilitate the acquisition and use of many types of education dependent cognitive capacities (paper, pens, books, desks, *Sesame Street*, laptops, Wikipedia etc). These kinds of material support were absent until historically recently: the first humans were never formally schooled, nor experienced the technologies that are now so supportive in agrarian and more recently, particularly in industrial societies, of nonevolved cognitions. Before the existence of such material culture, the capacities of the human brain for creating many kinds of cognition as a result had to largely go unrealized. Equally, it should be noted, that other kinds of cognition have been lost such as the hunter-gatherer expertise to track animals, and the capacity to cognate and participate socially within complex systems of kinship and social hierarchy. Contemporary humans have a different, but not necessarily a more advanced cognition to that of earlier humans.

*Content (symbolic culture).* Many human cognitions depend upon communally shared content. Mostly this is symbolic: that is a sensory token stands in a shared across community in a signifier signified relationship with a cognitive or affective entity (a letter for a speech sound; a peculiar hand gesture for the affiliative emotions of fraternity bonding) (Deacon 1997, Peirce 1932, Skoyles 2008, Skoyles and Sagan 2002). Anthropologically, human societies vary considerably in the nature of their symbols. Writing, diatonic music and all but the most simplest arithmetic depend or are extended by symbolic notation.

Abstract words, at least in Western culture, seem to be historically recent. The Archaic Greek vocabulary of Homer, for example, has nine concrete verbs to describe sight but no abstract ones (Snell 1953, pp. 1-5). The nine concrete verbs referred to specific aspects of vision such as to have a particular look in one's eyes, *δέρκεσθαι*; looking about carefully, *παπταίνειν*; or to look with ones mouth open, *θεασθαι*. It was not until the Classical Greeks that verbs existed such as *θεωρεω*, that specifically referred to the later decontextualized function of seeing (in the case of *θεωρεω*, to spectate). Numbers beyond three do not exist in many societies, and the numbers that do exist might be exclusively concrete rather than abstract (some examples of the existence of concrete numbers in English such as duet, trio, quartet, .. [players], or twins, triplets, quadruplets ... [births] ) (Menninger 1969). Concrete and abstract words are processed in different brain locations (Binder et al. 2005, Pulvermuller 1999), and by different neural processes (Crutch and Warrington 2005) with the processing of concrete words occurring in parts of the brain that deal with sensory experience as well as

language, while abstract ones link solely to those concerned only with language (Binder et al. 2005, Pulvermuller 1999).

The words available to people such as these provide a powerful means to organize and structure cognition (Hunt and Agnoli 1991). The presence or absence of words that describe objects in relative compared to absolute coordinate spatial frameworks effects the nature of spatial perception (Majid et al. 2004), and the availability of tense description words changes the temporal perception of events (Boroditsky, Ham, and Ramscar 2002). The lack of exposure to intentional state words (such as in children born deaf to nondeaf speaking parents) impairs and delays the acquisition of the appreciation that others have mental states (Peterson and Siegal 1995)<sup>8</sup>. Further, symbols can enhance the ability to engage in decontextualized cognition. Individuals literate in alphabetic scripts gain a better ability not only to focus upon the phone elements that make up speech, but also identify mispronunciations in the context of a communication (though they can identify them in isolation), as they can attend to the process of communication as well as its communicated meaning (Morais 1987). Literacy more generally upon ideas is also claimed to have this effect (Olson 1996). In addition, schooling together with literacy has been identified since Luria's expeditions to Uzbekistan in the 1930s as modifying the cognition so that events are understood in decontextualized and abstract conceptual terms rather than direct sensory and personal experience (for a recent review, Kotik-Fredgut 2006).

*Motivational culture.* Third, human societies have radically changed between hunter-gatherer bands and hi-tech societies in the goals they set people to be socially and technologically competent. For example, cultures differ in regard to individualism and collectivism (Markus and Kitayama 1991), and communal, ranking, equality, marketing values (Fiske 1992). They also differ in the types of autobiographical memory, autonomy, self-concepts and narrative co-constructions of the self that they encourage (Wang 2006). Many cognitions are forms of expertise and these are contingent upon cultural values that motivate the extensive practice, carried out regularly over many years, that are needed for their acquisition (Ericsson and Lehmann 1996). Without the cultural goals that motivate such sustained learning, many nonevolved cognitions would never be supported and so could not have arisen.

## Conclusion

A core question facing paleoanthropology and anthropology is the nature of the links that exist between the human genome, human cognition, and through its past evolution, the human brain. This paper has argued that due to the ignored role of neural plasticity, the links between them are more open and dependent upon culture than generally appreciated. Notably, the existence of neural plasticity establishes that the main process by which genes are at present assumed to underlie cognition -- the direct construction of cognitive functionality -- is unlikely to have played a role in shaping human cognitive capabilities. The existence of ectopic cognitions demonstrates that cortical areas start off with an underlying potential to acquire the basic abilities that develop in other areas (sight on auditory cortical areas, for example). Neural network simulations, further, show that diverse basic functionalities can be created upon nonspecialized networks of neuron-like entities. Evolution, therefore, fashioned the biologically unique information processing faculties of humans, not directly through genes, but *indirectly by neural plasticity interacting in multiple ways with enabling factors that shape their development*. Genes do effect the brain such as fating otherwise equipotential neural circuits into particular specializations -- retinal input directed to the visual cortex determines that it normally processes sight, and area expressed genes also make such fated areas more efficient for particular evolved tasks. But such connection fating and area efficiency occurs in the context of considerable cortical area

equipotentiality as happens in those born blind that reuse their “visual” neural circuits by co-opting them to aid their processing of language.

Neural plasticity, however, fails on its own to explain the origin of human specific cognitions since neural circuits have been plastic back to their earliest occurrence in the first vertebrates. This suggests the existence of a critical role for a variety of cognition enabling factors. Thus, this paper has explored the enabling factors that interacted with neural plasticity to turn what is a potential present in all brains into a capacity for nonevolved cognitions that is unique to humans. Three paleoanthropological factors have been identified: encephalization, human specific body capabilities (thoracic breathing, bipedalism, dexterous hands), and culture (material, content [symbolic] and motivations).

Expanding the size of the brain overcame the crowding out limit that otherwise acts to stop neural plasticity from underlying the creation of *de novo* cognitions in nonhuman animals. Many human specific cognitions depend upon specific innovations in the capabilities of the human body. The existence of speech, I illustrate, as an example, is closely linked to the respiratory adaptations that allow multiple vocalizations to be strung together so allowing the pronunciation of individual words and their combination into sentences. Such human specific innovations changed the human brain’s inputs and outputs, and so modified and enriched the opportunities of its neural circuits with the result that they could develop new -- human specific -- cognitions. Genes and human evolution therefore could have profoundly changed human cognition but without this directly involving the evolution of new innate cognitive faculties.

Another factor was human culture and so the rich world of learning experiences that humans create amongst themselves. This provides diverse factors that support and determine the development of cognition. Many cognitions depend for their existence upon material support -- teachers and pens and paper -- for example are needed for literacy. Culture also provides cognition with a rich source of symbolic content. Reading and writing need scripts and written texts. Culture also provides goals that reward people to learn and teach that control the kinds of cognitive faculties such as reading that are acquired. The complex world that humans create for each other, thus, exposes the human brain to a diverse web of enablement factors that when combined with its neural plasticity results in humans being able to acquire a wide variety of evolutionarily novel cognitions.

In summary, human cognition studied by anthropologists and paleoanthropologists is not determined directly by genes in the manner that they create body organs (even though the brain contains many cognitive specialized areas). Genes play an important role but this is indirect. Instead, it is developmental neural plasticity that is primary to human cognition since it is this factor that allows the human brain to acquire cognitions from, or with the assistance, of the highly stimulating and unique cultural world that humans make with each other.

## Footnotes

1. Here and below reading and writing are used as illustrations because of the extensive research upon them, and the incontestability that they are nonevolved. This does not, however, imply that literacy is more important than many other forms of human cognition, particularly those that are no longer found nor valued in contemporary humans, such as, for example, the ability to participate socially in complex kinship systems.
2. This includes those individuals that do not go to school but learn arithmetical skills in the course of trading with currency such as Brazilian street sellers.
3. The average 7 year old male child at 23kg has a basal metabolic rate of 50 W, an male adult aged 18-30 of 95kg one of 100W (Henry 2005). The basal metabolic rate of that child's brain is 32W, while that of the adult is 16W (using data in (Chugani, Phelps, and Mazziotta 1987)). Basal body metabolic rate is energy consumption at rest and so does not include activity: to give perspective for male adult aged 18-30 of 95kg this increases it by 55% for "light", 76% "moderate", and 110% "high" activity (Henry 2005, Table 21). Mental activity, in contrast, has only very small effects upon brain metabolism, and where it increases, this is localized, and occurs in the context of decreases in other brain areas.
4. Neural networks simulations are not the same as A.I. (artificial intelligence) though both are implemented upon computers. In A.I., computational abilities derive from how they are programmed but in neural networks these abilities come from the information with which they receive, or with which they are trained, that changes the "strengths" of connection within them that determine how they process information (Elman et al. 1996, Quartz and Sejnowski 1997, Rumelhart and McClelland 1986a, Rumelhart and McClelland 1986b).
5. One reason for metabolic decrease with increased brain size is that white matter has a much lower rate of metabolism than gray matter, together with the fact that with increasing brain size, white matter increases in relative proportion to gray matter (Zhang and Sejnowski 2000).
6. Elsewhere, I have discussed the nature of the change that happened to the human body in regards to manual, bipedal and vocal capabilities. Briefly, humans show across all three major motor domains a unique capacity to make anticipatory adjustment needed for their motor stabilization. For a manual activity such as knapping this concerns the hand hold the hit core stone, in bipedality, vertical posture, and for vocalization diverse factors but notably -- and critically for thoracic breathing -- the control of constant subglottal pressure. This does not link to a change in neural circuit capacities but a massive expansion of their number particularly in the cerebellum and cerebral cortex and so complimentary to ideas presented here (see Skoyles, submitted).
7. The human ability to make multiple vocalizations upon a single pulmonary out-breath is biologically unique. Birds, for example, use a different respiratory apparatus that employs anterior and posterior air sacs to create a unidirectional airflow through their lungs, and insert minibreaths in between their song notes (Suthers, Goller, and Pytte 1999)..
8. Deaf child born to deaf parents using sign language do not have such problems. Unfortunately, even when nondeaf parents of deaf children learn sign language it is usually too elementary in proficiency to provide a linguistic experience rich in intentional words for the mental state language learning needs of their young deaf child.

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