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Review

Plausible Mechanisms for Brain Structural and Size Changes in Human Evolution

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A B S T R A C T

Encephalization has many contexts and implications. On one hand, it is concerned with the transformation of eating habits, social relationships and communication, cognitive skills and the mind. Along with the increase in brain size on the other hand, encephalization is connected with the creation of more complex brain structures, namely in the cerebral cortex. It is imperative to inquire into the mechanisms which are linked with brain growth and to find out which of these mechanisms allow it and determine it. There exist a number of theories for understanding human brain evolution which originate from neurological sciences. These theories are the concept of radial units, minicolumns, mirror neurons, and neurocognitive networks. Over the course of evolution, it is evident that a whole range of changes have taken place in regards to heredity. These changes include new mutations of genes in the microcephalin complex, gene duplications, gene co-expression, and genomic imprinting. This complex study of the growth and reorganization of the brain and the functioning of hereditary factors and their external influences creates an opportunity to consider the implications of cultural evolution and cognitive faculties.

Key words: *encephalization, brain evolution, radial glia units, minicolumns, mirror neurons, neurocognitive networks, microcephalin complex, genes co–expression, genomic imprinting*

Introduction

The increase in brain size is considered to be one of the key aspects in human evolution. The origin of the genus *Homo* is connected to the growth of brain matter and most significantly in the lineage which led to *Homo ergaster* and subsequently to *Homo erectus* and *Homo heidelbergensis*. This trend gained speed and simultaneously the expansion of variability began to occur (is it only an artifact of increased brain size?). It is true that the remains of our ancestors allow us to describe approximately the quantitative aspect of encephalization, but structural changes cannot be characterized, nor can their causes be explained. For a deeper understanding of this process, it is necessary to attempt to reconstruct that which allowed brain development and cognitive functions to occur, and what internal mechanisms accompanied this development. From an evolutionary standpoint, brain size is of course very significant; however, behavioral and cognitive abilities and the cultural attributes created from them (Table 1) are more important, but do not necessarily have an immediate or close connection with brain size.

Paleoanthropology links increased relative human brain size compared with other hominids mainly to ecological, food-related, behavioral and sociocultural influences. Researchers relate encephalization to intake of meat[1], fat ingestion and metabolism of lipids [2], general dietary behaviour and nutrition[3], masticatory mechanics[4], lifespan[5], size of social groups[6], use and manufacture of tools[7], visual perception[8], verbal communication[9], intelligence and mental abilities[10], social behaviour, cognition and mind[11], cognitive reserves[12], neonatal brain growth[13]. Others set forth hypotheses involve a combination of environmental, social, dietary, or other factors, climatic conditions, ecological demands and social competition[14], genomic interactions with environment and culture[15] etc. These hypotheses provide explanations for the increase in brain

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TABLE 1
CHANGES OF VARIOUS COMPONENTS OF HUMAN CULTURE AND SOCIETY CONCERNING ENCEPHALIZATION AND BRAIN EVOLUTION (HYPOTHETICAL JUDGMENT)

Time period (years ago)	Around 7 millions to 2,5 millions	About 2,5 millions to 1,5 millions	2 millions to 1 million	1 million to 200,000	200,000 to circa 30,000	Circa 30,000 to some few thousands	Last some few thousands
Representatives	*Australopithecus* (except *Paranthropus*)	*H. Habilis* *H. Rudolphensis*	*H. Ergaster*	*H. Erectus* *H. Heidelbergensis*	*H. Neanderthalensis* *H. S. Sapiens*	*H. S. Sapiens*	Contemporary mankind
Brain volume	Over 450 ccm	600–750 ccm	850 ccm	800–1200 ccm 1100–1400 ccm	*H. N.*: 1520 ccm (1200–1700 ccm) *H. S. S.*: 1450 ccm (1100–1800 ccm)	Stagnation (?)	–
Complexity of cortex	Elevating of cortex plasticity, Potentiation of specific structures of ape brain	Escalation of neuron number in cortex Differentiation of cortex areas, formation of minicolumns Spreading of mirror neurons system Configuration of comprehensive neurocognitive nets					
Communication and speech	Grooming, gestures, Sound signalisation	Gestures, Labelling	Gestures accompanied by mimic and sound displays, signs	Beginning of vocal speech (?)	Speech (at *H. S. S.* Articulate speech), Syntax	Languages, Symbols, Instrumental notices	Modern languages, Writing
Substitence	Gathering, occasional hunting	Gathering, scavenging, occasional hunting	Gathering, frequent hunting (?)	Gathering, frequent hunting	Gathering and group hunting (specialization, grand animals hunting)	Before 10,000 years horticulture, pasturage, later Early agriculture	Advanced agriculture, civilization, complex society
Material culture	Branch and stick using, occasionally stone using	Origin of stone industry	Sophisticated stone tools (acheulleén)	Fire, residence protection	Burials, art	Before 7,000 years neolit	Advanced technologies
Society size	70 and more individuals (?)	80–100 individuals	120 and more individuals	150 and more individuals	More individuals ?	Hundreds to thousands individuals	Hundreds to millions individuals
Social interactions, behavior	Fission-fusion	Structuring into some smaller cooperative groups ?	Societies are structured into cooperative groups	Long prereproductive period, mating, man´s participation on childs care, grandmothering, sex (gender) division of activities	Marriage, kinship, incest tabu (?), three generation model, biginning of ethnicity (distinction we/others), Tribe structure	Alliance relationships between groups, nuclear family, ethnicity, division of works and specialisation	Nationality, higher social institutes (even states)
Concretely known gene alternations			ASPM		FOXP2		
Genomic processes	In parallel: mutations of single genes (microcephalin complex, LISP, FOXP genes etc.), genes duplication, genes co-expression, genomic imprinting, others regulation mechanisms (histo-compatible system, growth factors, microrna, skip genes)						

size throughout evolution with respect to selective advantages, but provide little detail concerning possible mechanisms responsible for these changes.

Qualitative and structural reorganization of the brain during evolution is as important as an increase in brain volume. This reorganization includes changes in cortical area proportions, and gross and histological characteristics of these areas. For example, the visual cortex underwent remodeling rather than an increase in size, as is evident from studies of primate comparative neuroanatomy[16]. Increases in frontal lobe size compared with other parts of the telencephalon is characteristic of Hominoids[17], while development of the prefrontal regions[18] and white matter in the frontal lobe[19] is specific to modern humans. The prefrontal region and its folding is a major evolutionary landmark in the emergence of human cognition. The cerebellum also underwent a three-fold relative increase in size likely related to its significant role in the learning and control of movement schema, most importantly including communication and speech production[20]. Genetic effets and the influence of environmental factors varied regionally within the brain – high heritability is found for frontal lobe volume, Heschl´s gyrus, Broca´s area[21] etc. Twin studies showed high heritability estimates for specific brain structures and for overall brain size in adulthood (between 66 and 97%)[22].

Other significant factors in the evolution of the human brain include: hemispheric asymmetry[23], changes of neurotransmitters activity in different parts of cortex[24] (e.g. GABA, glutamate receptors), increase white-matter (e.g arcuate fasciculus[25]), heterochrony and transcriptional neoteny[26], cortical folding in relation to encephalization, unique and extraordinary cortical plasticity[27], specifity of some human astrocytes[28].

Brain functions of modern humans are researched in detail with the developments of modern imaging methods in the neurosciences, especially functional magnetic resonance imaging (fMRI) and positron emission tomography (PET). A fundamental benefit of this research comes in understanding the specific significance of individual areas of the brain and the creation of neurocognitive networks. This plays a significant role in brain morphology and its relation to evolution. It also makes it possible, for example, to study the relation of brain function and the creation of tools[29] or to explain the beginnings of human instrumental activity. Scanning of endocasts by three-dimensional computer tomography (3D-CT) and 3D brain reconstruction of the ancient *Homo* provide information about brain surface[30].

Here we briefly discuss some innovative ideas drawn from recent work in genetics and developmental, comparative, and functional neuroscience which may inform our understanding of human brain evolution.

(1) Choice principles of neurosciences explaining human brain evolution

These concepts (which enable an explanation of brain evolution) include in particular the theory (1a) of radial units and minicolumns, (1b) mirror neurons, and (1c) neurocognitive networks. The human brain has grown larger and at the same time functionally and structurally shaped itself based on the processes listed (Figure 1).

(1a) According to the radial unit hypothesis, the cortex forms as newborn neurons migrate outward along vertically-oriented processes of radial glial cells[31]. Germinal cells undergo asymmetric cell divisions at the ventricular surface of the telencephalic vesicle and daughter neuroblasts migrate in tandem, aligned along the glial fiber to settle as a single-cell wide column in the developing cortical plate[32]. These ontogenetic cell columns are considered the basic cytoarchitectural unit of the neocortex. Each radial unit is derived from its progenitor according to a common regulatory gene program. This allows for genetic parsimony so that the same program can be iteratively initiated by each radial glial cell; increases in their numbers allows for expansion of the neocortical surface area in both development and evolution. The progenitor cell population expands in the ventricular germinal zone by means of symmetrical division, its numbers increasing exponentially with each cell division cycle. Thus, limited developmental changes in processes regulating cell-cycle duration or timing of apoptotic cell death may result in changes in the surface area of the neocortical sheet. Apoptotic selection likewise regulates proliferation and neurogenesis affecting cortical development[33]. Apoptotic regulatory genes APAF1, CASP3, CASP9 and Ephrins have been shown to influence proliferation and neurogenesis.

Ontogenetic cell columns provide the template for emergence of generic cytoarchitectonic columnar arrangements in mature cortex, and continuity of radial morphometry common to both structures has been demonstrated throughout prenatal, postnatal, and mature development[34]. Termed cortical cell minicolumns, these modular structures have been proposed to represent the basic functional microcircuit of neocortex[35]. Minicolumns are defined by a shared intrinsic connectivity and pat-

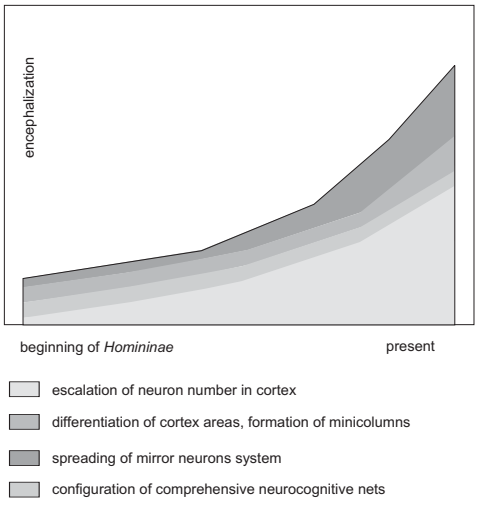

Fig. 1. Deepening of brain complexity in human evolution – scheme.

tern of inputs and outputs which subserve common functional operations. Variability of humans minicolums increased in tangential diameter relative to other primates[36].

This prototypical organization and functionality is adapted to the specific requirements of various cortical areas. Cortical patterning is governed by germinal zone expression of multiple overlapping gradients of morphogens and signaling molecules across the cortex. These establish progenitor cells within a germinal zone 'protomap', which guides neurogenesis, area- and layer-specification and axo-dendritic development[37] in the formation of minicolumns. Formation of cortical microcircuits is also affected by the composition of interneuron populations which migrate from the ventral telencephalon into the developing cortical plate[38], by intrinsic activity, but also by reciprocal interactions with developing area-specific non-local afferent projections[39]. Changes in the processes governing cortical arealization may have provided a basis for cortical parcellation and the emergence of novel specialized areas during evolution[40], while overall expansion of the progenitor pool and increased numbers of minicolumns constituting cortex is proposed to be an important mechanism of encephalization[41]. Minicolumns are a pervasive feature of the neocortex and have been identified in a great diversity of cortical areas and mammalian species, associated with characteristic differences in morphometry and morphology[35]. These differences can cast light on area-specific functional characteristics.

(1b) The discovery of mirror neurons in the frontal lobes of primates has potential importance for understanding the evolution of human cognitive functions. The original discovery was associated with the study of the F5 region of the premotor cortex in rhesus monkeys[42]. These neurons are activated in parallel with associated regions of the motor cortex both during motor activity and when passively observing corresponding activity in other individuals. In this manner, they take part in the recognition and generalization of such activities. Increased brain size created a space for the formation of new cortical regions, allowing the derivation of mirror neurons from those of analogous networks coordinating perception and action. This mechanism is proposed to be a key contributor to the evolution of human social behavior and language[43]. Arbib has proposed that speech formation is a vocal/orofacial communication system parallel to and differentiated from networks subserving gestural communication[9]. Mirror neurons are similarly linked to the emergence of instrumental activities[44], and the comprehensive reorganization of transcortical networks into a »mirror brain« to coordinate mirror neuron representations of intentional actions[45].

(1c) Another basic concept, the theory of neurocognitive networks[46], is important for understanding the relationships between the evolution of the brain, its ontogenetic development and influences of the sociocultural setting. According to this theory, various regions of the brain cortex, which are functionally related or physically connected, are interconnected in a network-like manner, thus forming a new unit known as the neurocognitive network. The verbal neurocognitive network is an important example, which manifests in the recognition and production of speech, including interconnection with the lexical and semantic component[47]. Another example is the neurocognitive network for the recognition of the face, its partial characteristics and mimic expressions[48]. The concept of neurocognitive networks is based on an individualized, plastic and hierarchically functioning interconnection of individual specialized regions.

(2) The role of genes in regulating the brain size and its differentiation

During human evolution, the following genetic mechanisms played an important role: (2a) gene sequence changes, (2b) addition or deletion of whole genes in the genome, and (2c) changes at the level of gene expression including genomic imprinting. Recent findings confirm the role of all these mechanisms. New advances in sequencing technology enable more thoroughly and complexly to argue about genetic correlates to human brain and its development[49].

(2a) Many genes have an additive effect on brain size (and also on intelligence to a certain degree), although more important is their mutual balance. However, some genes have evidently played a more significant evolutionary role than others, above all microcephalin complex. A mutation of the ASPM gene causes primary microcephaly with reduction of brain size by up to 70 %. Differences in ASPM between humans and apes are believed to be a cause of an increased size and expansive growth of neocortex[50]. Sequences of this gene enabling an increase in brain size underwent positive selection some 3 to 4 million years ago[51]. Similar evolutionary importance was confirmed for microcephalin MCPH1[52]. Positive selection is described for both genes in anthropoids[53]. Several ASPM haplotypes have been identified in modern humans[54] and are estimated to have emerged recently in human evolutionary history (37,000 years BP). The impact of these haplotypes on variations in brain structure remains unclear but it should be kept in mind that their effects should be observable primarily in population level variations, and common polymorphisms of both microcephalin and ASPM have probably little direct influence on individual brain size and intelligence[55].

The following genes identified as part of the microcephalin complex are considered to be important regulators of brain size: MCPH1, ASPM (identified as MCPH5), CDK5RAP2 (MCPH3) and CENPJ (MCPH6)[56]. Their effects probably took place in parallel; for example CENPJ gene took effect depending on the higher protein intake[57]. These genes have an importance in the formation of neuronal cytoskeleton and thereby probably interfere with the course of mitosis during the division/neurogenesis[58]. A mitosis disorder was identified as a direct cause of the insufficient brain growth known as microcephaly. We believe that the said genetic mechanisms, in association with the radial unit theory (see below) form a basis for the causal explanation of the process of increase in the brain size. The effects of these genes are subtle

and manifest in the arrangement of the brain cortex[59]. They do not determine the brain size and related behavioral and cognitive functions directly[60], but instead form regulatory factors, signal molecules, receptors, enzymes, and so on, in a complex network of relationships[61]. The overall influence and expression of the gene and balanced relationship between several (or many) genes were of more importance than the immediate selective advantage of the individual mutation of the given gene.

Apart from the said genes with an apparent evolution importance of their mutations, other genes are also known which might be expected to be related to an increase in the brain size (AHII, LIS1, BIRC1). For example the LIS1 gene encodes the protein dynein, which is known to act as a regulator of neuronal migration alongside radial glia in the cortex and influence the neuronal division process[62]. Mutation of this gene causes lissencephaly, an insufficient development of the cortex with lower gyrification. A wide group of factors that interfere with the transcription during the expression of other genes also includes genes from the FOXP line[63]. From this group of genes which regulate the differentiation of the cortex in the ontogenesis, the gene FOXP2 is considered to be important for the development of speech functions. It is interesting to note the theory on the possibility of genetic drift in the FOXP2 gene in the evolution of the modern human[64]. There is mounting evidence that new, so called brain genes might have contributed to the evolution of the human brain phenotype[65].

(2b) Gene duplications are a generally accepted mechanism of evolution. Recently, it was found out in a comparison of chimpanzee and human genomes that »multiplication« of, and subsequent changes in several non-structural genes occurred in the hominin line. The role of HAR (human accelerated regions) »genes« has been discussed recently: A total of 202 genes have been described in this group. Rapid selection in these genes is a feature specific to humans in comparison with other hominid species[66]. One of these segments, designated as HAR1, differs in 18 bases between humans and chimpanzees, while the chimpanzee differs only in two bases compared to the domestic fowl[67]. HAR1 serves as a template for transcription of two different RNA molecules, designated as HAR1F and HAR1R, which encode no proteins but have regulatory functions in the early phase of development of the cortex, between the 7th and 19th weeks of development[68]. Forty-eight other HARs have also been identified with genes involved in neurodevelopment.

(2c) As has become apparent, regulation of gene expression, rather than modification of specific gene products, is the principal process guiding brain development and evolution. Recently identified mechanisms influencing evolution of gene regulation include jumping (skip) genes and microRNA and other non-coding RNAs[69]. Evidence suggests that regulatory network genes evolved more rapidly in humans than in chimpanzees[70]. Theoretical principles based on these findings remain incomplete. It is clear however, that developmental pathways are based on co-expression of genes within regulated repertoires rather than on serial gene expression[71]. In contrast with chimpanzees, higher parallel activity of groups of genes has been found in humans. An example of gene co-expression is provided by a group of four genes (LDOC1, EYA1, LECTI, and PGAM2) involved in the development of the cortex and cerebellum[72]. Intensive genes expression is possible to convay as: »The human brain seems to be running hot in all sorts of ways«[73].

In recent times it has been discovered that the inhibition or activation of genes by means of chemical markers from one's parents – called genomic imprinting – has a direct effect on the creation and activity of the cerebral cortex. Put in different terms, the respective allele of the offspring's gene is applied according to whether it was transferred from the father or mother[74], the same as with the IGF2 growth factor[75]. Epigenetic factors can be considered to be one of the many adaptive evolutionary mechanisms[76]. Focusing on certain categories of objects in perception (people and social relationships versus objects and spatial organization) is determined by imprinting in relation to gender. This evidently has a direct connection to the conditionality of certain disorders (depression, schizophrenia versus autism). Imprinting can manifest itself for example in the expression of the FOXP2 gene in connection with the development of speech functions in evolution[77]. All of this suggests that by means of this gene imprinting, life style and other external living conditions can work together in affecting brain development. This is not necessarily a matter of genomic imprinting, but could also be the direct impact of gene expression in the creation of the structure of the cerebral cortex in the initial phases of its development. Hormones may also function as regulatory factors in such a manner[78]. Thus it is possible to conceive the creation of a relationship between the evolution of culture and mental abilities on one hand and brain evolution on the other.

Conclusions

It should be kept in mind that genetic changes regulating brain size have had effects on brain organization. That is, metabolic and geometric constraints of bigger brains with more neurons limit the density of long range connections in brain networks, with implications for the emergence of relatively autonomous, specialized cortical areas. Changes in these phenotypes have reciprocal interactions with the developmental, including cultural, environment. These processes provided a basis for the emergence of neocortex structure (minicolumns), mirror neuron system and functional interconnection of such structures within neurocognitive networks.

Encephalization should be understood in relation to the structural and functional remodeling of the brain rather than a »mere« increase in the brain size. The relationships between the emergence and elaboration of the mirror neuron system and of neurocognitive networks on the one hand, and specific human developmental phases on the other hand, remain speculative at present. Genetics has provided some preliminary results concerning

brain development. These results are now being informed by new concepts of the functional organization of the nervous system which will hopefully serve to understand how interactions between development and culture are mediated.

Findings gained from the given summary of brain research from the perspective of paleoneurology and neuroanthropology should also be taken into consideration while generating new hypotheses and their validity should be subsequently verified through experiments. Only then will it be possible to better understand the complex mosaic of the causes and sources of encephalization and, in the process, explore the specifics of the human species based on the properties of the brain.

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MEHANIZMI MOŽDANIH STRUTURNIH I VOLUMENSKIH PROMJENA U LJUDSKOJ EVOLUCIJI

S A Ž E T A K

Razvoj ljudskog mozga može se interpretirati na različite načine. S jedne strane, razvoj je vezan uz promjene u prehrambenim navikama, socijalnim vezama i komunikaciji, kognitvnom vještinama i umu. Uz povećanje veličine mozga s druge stane, razvoj ljudskog mozga povezan je sa stvaranjem kompleksnije strukture mozga, ponajviše korteksa. Potrebno je istražiti mehanizme koji su povezani s rastom mozga i proučiti koji od tih mehanizama dopuštaju i određuju rast. Postoje brojne teorije za razumijevanje evolucije ljudskog mozga koje potječu iz neuroloških znanosti. Te teorije koncepti su radijalnih jedinica, minikolona, zrcalnih neurona i neurokognitivnih mreža. Tijekom evolucije, očigledno je da se dogodio niz promjena s obzirom na nasljednost. Te promjene uključuju nove mutacije gena u kompleksu mikrocefalina, duplikacije gena, gensku koekspresiju i genomski imprinting. Ovo složeno istraživanje rasta i reorganizacije mozga te funkcioniranja nasljednih faktora i njihovih vanjskih utjecaja stvorilo je priliku da se uzme u obzir i sudjelovanje kulturalne evolucije i kognitivnih sposobnosti.