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The Nexus between Neurodegeneration and Advanced Cognitive Abilities

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Abstract. – This exploration of how the unique susceptibility of the modern human brain to neurodegenerative illnesses appears to be related to that brain's acquisition of complex cognitive functions is informed by both neuroscientific data and present understanding of recent human evolution. The near-absence of such pathologies in other extant primates prompts an analysis of the differences between the human and ape brains, which relate both to relative size and structure. Relevant human brain illnesses implicate specific brain areas in these pathologies, such as the frontal cortex, temporal and parietal lobes, limbic system, and basal ganglia in illness like schizophrenia, bipolar disorder, Parkinson's disease, and others. The expansion of these same areas has also facilitated the advanced cognitive abilities marking the emergence of *Homo sapiens*. The authors then explore the questions of when, in this process, the brain diseases first established themselves in the genome, and why natural evolutionary processes failed to select against them. Based on the similarly deleterious introduction of gracility in formerly robust human populations, attributed to breeding mate selection becoming influenced by cultural constructs, the hypothesis is proposed that the dramatic rise of culture over the past 40,000 years or so also rendered the toleration of these brain pathologies possible. Just as the modern human has become a fetalised, neotenous form of ape through unintended self-domestication, a similar process also protected unfavourable mutations of recent encephalisation, such as demyelination, against natural selection. *[Human evolution, cognitive functions, brain abilities, brain pathologies, natural selection]*

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1 Introduction

In comparison to most areas of the sciences, particularly those that have the potential of serving special interests of humans, palaeoanthropology and Pleistocene archaeology have made relatively little progress over the last one and a half centuries. To some degree this is an expected outcome, as these disciplines offer limited scope for deliberate strategies in knowledge acquisition: key finds are made randomly rather than by design, and interpretation of purported evidence is subjugated to social and political subjectivities. Archaeology caters to the need of societies to contemplate their histories; it creates their self-flattering narratives of how they became what they are; at its worst it explains, rationalises and justifies the replacement of past societies by usurping their interpretation in the service of today's powers.

An example is the universal depiction of the hominin ascent from lowly simians to likenesses of a deity. Archaeology is primarily a political pursuit (e.g., managing the past of the defeated and superseded societies on behalf of history's victors, today's nation states), and as such helps shape the current mythologies about the human past. On the whole, we are fond of perceiving ourselves as the

pinnacles of evolution; yet evolution does not create paragons; it adapts living things to everchanging niches and circumstances. In the case of our subspecies, the self-deception is easily exposed. Far from being an "evolutionary pinnacle," *Homo sapiens sapiens* offers a good example of a creature that has experienced some rather deleterious changes in its recent evolution. To begin with, its brain volume has decreased by about 10% in a geological instant (Henneberg 1988). In previous hominin history, it took hundreds of millennia to gain such an increase in brain size, an encephalisation regarded as remarkable, but the rapid decrease during the final Pleistocene – a time of unprecedented demands on the hominin brain – has barely been commented on. During this very same period, cranial robusticity of the *H. sapiens* lineage waned so dramatically that many researchers perceived different species. Not only is the skull of a robust human several times as resistant to fatal impact as that of an extant gracile, the differences in robusticity of the postcranial skeleton are almost as dramatic. Moreover, based on the comparative development of skeletal muscle attachments, robusts such as "Neanderthals" are thought to have possessed up to twice the physical strength of graciles. And to cap it off, it appears that humans are experiencing neotenisation, i.e., the retention into sexual maturity of physical characteristics previously seen only in juveniles. Modern humans resemble chimpanzees anatomically most closely in the latters' fetal stage;[1] they are fetalised apes.

Neoteny or the fetalisation of a species is in some respects detrimental to it, but it also involves evolutionary benefits. Most importantly, it facilitates the retention of plasticity or "morphological evolvability" (de Beer 1930: 93; cf. Fuentes 2009). Adaptively useful novelties can become available as maturation genes are freed by pedomorphosis (juvenilisation of the body morphology). Nevertheless, seeing our subspecies as a neotenous ape does conflict with regarding ourselves as noble creatures destined for great things, and has remained so unpopular that we raise it reluctantly – and only because it is essential in this article.

However, the deleterious somatic effects accompanying the change from robust to gracile humans, occurring in Europe essentially during the past 40,000 years (40 ka), and elsewhere within roughly similar timeframes, are not the only detriments we have experienced in our most recent evolution. There can be no doubt that they left us more susceptible to injury, physically weaker, and with a smaller brain than our robust ancestors. But to make matters worse, our brain has become vulnerable to a range of neurodegenerative pathologies, which other extant primates are largely free of (Walker and Cork 1999; Olson and Varki 2003). These range from dementia to bipolar illness, to schizophrenia, from obsessive compulsive disorder (OCD) to sociopathic or antisocial personality disorders and diseases involving demyelination or dysmyelination of axons such as multiple sclerosis. They affect largely the very same areas of the brain that are involved in its higher cognitive functions (Damasio et al. 1990), rather as if they were the price our lineage pays for its relative level of intelligence (Bednarik 2011). For a detailed description of the specific types of brain illness associated with *Homo sapiens sapiens*, see Helvenston and Bednarik (2011).

Although much progress has been made in recent years in determining the nature of these human brain illnesses, some key aspects of them remain largely unknown: how did these pathologies initially develop; at what time in our evolution did they appear; and especially why did evolutionary processes apparently fail to select against the relevant genetic predispositions? In this article we will explore these questions, and we will do so within a revolutionary frame of reference that rejects certain constructs created by recent Pleistocene archaeology. The perhaps most consequential of these is the notion that the world population of robust *H. sapiens* groups was replaced by a new species of humans, unable to interbreed with the incumbents. Not only does this "replacement model" lack any archaeological, palaeoanthropological, or genetic evidence, it is demographically naive, was initially based on fake evidence, and is contradicted by some of the recent genetic evidence (Bednarik 2008b). For instance, the presence of the same variation of the FOXP2 gene in a "Neanderthal" as the one found in extant humans, where it is the only known gene implicated in the development of speech and language (Krause et al. 2007), renders the replacement notion unrealistic. Recent genetic analyses confirm not only that "Neanderthal" genes persist in recent Europeans, Asians, and even Papuans, but suggest also that Neanderthals interbred with the ancestors of Europeans and Asians, but not with the ancestors of Africans (Green et al. 2010; Gibbons 2010). Rather than replacement or genocide, the record implies that all reasonably habitable regions of the Old World and Australia were fully occupied by tribes before 40 ka ago (see evidence of the presence of robusts in the Arctic Circle; Schulz 2000–01; Schulz et al. 2002; see also Pavlov et al. 2001), and that these experienced introgression or genetic drift. Certainly in

1 Haldane (1932); De Beer (1940); Ashley Montagu (1960); Bednarik (2008b).

central Europe, the change from robust to gracile occurred gradually, and was pioneered by the females (Bednarik 2008a). Similarly, the changes from Mode 3 to Mode 4 technologies were gradual and took tens of millennia in most regions of the occupied world. The view that gracile genes gradually replaced robust ones is considerably better supported by empirical evidence than the view of a sudden cultural or genetic replacement still favoured by most archaeologist commentators.

Be that as it may, the issue concerning us here is not how but *why* the change occurred. The most parsimonious explanation currently available is that of unintended self-domestication (Bednarik 2008a, 2008b). Culture-induced mate choice, universal among extant humans and based on cultural constructs, is unknown among any other animal species, so it must have been introduced at some point in time, and the evidence as it stands suggests that it was phased in roughly 40 ka or 30 ka ago, gaining influence over time. It can account fully for the greatly accelerated human neotenisation since that time and is thus the most likely reason for the inexpedient somatic changes in recent *Homo*. It is, therefore, highly relevant to the questions concerning the development of our species' neurodegenerative pathologies, which we intend to explore here. Before doing so, we propose to review the relevant empirical evidence concerning a number of brain illnesses, most of which are thought to have been absent in our Miocene or Pliocene ancestors. As we will see, the very areas of brain enabling *Homo sapiens sapiens'* unique cognitive abilities are areas affected by these brain illnesses. We will begin by considering those features unique to human brains, for these are most implicated in brain disease.

2 Features Unique to Human Brains

2.1 Overall Brain Size

Few studies in the last decades have addressed comparative issues of the organisation of the primate brain[2] and discussions of the human brain have until very recently focused mainly upon the issues of size.[3] Thus, we will first discuss the size of the human brain as one of its most distinguishing attributes. The data available included body weight, brain weight, and the size of the endocranial space of adult animals. *Homo sapiens* were shown to have the largest absolute and relative brain size. These facts lead to the question: Is size alone the main factor in distinguishing the human brain (Jerison 1973)? Large brains have cell bodies that are more scattered, leaving room for many more interconnections between areas (Semendeferi 2001b: 114). Or, has the internal organisation of the brain changed through mosaic evolution in *Homo sapiens sapiens*?

It appears that both factors are involved in differentiating the human brain. But the human brain is larger than would be expected for a primate with the human body size, so hominisation in brain size and the cortical mantle, particularly for the past 700,000 years (Potts 2001: 147), exceeds the primate norm in humans. Also, the expansion of association cortices has made them disproportionately large (Preuss 2000) in the human brain. The human cortex is 10 times larger than the macaque cortex and the human brain is 3–4 times larger than any ape brain (Semendeferi 2001b: 108). This large amount of increase in the human brain obviously matters, as proven by Jerison (1973) who was interested in relative brain sizes and formulated the EQ, encephalisation quotient. The EQ is the ratio of actual brain size to expected brain size. Expected brain size is based on an average for living mammals that takes body size into account (Kolb and Whishaw 2008: 41). For every unit of body size increase there is a corresponding increase of 0.75 in brain size (Martin 1996). The average mammal is the cat with an EQ of 1.0. Thus, EQ is a number reflecting the increase in brain size over and beyond that explainable by an increase in body size.

Jerison's studies led him to believe that brain size is a *natural biological statistic* that can be used to estimate other statistics such as gyrification and fissurisation, the size of other structures, neuronal density, and connectivity. The sheer size of the human brain is one of its most remarkable characteristics and must be reckoned with in understanding the human creative, cognitive, motoric, emotional, sensory, and associational palette of abilities (Gibson 2001). Moreover, as mentioned, large brains have more space for interconnections between cortical areas, and cortical-subcortical regions, giving rise to complex interactions between cortical areas and the brain stem and spinal cord.

There is one exception to the relationship between brain size and size of other neural structures and that concerns the olfactory bulb and the limbic structures to which it projects, thus constituting an expanded limbic system (Heimer et al. 2008). These structures seem to develop their own size and diversity somewhat independently of gross brain size

2 Allman (1977); Kaas et al. (1979); Stephan et al. (1981); Semendeferi (1994).

3 Jerison (1973); Aiello and Dean (1990); Changeux and Chavaillon (1996).

and are large and complex in humans. Areas 10 (appears to be uniquely large and interconnected) and 13, to be discussed subsequently, represent areas involved with social cognition and complex social interactions.

The ability to conduct scientific research is the culmination to date of the ability to detect causal sequences and is most highly developed in *H. sapiens sapiens*. Jerison believed that the ability to detect causality might be a key component of the human ability to conceive of ourselves as continuing, self-contained beings. The precuneus area of the parietal lobes, more highly developed in humans, may enable this function, which will be discussed subsequently.

What might be some of the mechanisms resulting in enhanced encephalisation? First, adult brain size depends partially on the number of neurons produced and retained during ontogeny. Kaskan and Finlay (2001) have found that different neural areas differ in the length of the embryonic period of neuronal cytogenesis (cell birth) of precursor cells. Portions of the brain that exhibit the greatest degree of enlargement such as the cortex have the longest periods of cytogenesis, and areas such as the medulla that have not enlarged that extensively have the shortest. From this it follows that heterochrony (changes in developmental timing) could account for species variations in both absolute brain size and in differential size of various structures. Kaskan and Finlay (2001) found that two major factors account for most of the variation in brain size in bats, insectivores, and primates and this implies that as few as two major genes or gene complexes may be responsible for the overall size of the brain and the differential size of the limbic system.

A second major factor leading to enhanced encephalisation could be explained by the radial unit hypothesis of the development and evolution of the neocortex as formulated by Rakic and Kornack (2001). Cortical neurons are formed from precursor cells that line the ventricles and these neurons migrate radially along glial fascicles until they reach their ultimate cortical destination. The neurons that are formed first compose the deepest layers of neocortex; those formed last the most superficial layers.

The number of columns generated determines the surface area; the number of cells incorporated in each column determines the thickness of the cortex. The neocortical surface area of the human brain is 10 times as large as that of the macaque, but the thickness of the cortex is similar. This means that the increase in size of the cortex results from the generation of increased radial columns. One factor that could account for this is an increase in the numbers of mitotic cell divisions involved in the cytogenesis of neurons prior to or during the early stages of the initial formation of radial units. In the macaque, cortical neuron precursors undergo 28 mitotic cell divisions in the ventricular zone prior to neuronal migration, but in mice the neuronal precursors only undergo 11 mitotic cycles prior to migration. Rakic and Kornack speculate that the enlargement of the human brain in comparison to the macaque brain requires approximately four extra mitotic divisions or a few extra days of mitosis prior to neuronal migration.

A third mechanism that could contribute to increased size is a decrease in rates of apoptosis (programmed death of neurons or neuronal precursor cells). These findings complement those of Kaskan and Finlay in confirming that minor changes in developmental processes, potentially controlled by only a few genes that alter cytogenesis, could account for species variations in brain size. So, although the result of these three mechanisms enables a complex development of cerebral structures, the mechanisms governing the processes of enlargement may be quite simple.

Most authors have assumed that the EQ was a better measure of intelligence than absolute brain size, but that was before recent decades of study of the cognitive capacities of nonhuman primates. Gibson et al. (2001) have found that absolute brain size, "extra" neurons, and body size all correlate strongly with a test of mental flexibility, suggesting that measures of absolute brain size are powerful estimators of advanced cognitive functions.

2.2 Structural Differences between Ape and Human Brains

For decades, studies of mammalian and primate brain evolution have been traditionally focused upon encephalisation and according to Preuss (2001) they have ignored the many differences in cortical organisation found among different species of mammals and primates. Preuss believes that the prefrontal cortex has enlarged in humans beyond what would be expected in comparison to primary sensorimotor structures, as did portions of the posterior association areas (temporal and parietal areas). Moreover, Preuss and Kaas (1999) have found that the primary visual areas (area 17 Brodmann) in humans differ from both apes and monkeys in the way they segregate information from the magnocellular and parvocellular layers of the lateral geniculate nucleus. Human beings also possess other structural and functional characteristics of higher-order visual cortical areas that distinguish them from mon-

keys. Similar studies have not been carried out with apes. Nevertheless, Preuss and Kaas (1999) suggest that humans have a higher-order capacity for evaluating moving stimuli. Helvenston and Hodgson have suggested that *Homo sapiens'* acute perception of movement (along with direct visual fibers to the amygdala) forms one of the foundations for animistic beliefs as the world's original religion (2010). Might the use of projectile weapons to kill prey and predators have required such higher-order developments dating from 700,000 to 500,000 years ago (Hodgson and Helvenston 2006)?

Related to reduction in size of visual areas, our visual striate cortex is 121% less than expected for an ape of human size and the lateral geniculate about 140% less than expected (Holloway 1996a, 1996b). Also, there is much greater variation in the volume of human and ape striate cortex than would be expected with the human striate cortex smaller than that of apes. Holloway suggests that such variation in striate cortex began with australopithecines (Holloway et al. 2001). This change in the size of striate cortex, where it is reduced and pushed to the rear by an expanding parietal lobe, represents one major example of brain reorganisation that, of course, can only be studied in the fossil record with some difficulty.

Another striking difference between humans and apes is found in the pyramidal motor system, the phylogenetically newest area of the motor cortex, and motor systems in humans are considerably more highly developed cerebrally compared to apes. For example, in humans the primary motor cortex contains a rostral (forward) section that is evolutionarily relatively recent, wherein motor neurons in the cortex directly innervate the shoulder, elbow, and finger muscles, contributing to a much more highly sophisticated and refined response system in humans. It is located in front of the central sulcus in contrast to apes where motor cortex is located on both banks of the central sulcus. Thus, the pyramidal motor system, while being more efficient and advanced than that of apes, occupies less cortical area, leaving more room for prefrontal and parietal association cortex in *H. sapiens sapiens*. This evolutionary advance is termed the pyramidal motor system because at the microscopic level the motor neurons are huge and pyramidal in shape (the Betz neurons) with extremely long axonal connections to the cortex, brain stem, and spinal cord.

This is, as mentioned, another example of cortical reorganisation with enhanced hominisation. The caudal (rear) primary motor area is mediated by corticospinal efferents from "old" cortical neurons in the extrapyramidal system (includes the caudate and putamen which together equal the striatum). The striatum is heavily interconnected with the globus pallidus and other nuclei and is known collectively as the basal ganglia, which serve very important functions in integrating emotion and reason. Unlike the motor cortex in the pyramidal system, the basal ganglia must use the integrative mechanisms of the spinal cord to generate motor neuron activity and motor output (Rathelot and Strick 2009). This pattern of anatomical connections underscores the significant evolutionary development in humans of refined, highly developed, response systems that convey information very rapidly. As Donald (1991) suggested, the response systems in humans are much more highly developed than in apes in motoric, communicative calls, language and gestural behaviours constituting one of the greatest differences between apes and humans as exemplified by the hominin and hominoid fossil and cultural record.

There may be a parallel here between the reorganisation of cortex, wherein the parietal association areas develop at the expense of the striate cortex and the reorganisation of motor cortex from chimpanzees to humans wherein motor cortex is reduced in humans but carries out some unique functions, leaving room for more frontal and prefrontal association areas.

For many years it had been rather assumed that the frontal lobes in humans were much larger than would be expected for a primate of human body size (Blinkov and Glezer 1968; and Brodmann 1912), but preliminary evidence (Semendeferi et al.1997, confirmed in Semendeferi 2001a) suggests that the frontal lobes in humans are about what frontal lobes would be in an ape of similar body weight.

Since the frontal lobes or divisions of them *may not* be larger than would be expected according to Semendeferi, researchers are examining whether the cortex is larger in the frontal lobes than would be expected, but preliminary results suggest this *may not* be so either. A number of brain illnesses are characterised by abnormalities in the structures and connections of the frontal lobes, which may be fairly recent in evolutionary time, because anterior gross brain morphology has been similar for 300,000 years (Bookstein et al. 1999), this being a period of time, when modern human cognitive capacities begin to be manifest in the endocranial and behavioural, i.e., archaeological record (Bednarik 1992).

Smaller structures in the frontal lobes such as areas 10 and 13 may be different from ape brains, based upon preliminary studies. Area 10, which is the frontal pole, is an area of brain that is important in the planning of future actions and taking the initiative and it has more room for interconnections in human than in ape brains. Also, area 10, accord-

ing to preliminary results, is larger in humans than would be expected in an ape of human body size and it is almost twice the size of Pan and Bonobo chimpanzees. Other features of area 10 are similar in human and ape brains. Semendeferi (1994) has hypothesised that area 10 first began its enlargement in the first Homo genus, i.e., *habilis*, accompanied by early signs of stone tool making.

Allman et al. (2001) have proposed that those areas of the anterior cingulate cortex (ACC) that contain von Economo neurons (VENs) are a phylogenetically new specialisation of neocortex rather than a more primitive state of cortical evolution as most other areas of the cingulate cortex are (Nimchinsky et al. 1995). As we will discuss subsequently, the anterior cingulate is an area involved with a variety of emotions, both positive and negative. Allman et al. (2002) have suggested that the VENs in the ACC project into area 10.

This relay conveys the motivation to act upon the emotional information provided by the ACC. It particularly concerns the recognition of having made an error. This leads to (except in OCD where the behaviour only feels adaptive briefly) an adaptive response to these adverse events so as to reduce error commission. According to Allman et al. (2002) this capacity is related to the development of self-control as an individual matures and gains social insight. The ACC deals with the individual's instant response to changing conditions, but area 10 is involved with the retrieval of memories from the individual's past experience and the ability to plan adaptive responses. Thus, immediate circumstances and the emotional and social valences contained within the experience are compared with longer-term memory to better enable a rational and considered decision for appropriate behavioural reaction. Allman and Watson further argue that this contributes to intelligence, which helps mature individuals such as grandparents confer food, information, affection, and wealth to the young, as is characteristic of extended human families.

Area 13, in the orbitofrontal cortex, is similar qualitatively in apes and humans but in the human there are fewer neurons and increased space for interconnections. This finding suggests that fewer neurons are necessary to complete the specific tasks, but interconnections with other brain areas, some of which may now be involved in perception, consideration, and action, have flourished. Area 13 in the orbitofrontal cortex is similar qualitatively in apes and humans, is part of the limbic system (Heimer et al. 2008) and heavily involved in emotional, motivational, and social behaviour via its interconnections with other limbic and cortical structures.

The human limbic system is larger in absolute size than that of the great apes, but not as a percentage of overall brain weight. The cingulate gyrus is a significant part of the limbic lobe and is of substantial size in humans. From anterior to posterior it subsumes viscero-motor, cognitive-effector, instant emotional experiences leading to adaptive motor responses, and sensory processing areas (Mega and Cummings 1997). A wide variety of affective responses including fear, euphoria, depression, and aggression have been elicited upon cingulate stimulation. Lesions of the anterior cingulate ACC produce frontal lobe syndrome with emotional blunting, decreased motivation, impulsivity, hypersexuality, coarseness, and many other cognitive and behavioural changes. Reduced blood flow in the cingulate cortex has been found in patients suffering from depression (Armstrong et al. 1986).

Other structures comprising an extended and in some ways unique human limbic system (Heimer et al. 2008) include the hippocampus and the amygdala, the septum, olfactory nucleus, entorhinal cortex, bed nucleus of the stria terminalis, and the nucleus basalis of Meynert. A number of the smaller limbic nuclei in primates have no counterpart in other mammals like rats and cats. Both the hippocampus and amygdala have been studied extensively in the past few decades. The hippocampus receives its input from the entorhinal cortex, which receives its inputs from the associative isocortex (the most recent cortical development, also referred to as neocortex; it has six layers as opposed to allocortex, which has three layers). The entorhinal cortex is involved in spatial orientation and is an area that is involved in the earlier stages of Alzheimer's wherein one of the first symptoms is a loss of a sense of direction. Experiments with rats indicate that this area is necessary in order for the animal to remember its body location in space, where is has come from and where it is going (Frank et al. 2000).

The entorhinal and parahippocampal neocortex are located in the inferior and medial temporal lobe and are adjacent to and richly interconnected with the hippocampus and the amygdala, which are buried within its depths. Like the hippocampus and amygdala, the entorhinal cortices are involved in memory functioning (Gloor 1990, 1992). Inferior medial temporal lobe neurons that overlie the hippocampus are involved in encoding, storage, and recall as are neurons of the hippocampus which is also involved with learning, memory, and recognition and they interact with the amygdala, which processes information about observed conspecific's emotional state.

In recent years, more research has begun studying the structure and function of the amygdala

and Heimer et al. (2008) have added a number of structures to the amygdala, terming it the "extended amygdala." The boundaries of the amygdala are complex, particularly with the now accepted neuroanatomical extension of amygdala through the subpallidal region and into the bed nucleus of the stria terminalis. In clinical practice there are few conditions that involve almost exclusively the amygdala, but one of those is Urbach-Wiethe disease, which is subject to bilateral degeneration.

On the basis of clinical observations of these patients and experiments wherein people are imaged with fMRI while viewing faces of varying emotional valence, Adolphs et al. (1994) suggested the amygdala is involved in the social appraisal of the emotional state of others, especially for negative emotions, but also is implicated in a broader spectrum of social attributions, related to value judgments such as trustworthiness, and other complex social emotions (Adolphs et al. 1999). The amygdala provides a specific emotional valence to sensory stimuli and with widespread reciprocal connections with neocortex and various subcortical structures, the amygdala can moderate the level of sensitivity of the individual to incoming environmental events, thereby attaching emotional colour to the percepts.

Further, the amygdala is linked to the emotional tone of memory consolidation and restructuring. In patients with intractable temporal lobe epilepsy electrical stimulation of areas of the temporal cortex prior to surgery results in memories so vivid they are perceived as happening now, although the subject knows they are not "real" and these memories are accompanied by the very same emotional tone that initially accompanied the original event. The amygdala has been associated with aggression in animals and with the maintenance of a given position in a social hierarchy. Associations between amygdala and frontal pathology and aggression have been shown in patients with temporal lobe epilepsy, aggression being one behaviour reliably reduced following temporal lobectomy as a treatment for intractable epilepsy (Trimble and van Elst 1999).

Finally, as Helvenston and Hodgson (2010) have shown, the amygdala is involved in the process of attributing human characteristics to nonhuman creatures, inanimate objects or acts of nature so characteristic of animistic beliefs around the world. One might say the amygdala is so finely tuned that humans can detect the emotion that animals, inanimate objects, and acts of nature are feeling at the moment.

The cerebellum is smaller in humans than would be expected in an ape of human body size and larger than expected in the gorilla (Semendeferi 2001a). It seems the cerebrum has enlarged in humans at the expense of the cerebellum, which subsumes such functions as fine motor tuning, balance, and some aspects of cognition. The dentate nucleus of the human cerebellum is larger than would be expected for an ape of human body size, which is striking given the smaller cerebellum in humans. It receives inputs from the lateral cerebellar cortex and projects to the cerebral cortex via the thalamus, the latter thus serving as a major relay station between the cerebrum and cerebellum just as it is a relay station from the brain stem and spinal cord to the cerebral cortex.

The cerebellum has been implicated in cognitive tasks that do not involve motor deficits (Leiner et al. 1995: 244). Karmiloff-Smith (1992) has suggested that the emergence of higher-order representations depends on the routinisation and consolidation of lower-order schemas; thus, interactions between the cerebrum and cerebellum could play an important role in the development of higher-order cognitive representations such as are required in learning to play an instrument, ride a bicycle, or many higher-level motor skills.

Thus, the cerebellum contributes to the routinisation of complex cognitive procedures, leading thereby to mental agility, and is involved in error detection and language. Müller et al. (1998) have described the cerebellum as performing fundamental functions such as prediction of others' actions and preparation for behavioural responses. These functions are based on cerebellar learning of sequences. The cerebellum is implicated in autism spectrum disorders (Semendeferi 2001b: 111).

The parietal and occipital cortices integrate visual and somatosensory information, but like the frontal lobes, they do not stand out as particularly larger in humans than in apes based upon body weight (Semendeferi 2001b). However, Preuss and Kaas (1999), Gannon et al. (1998), and Holloway et al. (2001) emphasise that the parietal association area is larger in humans than apes at the expense of the occipital cortices. The planum temporale located deep in the Sylvian fissure at the posterior end has long been known to present a left-right asymmetry favouring the left (Geschwind and Levitsky 1968). This asymmetry has been associated with the fact that in humans this area is critical in language reception. However, the asymmetry is also present in apes and suggests that this was the cerebral organisation of the last common ancestor before the ape-human split. In fact, Gannon et al. (1998) have shown that chimpanzees are characterised by a L > R planum temporale.

Gannon et al. (2001) followed that research up in a study that compared the planum temporale in macaques, apes, and humans. They propose that the

distinguishable suite of neurological features related to receptive language began a gradual evolution 20 million years ago, resulting in the appearance of an asymmetrical gross anatomy for the planum temporale 10 million years later in the common ancestor of gorillas and humans. The identification of this structure as a common great ape-human structure challenges the idea of a simple relationship between the asymmetry in this part of the brain and language. It is far more likely that these areas are homologous in apes and humans, with special modifications and reorganisation developing in humans that enabled the expression and reception of oral and written language. For example, when the homologue of Broca's area in the ventral premotor region is stimulated in nonhuman primates like the owl monkey it produces oral and laryngeal movements (Stepniewska et al. 1993).

In the parietal lobe on the mesial surface of the cerebral hemispheres is an area known as the precuneus, which integrates body space, self-reflection, environmental space, and visceral sensations from the nearby parietal operculum and insula. This area of the brain is active when the brain is idling in moments of self-reflection. Furthermore, with diminished activity in the precuneus there is a diminished state of self-awareness, such as is seen in dementia, sleep, or when focusing upon other stimuli (Cavanna and Trimble 2006). These authors conclude the precuneus subserves higher-order behaviour related to conscious self-percept. This area certainly seems prerequisite for the experience of that "continuing, self-contained being" of Jerison.

The temporal lobes are well developed in *Homo sapiens* (Semendeferi and Damasio 2000) and with a larger sample size may be relatively larger in humans than in apes. Preliminary results did not reach statistical significance but suggest that the insula (deep in the anterior Sylvian fissure of the temporal lobe) is also large in humans and may be specialised for such functions as the processing of autonomic functions, internal stimuli, olfactory stimuli and tastes, and constitutes part of the extended limbic system (Heimer et al. 2008). In the basal forebrain there is a nucleus subputaminalis (NSP) that is part of the cholinergic system. This area is unique in humans and the NSP provides cholinergic innervation to the inferior frontal gyrus where Broca's area (speech production) is located. Thus, Broca's area and associated structures may have some unique features in humans not found in the great apes because of NSP.

Located in the posterior temporal lobe is Wernicke's area, highly specialised in humans for the reception of sounds, especially language comprehension. This area of cortex is 6-layered, as opposed to the 3-layered allocortex (hippocampus and olfactory cortex), but in humans there are fewer cells and more interconnections between columns of cells (Buxhoeveden et al. 1996). These authors conclude that in humans, evolution has favoured the integrative functions of layers II and III rather than the receptive layer IV, or corticofugal layers V and VI. Thus, there is human specialisation for integrative language reception and comprehension. This area is frequently damaged by stroke (Helvenston and Bednarik 2011).

There are two thalamic nuclei that appear unique in humans. One is the anterior principal nucleus (AP) and the other is the mediodorsal (MD) nucleus (Armstrong 1980, 1991). The AP is richly connected with the cingulate gyrus, prefrontal, parietal, and inferior parietal cortices, the hippocampal region, the subiculum, and the mammilary bodies. It has been suggested that the AP is active in the encoding of increasing bits of information and sustaining attention to sensory stimuli.

The MD nucleus is larger than would be expected for a primate of human body size and it is heavily connected with the dorsolateral prefrontal cortex. The dorsolateral prefrontal cortex is the last (45th) to myelinate during human development and is believed to be involved with short-term working memory helping to keep the subject focused on the task at hand. Phylogenetically older fibers tend to myelinate earlier than newer fibers during ontogenesis, suggesting the medial dorsal nucleus and the dorsolateral prefrontal cortex may be uniquely specialised in humans.

There are a variety of other microscopic features that are unique in the human brain (Semendeferi 2001b) such as structures in the extended limbic system involving neural circuits that regulate emotions and complex social behaviour (Heimer et al. 2008). Another of these cytoarchitectonic specialty areas in humans involves layers of primary visual cortex V1 or area 17 of Brodmann. This well-documented difference in cortical histology of macaques and humans involves cortical layer IV which in primates stains densely for the metabolic enzyme cytochrome oxidase (CO). In macaques and many other Old and New World monkeys CO staining occurs in two bands, IVA and B. Humans are different. They possess a similar layer IVA, but it does not stain densely for CO. This area is a terminal for afferent fibers projecting to it from the thalamus. The fact that this area does not stain heavily with CO, which indicates a high level of metabolic activity, suggests that layer IV is more active in the macaque than the human, and that in the human this

layer has become either more efficient or other areas (II and III) have reorganised and are subsuming some of the functions of layer IVA, most likely via cortical to cortical fiber projections.

There are several other such examples but we do not know much about their functional importance. Since scientists have only just begun to study the comparative differences in cytoarchitectonics (organisation and staining characteristics of neurons and appendages) in various primate species undoubtedly more examples will come to light in future studies.

Von Economo neurons have huge cell bodies but only one apical axon and one dendritic appendage. They appear to participate in very rapid signal transmissions and are found in two very restricted regions in the brains of hominins: the anterior cingulate cortex (Allman et al. 2002; Hayashi 2006) and the frontoinsular cortex (Sridharan et al. 2008). VENs are relatively newly evolved, being present in the great apes and humans. They are also found in sperm whales, bottlenose dolphins, Risso's dolphin, and beluga whales, and in the brains of African and Asian elephants.[4] This distribution suggests VENs may be restricted to large animals with large brains and extensive social networks and have been especially evolved to rapidly transfer information to various areas of the brain and spinal cord (Fajardo et al. 2008).

In schizophrenia and bipolar depression the anterior cingulate cortex is affected with reductions in the density of layer 2 (Benes et al. 2001). In Alzheimer's disease (Nimchinsky et al. 1999) the cingulate cortex is reduced and the anterior cingulate cortex is reduced in both size and metabolic activity in autistic patients (Haznedar et al. 2000). Both the size and activity of the ventral part of the anterior cingulate cortex are reduced in depressed patients (Drevets et al. 1997).

As mentioned, the frontoinsular cortex has a number of VENs and this area seems to be critical, especially the right side, in switching between distinct brain networks across various tasks and stimulus modalities (Sridharan et al. 2008). These authors believe that their findings have important implications for a unified view of network mechanisms that underlie both exogenous and endogenous cognitive control, certainly a high-level cognitive capacity.

Other deep telencephalic nuclei involve subcortical structures in the basal ganglia that may have undergone some reorganisation over hominoid evolution that are involved in emotion, movement, and complex behaviour.

4 Coghlan (2006); Hof and Van der Gucht (2007); Butti et al. (2009); Hakeem et al. (2009); Seeley et al. (2006).

3 Human Susceptibility to Neurodegenerative Diseases

This raises the question, are more recently evolved or reorganised brain structures that may characterise *Homo sapiens* more subject to brain illnesses? To assist us in parceling out the factors that would help us answer this question we will consider the common causes of death for hominins, hominoids, and *H. sapiens*. Humans have unquestionably the longest lifespan of any primate. Infections cause most of the mortality in wild chimpanzees and in traditional forager-farmers. Even under the conditions of high mortality experienced by human hunter-foragers, the human life expectancy at birth is twice that of wild chimpanzees. According to tooth wear, early *H. sapiens sapiens* and the best known of their robust predecessors, *H. sapiens neanderthalensis*, had a larger proportion of older adults than prior Homo species and australopithecines (Caspari and Lee 2006).

Turning to historical times, the demographic data from Sweden from 1751 and 20th-century hunter-foragers will be compared. Both lived under unhygienic conditions with high burdens of infection and limited access to effective medicine. Both the Swedish and 20th-century hunter-foragers had high mortality rates at early ages of 10–30%, which restricted their life expectancy to 30–40 years. Despite low survival rates, half of those reaching age 20 lived to be 60 years of age (Finch 2010). The greater survival to later ages allowed the evolution of stable multigenerational support of the young, which is a uniquely human trait among primates. One wonders if the expansive development of the human limbic system, which is so involved with social and emotional aspects of life, might not have resulted in the grandparents being prominently involved with infants and young children, thus leading to higher survival rates of the family or small groups of families (Hawkes 2004; Gurven and Kaplan 2007).

Mortality across life spans forms a J-shaped curve in nearly all mammalian populations: the high early age mortality declines to a minimum (q_{min}) at the approach of adulthood. This is followed at midlife by an exponential acceleration of mortality associated with higher levels of chronic degenerative disease and dysfunction that collectively define senescence (Finch 2007; Finch et al. 1990). Humans differ from apes by their lower mortality in juvenile and adult ages and by the later onset of mortality rate acceleration.[5]

In healthy populations of humans and lab animals, the acceleration of mortality is preceded by

5 Kaplan et al. (2000); Hawkes et al. (2009); Hill et al. (2001).

increasing morbidity from chronic degenerative diseases (Finch 2007; Finch et al. 1990). For wild chimpanzees, typical early mortality rates are 20% per year in infancy, within the range of hunter-foragers, then decreasing to a q_{min} of about 3.5% per year in preadult ages. The chimpanzee life expectancy at birth is about 13 years, whereas those reaching adulthood (age 15) have about 15 more years of life expectancy (Kaplan et al. 2000; Hill et al. 2001). Very few have survived beyond age 50, even in captivity with modern veterinary care (Rosen 2008). One exception was the chimpanzee Cheetah, from the old Tarzan films who died December 28, 2011 at age 80 in a chimpanzee sanctuary. In contrast, human mortality after the early years is much less, with a >2-fold longer life expectancy and >3-fold lower q_{min}, even with lower access to medicine.

The two major factors leading to longer human life expectancy are delayed mortality rate acceleration and a lower q_{min}. According to Finch et al. (1990) the human q_{min} merits further attention in human evolution. Even in populations with high infectious burdens and neonatal mortality, the human q_{min} is >50% lower than in wild chimpanzees. Since 1800, the industrialised countries have further lowered this figure by 26-fold. This is likely to be a result of comprehensive and early vaccinations of human infants and children in the industrialised countries. Since 1800, the life expectancy in developed nations rose progressively to >70 years and recently the life span has increased to >90 for a substantial minority of the population. In contrast to chimpanzees a definitive proportion of elderly forager-farmers aged 60 or older die from nonspecific senescent causes whereas chimps tend to die more frequently from infections (Finch 2010), which humans are eliminating in themselves in industrialised countries with relentless determination.

But the reduction in the q_{min} and the increased longevity of humans allows for processes of neoteny to continue and further lower the q_{min} thus prolonging the human life span even further. The neotenisation of the human brain and the low q_{min} allow for developmental mechanisms to unfold that confer an advantage (from a subjective point of view and in terms of adaptations such as grandparents assisting grandchildren to accumulate wealth) on those who do not seem to age significantly until their 80s and even remain sprightly into their 90s. The question is, do these newly evolving mechanisms also enable more degenerative diseases of aging, and more brain illnesses of newly developed brain areas or neurons in humans? There is some evidence for this speculation as von Economo neurons (VENs), or spindle cells, are uniquely numerous in humans, phylogenetically recent and they are severely affected in the degenerative process of Alzheimer's disease and frontotemporal dementia as well as in illnesses wherein the anterior cingulate cortex and the dorsolateral prefrontal cortex are involved. This suggests that some of the differential neuronal susceptibility that occurs in the human brain in the course of age-related dementing illnesses may have appeared only recently during primate evolution. On the other hand, atherosclerosis, one disease of aging contributing to strokes is known to occur in captive chimpanzee populations, but rarely. One thing seems certain, the majority of brain illnesses appear to be recent phylogenetically, maybe only 200 years or so for schizophrenia and >2000 years for bipolar disorder.

On the other hand, many brain illnesses are clearly not old-age derived. Schizophrenia, bipolar disease, and obsessive compulsive disorder tend to set in relatively early, and even Parkinson's, Alzheimer's, and especially frontotemporal dementia and Huntington's are not limited to the old. Autism and Asperger's are typically manifested in early childhood, as are Rett and Down syndromes and dozens of other known genetic impairments endemic to humans. Of particular relevance are two observations. The most debilitating or prominent brain illnesses, such as schizophrenia, bipolar disease, autism, multiple sclerosis, and so on are basically absent in the great apes. Therefore, it can be assumed that these primates do not share our genetic predisposition in that respect, and that it appeared after the Miocene separation of our respective phylogenies.

This introduces the second issue: At what point in our genetic past should we expect to find these neurodegenerative features appearing first? Our analysis confirms the view that at some stage in the evolution of our species, the neostriatum, orbital frontal, lateral prefrontal, and cingulate cortex, basal ganglia and thalamus became susceptible to specific pathologies, be it through demyelination, VENs, or any other factor. VENs (Nimchinsky et al. 1999; Watson et al. 2006), we noted, are shared by humans and apes (and other species), but they are larger and far more numerous in the human anterior cingulate cortex and the frontoinsular cortex. These structures are thought to be involved in complex social cognition, rapid information transfer, and the rapid assessment of complex situations. There are several other significant differences between the brains of humans and apes, for instance, nerve cells in humans are arranged in far more complex patterns than in apes (Preuss and Coleman 2002). The minicolumn in the left planum temporale (basic information processing and language production)

is significantly enlarged in the human, relative to the chimpanzee or rhesus monkey (Buxhoeveden and Casanova 2002). The human brain produces about six times as much thrombospondin as that of the chimpanzee or macaque (Cáceres et al. 2007), which is a protein secreted by astrocytes (Ullian et al. 2001, 2004; Barres 2008) triggering synapse formation (Christopherson et al. 2005). The human brain also produces about twice as much of THBS4 and THBS2 messenger RNA (mRNA) respectively – ribonucleic acids that, like DNA, can carry genetic information. Increased expression of thrombospondins in human brain evolution could result in changes in synaptic organisation and plasticity, and contribute to the distinctive flexible cognitive abilities of humans, as well as to the vulnerability to neurodegenerative disease that seems unique to humans (Walker and Cork 1999; Olson and Varki 2003). It is noteworthy that Damasio et al. (1990) provisionally implicated the ventromedial or orbital prefrontal cortex of humans in both our rapid cognitive evolution and the attendant pathologies.

4 Evolutionary Perspective

Evolutionary theory attributes evolutionary change essentially to two factors, natural selection and sexual selection. In the first, specific phenotypes representing aspects of morphology or behaviour are preferentially reproduced across generations of a given population. In the second, phenotypes become overrepresented either through mate choice or intra-sexual competition. Developmental systems theory challenges the overly restrictive focus on the genes with a model of interacting systems (Oyama 2000, 2001), emphasising nongenetic inheritance of traits and the cybernetic feedback from organism-environment systems changing over time. Niche construction has been presented as another major force of evolution (Odling-Smee et al. 2003), operating similar to natural selection. Organisms engaged in it modify the evolutionary pressures acting on them, and humans are seen as the "ultimate niche constructors" in which their complex cultures play an important role. Laland et al. (2000) see much of niche construction as guided by socially learned knowledge and cultural inheritance (cf. Silk 2007).

Other dimensions of evolution are termed epigenetic, behavioural, and symbolic inheritance systems (Jablonka and Lamb 2005). All organisms are said to be subject to epigenetic inheritance, which refers to physiological/biological processes above the level of DNA. Behavioural inheritance is found in most species, and defines the transference of information or behaviour through learning rather than genetically. Thus evolution is not a simple genetic process relying on the appearance of mutations (Dobzhansky 1962: 18; 1972). The notion of a progressive moderation of human evolution by culture is the central plank of the gene-culture co-evolutionary model (Boyd and Richerson 2005; Richerson and Boyd 2005). Fuentes (2009) has sought to reconcile the pronounced duality of evolutionary biology and sociocultural anthropology, pointing out that symbolic and other cultural processes influence behaviour and potentially physiological and even genetic factors. His demand that behavioural plasticity has a specific role in human behaviour runs again counter to neo-Darwinism, but it seems impossible to explain hominin development, especially of the Late Pleistocene, without that factor.

The most distinctive and revolutionary model to challenge the orthodox archaeological notion of a complete replacement of all robust hominins by a "new species" arising in the Late Pleistocene (known as the "African Eve hypothesis") is the domestication theory (Bednarik 2008a, 2008b). According to it, most eligible regions of three (and eventually four) continents were occupied by people during that period, and no mass migration, genocide, or other form of complete replacement occurred. The somatic changes from robust to gracile types took some tens of thousands of years, as did the change from Mode 3 to Mode 4 technologies (Bednarik 2007). The dramatic physical and genetic changes occurring in a near-global population of humans within such a relatively short time were primarily driven by *culturally moderated selection*.

In all sexually reproducing species, all characteristics of individuals are said to be inherited through genes. It does not necessarily follow that all inheritance must be encoded in DNA. The principles and mechanisms of genetics apply to the molecular structure of cells and tissues, the development of individuals and the evolution of whole populations. Selective breeding defies natural evolution in the sense that it can rapidly change the characteristics of a population without any natural selection in the Darwinian sense. The process of selecting genetic traits by means other than Darwinian evolution is domestication, a radical hereditary reorganisation of the genetic constitution of a species. In the case of robust *Homo sapiens*, it significantly accelerated the fetalisation of the species that had already begun earlier, promoting behavioural and other plasticity.

In all animals, including all hominins, reproductive success determines phylogenetic direction. It is obvious that today the processes of natural evolution are largely suspended in our species' development,

having been widely replaced by cultural mating imperatives. Deliberate breeding mate choice determines procreational success today, so at what point in time did it first appear? Other primates (indeed, all other animals) exhibit no preferences in mate selection of youth or specific body ratios, facial features, skin tone, or hair; yet in present humans these are deeply entrenched, perhaps "hardwired." Facial symmetry, seen to imply high immunocompetence (Grammer and Thornhill 1994; Shackelford and Larsen 1997), is preferred, and in female humans neotenous facial features are strongly favoured by males (Jones 1995, 1996). The most rational explanation for our rapid and universal fetalisation during the final third of the Late Pleistocene is that cultural practice had become such a determining force in human society that breeding mate selection became increasingly moderated by cultural factors, i.e., by factors attributable to learned behaviour. These could have included the application of a variety of cultural constructs in such choices, such as social standing, communication skills, body decoration (which becomes notably prominent 40 ka ago), and most especially culturally negotiated constructs of physical attractiveness.

Mating preferences and their genetic results in respect of personality and anatomical traits (Laland 1994), which could become cultural selection variables, can be modeled by methods of the gene-culture co-evolutionary model.[6] Some of the traits selected for can be detected in the recent palaeoanthropological record, which shows a distinctive trend towards gracility, beginning in the females, with the males lagging many millennia behind (Bednarik 2008a, 2011). This trend is still accelerating today, as evidenced by the perpetual adolescence of our popular entertainment stars, both female and male, and anorexic fashion models with hair bleached the white or pale blonde of infants. Generational mating site distance (Harpending et al. 1998) and reticulate introgression or introgressive hybridization (Anderson 1949) easily account for genetically observed changes in human populations over the past 40 ka, rendering it unnecessary to resort to mass movements in explaining them.

Domestication is the collective genetic alteration of physiology, behavior, or life cycle through selective breeding. In general, the term has been used for such alterations caused, deliberately or not, by humans, but there are numerous examples of domestication by other species. Many animal species, vertebrate and invertebrate, have domesticated others, for instance, to modify foods indigestible by the domesticators, or for their labour or simply to serve as a staple food source. Domestication demonstrates that the continuous selection of a single trait does not necessarily evolve a population of better-adapted organisms, as natural selection would be expected to yield. Rather, it shows that selection for a single trait results in changes in numerous traits, changes that are usually deleterious, be they physiological or a range of others. For instance, domestication of mammals typically results in decreased cranial volume relative to body size, a decrease that in some species (e.g., pigs) can be as much as 30–40%. As in recent humans, it leads to the abolition of estrus. It also decreases robusticity and increases susceptibility to detrimental changes, including neurological. (For example, a prion degenerative disease of the brain, bovine spongiform encephalopathy ["mad cow disease"], has decimated large populations of domesticated cattle.) All these changes are possible because the domesticators eliminate natural selection through their guardianship, but the patronage for the human domesticates is provided by their culture and technology; they need no protector.

This perhaps offers a clue to the initial timing of these developments. In a self-domesticating species, deleterious changes can only establish themselves if certain processes of natural selection are suspended, which presumably requires a specific level of cultural and technological development. Although human culture developed gradually over a period of more than two million years, it was apparently only in the final Pleistocene that tangible evidence of its genetic consequences can be readily detected, in the rapid gracilisation of our ancestry. If the rise of neurodegenerative diseases is attributable to the same factor, a diminishment of Darwinian evolution's ability to select against significantly unfavourable mutations – as we propose – then they too may have established themselves relatively recently. But how recently?

In their most consequential forms, these diseases can also only have become widespread after cultural imperatives had developed to the level of being able to provide the existential shelter, except those affecting only the old. Once individuals have already contributed to the gene pool, their loss from it no longer influences phylogenetic direction. But this is very different with those affecting younger people, and especially the very young. For most of hominin evolution these disadvantageous genetic predispositions would have been vigorously selected against. This includes not only the neurodegenerative diseases we have discussed, but also thousands of other genetic disorders humans are now subjected to (e.g., Mendelian disorders; Bednarik 2011).

6 Cavalli-Sforza and Feldman (1973); Feldman and Cavalli-Sforza (1989); Aoki and Feldman (1991); Durham (1991).

5 Conclusion

The hominisation of the human brain is a complex process but one rarely studied until recently. This series of transformations that human brains have undergone since the assumed human-chimpanzee split to produce the individuals of today has been largely ignored. There are technical hurdles that make the study of hominisation of the brain difficult but surely not impossible (Preuss 2000). Preuss even refers to *Homo sapiens* as "the undiscovered primate" (2000: 1219). One outstanding exception is the work of Allman and associates whose work we have considered throughout this article, especially their research on spindle or von Economo neurons. The evolutionary study of the human brain depends upon research into the fossil record and the study of the brains of extant humans, apes, and other anthropoids. (Due to the endangered status of most primates it is difficult to carry out postmortem studies of their brains, and imaging studies are difficult to accomplish on them.) For many years, the evolutionary approach focused upon the commonalities in mammalian and primate brains. The question of what makes the human brain different from the chimpanzee or other ape brain has rarely been asked. Yet, this is one of the most interesting questions regarding the evolution of the brain. It is only more recently with the various imaging techniques such as MRI, fMRI, and PET scans that researchers have begun to tease out the attributes of the human brain that distinguish and differentiate it from the brains of the great apes. Surprisingly little is known about this latter field of study, but we discussed the current state of knowledge as it points to the fact that the size and interconnectivity of the human brain (Semendeferi 2001b: 107–120), which confers *H. sapiens'* superior cognitive abilities, also appears to give rise to certain brain diseases, such as schizophrenia, bipolar illness, obsessive compulsive disorder, multiple sclerosis, autism, dementia's including Alzheimer's, most of which are only known in humans. Thus, with the expansion of the human frontal cortex, temporal and parietal lobes, limbic system, and basal ganglia, hominins have also developed brain illnesses that predominantly affect these same areas. Understanding how the human brain organisation differs from other species is essential to understanding human cognitive and behavioural specialisations, because significantly other extant primates are largely free of these illnesses. We have highlighted the areas of the brain involved in these illnesses.

We then presented a discussion of how and why natural selection has permitted the development of these illnesses in *Homo sapiens sapiens*. In this we noted that somatic "modern" features of humans contradict the canons of evolution. We are more susceptible to injury, physically weaker, and have a smaller brain than our robust immediate ancestors. These changes characterising hominins of the final third of the Late Pleistocene and the Holocene define us as a neotenous adaptation, which was introduced through the rise of culture as the determinant of breeding patterns, through mate choice. Thus, evolutionary processes were supplanted by cultural selection. This very same mechanism is capable of explaining the establishment of neurodegenerative and other genetic pathologies in the course of the last 40 ka. The enhanced connectivity that led to man's most brilliant achievements has apparently led to brain diseases that are relatively common, but it also needs to be explained why they were not selected against. In Darwinian evolution deleterious factors should be eliminated from a species' genome, but that mechanism can be weakened or even replaced by cultural selection; i.e., domestication. We posit that there has to be a distinctive reason for our susceptibility to such pathologies, and on the basis of the current, very limited evidence, our explanation is the most economical and robust.

We have thus sought to address the question why natural selection has allowed so many deleterious traits to arise in *Homo*. If our reasoning is correct, these developments are all relatively recent, and limited to *H. sapiens sapiens*. Some of them may even be outcomes of the last few millennia, such as schizophrenia and bipolar illness.

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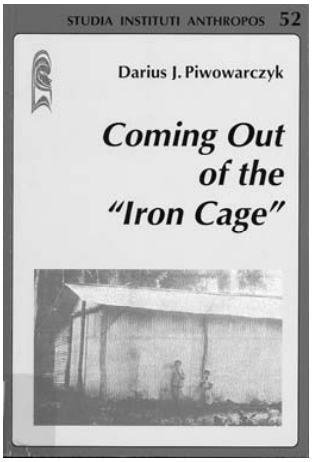

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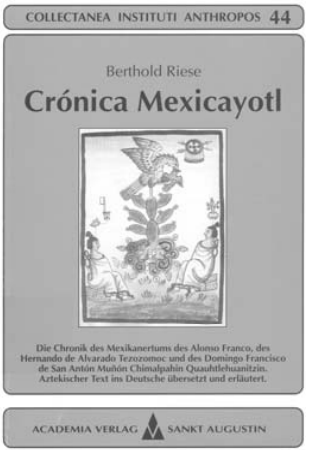

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