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The Impact of 'Anthropotechnology' on Human Evolution

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Abstract

From the time that they diverged from their common ancestor, chimpanzees and humans have had a very different evolutionary path. It seems obvious that the appearance of culture and technology has increasingly alienated humans from the path of natural selection that has informed chimpanzee evolution. According to philosopher Peter Sloterdijk any type of technology is bound to have genetic effects. But to what extent do genomic comparisons provide evidence for such an impact of 'anthropotechnology' on our biological evolution?

Keywords: philosophical anthropology, (anthropo-) technology, comparative genomics, epigenetics, human evolution

Introduction

Why did humans turn out so different from the animal with whom they share a common ancestor, the chimpanzee? Ever since Darwin many have sought an explanation for our divergent anatomy, behaviour, and state of consciousness within a framework of evolutionary thinking. To understand human evolution one must, besides biological selection pressures, also take relatively new socio-cultural and technological factors into account. But how do all these factors tie in together? The

tradition of philosophical anthropology seeks answers to this question by combining findings and formulating theories based on evidence from evolutionary biology and etiology, phenomenology, cultural and structural anthropology, paleontology, archeology, linguistics, psychology and philosophy. All these disciplines seek to explain certain elements of the human (extended) phenotype[1], and to understand the path by which such a novel way of being in the world could come to exist.

In his books '*Nicht gerettet: Versuche nach Heidegger*' (2001) and the '*Sphären*' ('spheres') trilogy (originally published in 1998, 1999, and 2004) philosopher Peter Sloterdijk has gathered together ideas from several contributors to the tradition of philosophical anthropology to embark on a '*fantastic reconstruction'* (Sloterdijk 2001, p.169, my translation) of our coming to be. A 'sphere' stands for the social, cultural and technological environment that humans build for themselves, and that isolates them from the natural environment that is typical for wild animals like the chimpanzee. Buffered from the demands of nature, humans become ever more tuned to the demands of their artificial environments, and become ever less likely to be able to survive and thrive in natural circumstances. How to understand this process of change? How to reconstruct this evolutionary history? From theories handpicked and combined from many disciplines and many thinkers, Sloterdijk converges on four synergistic mechanisms that, in combination, are responsible for the divergent evolution of humans: group-internal criteria for selection, the unburdening of the body through technology, a tendency towards 'neoteny' (a delayed and prolonged juvenile stage), and 'transference', the (re-)generation of self-created 'spheres' in space and time. None of these mechanisms works independently, and combined they form what he calls the 'anthropogenetic mechanism' - the mechanism responsible for having 'produced' modern humans.

For Sloterdijk the central role in human evolution is for technology. For him, all technology is spherical '*Brutkast*/hothouse' technology[2], and *ipso facto* all technology, in an indirect way, is gene technology (Sloterdijk 2001, p.197), or genetic manipulation. It is the sum of all these techniques that he calls 'anthropotechnology', a term that embraces both 'hard' technologies (like the 'lithotechnology' of stone tools) and symbolic or 'soft' technologies (like language, or cultural rules of behaviour). It is anthropotechnology that has produced the modern human being.

The human being is a being that is profoundly technological, unnatural, and meta-biological, but it is still biological as well. Will we find evidence for Sloterdijk's anthropogenetic mechanisms (even) in our DNA? Inclusion of evidence from molecular biology and genetics to understand human nature is rather new tothe tradition of philosophical anthropology. But today the new field of comparative genomics, where the full sequences of DNA of different organisms are confronted with each other in order to find differences and similarities present in entire genomes, gives access to new relevant evidence that is worth exploring, and new research fields are starting to blossom, like molecular anthropology and bioarcheology. This paper represents a similar approach to investigate the fit of new, empirical, genomic data in the, until very recently, genomically uninformed theoretical framework of philosophical anthropology, or rather, an attempt to test this approach, using the theoretical framework of one of its current representatives, Peter Sloterdijk. Besides using the genomic data to inform the humanities, existing theories such as Sloterdijk's can also inform biological sciences like genomics by helping to direct the gaze to retrieve the sort of information from genomic data that can help our understanding of our evolution.

The approach in this paper will be to first elaborate on the four synergistic, anthropogenic mechanisms that Sloterdijk proposes. Next, the outcome of the genomic comparison of humans and chimpanzees will be discussed. What salient phenotypic differences correspond to underlying

genetic differences? In the discussion the implications of these findings for Sloterdijk's theory will be evaluated. Does the genomic evidence corroborate the theory that an anthropogenic mechanism is (co-)shaping the human species? To what extent? Independent of the answer to this question, another question will need to be addressed: how are we to understand the 'freedom' and the 'evolutionary plasticity' of humans that is central to Sloterdijk's theory? For so much is evident: humans are a special case in evolutionary history.

New mechanisms for human evolution? Four synergistic mechanisms

In his search for anthropogenetic mechanisms for human evolution Sloterdijk was not interested in explaining the difference in the body or behaviour of humans compared to the other primates , but rather the different way of 'being in the world' of the human being. His work is an effort to get an improved grasp on Heidegger's notion of '*Lichtung*' (Heidegger 2002), the 'world' humans live in.[3] Sloterdijk goes on a journey to retrace the evolution, or rather, the 'production', of what Heidegger wrongly takes as a given and a starting point: the human being. How did the original ape come to be human? When, how, why, where and to whom could the '*Lichtung*' appear? It is in search of an answer to this question that Sloterdijk sets out to describe anthropogenetic mechanisms. Unlike Heidegger, Sloterdijk emphasizes the bodily aspect of what it means to be human (Verbeek 2008). But it is not the human being as a biological species that he seeks to understand. It might, therefore, be considered a bit farfetched to confront Sloterdijk's theory with genomic and other biological data. Still, it is worthwhile to proceed with the chosen approach and answer the question whether the evolution of the biological human is profoundly influenced by the aforementioned anthropogenetic mechanisms, and if so, how human biology is affected by them.

None of the four synergetic mechanisms work independently, and combined they have 'produced' the human being of today (Sloterdijk 2001). The first mechanism of 'group-internal criteria' explains why humans (and some other animals)[4] are not entirely under the influence of natural selection pressures anymore. According to Sloterdijk, the truly typical traits of humans are '*non-adaptive'* (Sloterdijk 2001, p.177). They do not make humans more fitted to survive and reproduce in a natural environment, but are attuned to the social, cultural and technological environment that humans create for themselves, the 'sphere' or 'hothouse' in which they live. This insulation from natural selection pressures does not result in a regular evolutionary advantage, but rather in a '*tolerance of the human lifestyle for variation'* (Sloterdijk 2001, note on p.195, my translation), which may be understood as the creation of an 'opening' for humans to explore more of their 'plasticity' than a natural environment would allow for. Many of the specifically human phenomena would not of necessity embody an adaptive evolutionary advantage. In another note (on page 195, unfortunately without reference to a source), Sloterdijk reports that in humans a lot of variation on a molecular (DNA) level, 84%, is 'selection neutral' variation, and that only 16% is adaptive in the natural sense and/or in a 'race- and ethnic sense'; a mixed adaptation to the geographical environment and to local culture.[5] However, the genomes of other organisms also house a lot of neutral genetic variation (Orr 2009); this is not in itself a source for increased adaptive freedom and 'plasticity'. The difference between humans and animals would lie in the fact that the only selective process that creates pressures on non-neutral variation in the other organisms is the process of natural selection. A question that arises here is: if humans adapt not only to natural, but also to techno-cultural circumstances and if this would mean that adaptation is not (just) 'survival of the fittest' in the regular Darwinian sense any more, then what (or who) gets selected, and how? Sloterdijk mentions that the 'fittest' in the 'sphere' are not those that prove most capable at confronting any given hardships in the 'sphere', but those who are the '*luckiest'* (Sloterdijk 2001, p.187, my translation) in being able to make use of the 'climate' in the 'sphere', and who cash in on the opportunities that the 'sphere' provides

internally. Adaptation becomes more and more ‘hothouse relative’, resulting in qualities that take the human further out of his natural environment. So do these ‘luckiest’ create more offspring than the ‘less lucky’ humans? Are these (cultural) adaptations preserved in this (old-fashioned) way? In the natural world, only genetic information can be passed from one generation to the next. Humans have more ways of information transfer at their disposal. Through teaching, imitation and technology (writing for example) that which provides an advantage can be copied. This is especially the case with adaptive behaviour, but also works through copying the devices that support the superiority of the ‘luckiest’ during one’s lifetime. If an (artificially supported) phenotypic capacity gives rise to the sort of flexible response that amounts to a cultural adaptation, then in recurrent circumstances genotypes that support phenotypes that can make further use of opportunities in (ever more) artificial environments will also start to fall under ‘old-fashioned’ selection pressures. In this process of co-evolution, ‘sphere’-related trait variants *can* also start spreading through the population genetically. For Sloterdijk, this explains why our physical appearance (think of our skin, our hands, our face, our brain, and the womanly form, to name a few examples) and behaviour have come to differ so much from that of our closest animal relation, the chimpanzee. There is a Lamarckian, or rather, Baldwinian touch to Sloterdijk’s train of thought that is controversial, and that I will address later on. But importantly, what Sloterdijk also tries to say is that the biased transmission of certain gene variants is not the only way to evolve in techno-cultural ‘spheres’. ‘Fitness’ is not only about the number of genetic offspring any more.

The second anthropogenetic mechanism is that of the ‘unburdening’ of the body.[6] Sloterdijk places the start of this development at the time where humans started to make use of stones as tools, a defining moment where humans started taking matters into their own hands. Hands were shaped by this early ‘paw-work’. But not only our hands adapted to tool use; also new and more

complex ways of thinking were jumpstarted by the first putting to use of stones as projectiles to stave off predators and competitors around 5 million years ago, at the beginning of our hominid line (Young 2003).[7] The '*Homo technologicus'* appears. The subsequent exploration of new possibilities of transfer of the burden of many types of hard work to tools, and in a later stage to machines and symbols, has allowed for a 'drift towards '*luxuriation*'' (Sloterdijk 2001, p.188, my translation). The human being is the '*Luxus*-animal', defined not by hardships, but by comforts. In biology, the concept of 'drift' refers to chance (non-selected) changes in frequencies of gene variants that may turn out neutral, beneficial, or detrimental to reproductive success. Where in the other primates many bodily traits and behaviours similar to ours are shaped and 'kept in check' by the selection pressures of their natural environment, in humans some of these traits are 'liberated' and might drift towards disappearance[8] or redeployment through change in a new adaptive sense. 'Luxuriation' is therefore tied to the opening up of a space of variation or 'evolutionary plasticity', which under the influence of group-internal criteria evolved in the direction of esthetic and cognitive traits. This is reflected, for example, in the fast development of the relatively large human brain. Here, according to Sloterdijk, the brain starts a 'luxuration' in which it almost seems to be hurrying ahead, mysteriously building a potential for achieving matters that are far beyond of what is asked of it at that time.[9] Currently, there are differen hypotheses as to why humans have developed such a big brain, which cite coping with social complexity in large groups, the planning of manual actions, sexual selection, or memetic selection as the trigger for this development.[10] For Sloterdijk, all these elements could play a role, and interactively so, as social relations, sexual preferences, tool production and tool use, and the symbolic power of language all represent 'soft' and 'hard' anthropotechnologies at work to produce human beings in the 'anthropotope' or 'sphere'.

Third, Sloterdijk describes a process that has resulted in 'neoteny'.[11] The increase of brain and skull volume (due to the abovementioned opened up possibility of 'luxuriation') causes the human child to be born in a premature state, before the skull becomes too big and too little pliant to move through the birth canal without damaging the mother. The necessary mother-child 'symbiosis' after birth is supported by the 'group hothouse' that functions as an '*external uterus*' (Sloterdijk 2001, p.190, my translation). The tendencies for cerebral growth and early birth got locked in a circular causation pattern, resulting in babies that, when compared on a level of development scale to our cousin the chimpanzee, are born a year too soon. Also, human beings reach adulthood only quite late, giving them, in general, a 'fetal image', which is likely to depend on changed endocrinological (hormonal) and chronobiological (timing) mechanisms (of which we would expect some genetic evidence in a genome-wide comparison with the chimpanzee). Associated developments may have been the loss of body hair, the development of the thin human skin, development of the human eye, and of course the further development of the human brain and its receptivity to learning, and the development of speech. Sloterdijk calls the newly developed organs the '*organs of the 'Lichtung"* (Sloterdijk 2001, p.195, my translation), by which he means to say that they have more than just biological functions: they are meta-biological. They turn humans into beings that can relate to the world in a fashion 'beyond biology'.

The vulnerable state of the dependent human infant (and also of the adolescent and adult human that remain to a large extent dependent upon their 'sphere') creates the necessity of a continuous, unrelenting upkeep of the created 'hothouse' or 'sphere'. It is in this activity that Sloterdijk recognizes a polarity that typifies human nature. On the one hand, man is an 'animal of luxury' that enjoys an environment adapted to its needs (and wishes), but on the other hand he has to work continuously to keep that environment intact and functional, a life of preventing and caring

for, or, in short, of constant worry. It is after ’hominization’, which occurs in the period of the discovery of stone tools and the evolution of the brain and the hands for tool use (and subsequent refinement of the tools, etc.), that the upkeep of the ‘sphere’ becomes more and more important. This leads to a new selective process of ‘*culture competition*’ (Sloterdijk 2001, p.191, my translation). Each member of a culture has evolved a disposition to defend (and expand?) his ‘sphere’ to the fullest. This leads us to the fourth and final mechanism, ‘*Übertragung*’ (Sloterdijk 2001, p.207), which I shall translate as ‘transference’. Sloterdijk’s ‘*Sphären*’ trilogy is an elaboration of this concept. Qualities from the first space (the maternal uterus) are overlaid on other, ‘outer’ situations. Natural catastrophe and violence from others can enter and destabilize these self-created inner spaces. By continuously returning to a stock of memories and routines, for example religion and other rituals and myths, humans can prevent their ‘spheres’ from imploding under too much influx of the ‘new’ (or ‘other’), or they can use them to regenerate the former order if an instability has occurred. This, in the context of the ‘sphere’, is an evolutionary successful system to ‘dampen’ the vulnerable state of openness to the world, while at the same time providing a virtual kind of protection or ‘*symbolic immunology*’ (Sloterdijk 2001, p. 208, my translation) for encountering and unlocking it. The quality of the ‘sphere’, kept up by culture and technology, can be transferred to new, unfamiliar spaces. Speech plays a central role in assimilating new ‘hothouse territory’; that is, in the symbolic building and extension of the ‘spheres’. Sloterdijk calls speech the ‘*general organon of transference*’ (Sloterdijk 2001, p.210, my translation); it is a medium to make friends with the world, or, put less positively, a means to drag the ‘Other’ into the world order of the ‘sphere’.

Based on Sloterdijk’s synergistic mechanisms one would reach the conclusion that in human evolution natural selection has become, more and more, overshadowed by a cultural process of change. But is there any way of knowing to what extent the evolution of the human organism has become ‘uncoupled’ from old-fashioned natural selection? Is there biological evidence that

Sloterdijk's four mechanisms are a driving force behind human evolution? The best available approach at this moment to see if there is evidence for a complex of anthropogenetic mechanisms impacting on our biology at the level of DNA is to compare the complete genome of *Homo sapiens* to the complete genome of our closest kin in the animal world, *Pan troglodytes*, or the chimpanzee.

Genome-wide comparison of *Homo sapiens* and *Pan troglodytes*

The first question that genome-wide comparisons cleared up was that of the relationship between human, gorilla, and chimpanzee. Although humans have only 46 chromosomes and gorillas and chimpanzees have 48 (human chromosome 2 is apparently a fusion of ape chromosomes 12 and 13), humans and chimpanzees share identical inversions on chromosomes 7 and 8 that are not in the gorilla chromosomes, making humans and chimpanzees sister taxa, and gorillas the 'odd ape out' (Bradley 2008). Gorillas split off about 6-10 million years ago, and humans and chimpanzees split off from their last common ancestor about 5 million years ago. Pre-modern *Homo* taxa with obligate walking on two feet and medium-sized brains appear in the fossil record 1.9 million years ago, and modern *Homo sapiens* and (now extinct) *Homo neanderthalensis* 200,000 years ago (Wood 2008).

There is evidence that a single recent origin of modern humans in East Africa has replaced all other hominin forms (Sykes 2001), which means that we are all descendants of one small community of 1,000-10,000 individuals around 50-60,000 years ago. Geneticist Luigi Cavalli-Sforza (2001) was the first to suggest that humans underwent a series of successive 'population bottlenecks' while expanding out over the globe in small groups. Such a 'bottleneck' is caused by an event (often a natural disaster) that decimates a population and leaves only a small group to reproduce, offering an explanation for the phylo-geographic distribution of human diversity.

These bottlenecks may have been one of the reasons behind the finding that there is more genetic variability in a single chimpanzee community (chimps having maintained a steady population number over time) than in a global sampling of humans. More surprising, perhaps, was the discovery that most variation in humans lies within, rather than between, populations (Bradley 2008).

What has surprised (and even shocked) many is that we share with chimpanzees almost all of our genes and 98.8% of our DNA (or rather, 96%, if one includes differences in duplications, insertions and deletions, which, we shall see, are increasingly thought of as significant differences). Something special is going on, because, for example, in species of mice identical in 98% of their DNA the phenotypes do not differ as much (Bradley 2008). Therefore, indications are that, more than to modifications of the genes themselves, changes in the regulation of genes are likely to cause many of the differences between modern humans and chimpanzees.

In 2009, physician-scientist Ajit Varki[12] and his colleague Tasha Altheide listed many phenotypic traits for comparison with those of great apes. Covering many categories, the list does not only contain phenotypic differences that are obvious to any discerning human eye, like anatomical, behavioural, and life cycle differences, but also differences at organ-, tissue-, and molecular levels[13], as well as differences in cognitive capacity, communication, social organization, and culture. Varki and Altheide point out that a major limitation of translating genomic comparative information into an understanding of 'humanness' is the relative lack of knowledge about the basic phenotypic features of the great apes.

Of the many items on Varki's categorized list, Table 1 presents a shortlist comprising the phenotypic differences that Sloterdijk explicitly mentions. Varki also listed currently known

candidate genes and gene families that may contribute to phenotypic differences between humans and apes, and these have been added to Table 1 where possible. Also, other genetic sequence candidates have been will be added that Varki does not mention (*in italics*).

Table 1. Human phenotypic traits mentioned by Sloterdijk for comparison with those of the chimpanzee, and candidate genetic sequences involved in their generation

Category	**Phenotypic trait**	**Candidate genetic sequence**
Anatomy	Skull size	**ASPM, MCPH1**, genes, deletions linked with microcephaly (miniature skull)
	Brain size/development	**PCDH11Y**, duplicated genes, protocadherin, a cell adhesion molecule expressed in the brain **COX** gene family, mitochondria cytochrome oxidase units, perhaps to support increased brain energy consumption ***HAR1,*** *non-protein coding region perhaps necessary for development of (large) cortex (brain) (Pollard 2009, 2006)**
	Speech organ	No data (nd)
	Brow ridge	nd
	Chin	nd
	Visible whites of eyes	nd
Biomechanics	Walking on two feet	nd
	Adductive thumb	***HAR****2, non-protein coding region that drives gene activity in the wrist and thumb during*

		development (Pollard, 2009, 2006)
	Skeletal muscle strength	**MYH16**, gene, frame shift mutation, reduction in type II muscle fibres in human jaw
	Hand-eye coordination	nd
	Fine motor coordination	nd
Organ physiology	Olfactory sense	**OR**, gene family made of duplications of olfactory (smell) receptors
	Taste sense	**TAS**, gene family made of duplications of bitter taste receptors
Skin biology	Skin	**LCE**, rapidly evolving genes, epidermal differentiation complex involved in forming cornified (top) layer of skin
	Hair	**KRTHAP1**, single base pair mutation in gene, different hair protein (keratin) expression pattern
	Both	**KRT**, keratin and associated proteins, components of cytoskeleton (cell shaper) of epithelial cells
Nutrition	Wide range of foods and cooking	***LCT**, gene polymorphism (functional base pair mutation only present in part of human population), linked to lactase (milk digestion protein) persistence in Europeans in last 20,000 years (Burger, 2007)*
		***AMY1**, gene duplications, involved in starch digestion (Pollard 2009)*
Life history	Helplessness of the newborn	nd

	Prolonged helplessness of young Extended care of young Childhood, adolescence Age of first reproduction	nd nd nd nd
Reproductive biology	Virgin breast development	nd
Pregnancy, parturition	Disproportion between head and hips Need for childbirth assistance	nd nd
Postnatal development	Infant-caregiver attunement Maternal-infant eye-to-eye gaze	nd nd
Behaviour	Control of facial expressions Planning ahead Domestication of animals/ plants Secondary tool making	nd nd nd nd
Cognitive capacity	Symbolic representation	***DUF1220****, humans 212 gene copies vs. chimps 37, expressed at high levels in brain regions associated with higher cognitive function (Bradley 2008)*
Communication	Grammar and syntax Speech	nd **FOXP2,** gene, 2 single base pair mutations, transcription factor (regulates other genes);

	Writing	mutations linked to motoric speech disorder and speech understanding nd
Social organization	Institutions Social conventions	nd nd
Culture	Construction of shelters Physical modification of the body Customs and rituals Belief in the supernatural/religion[14] Weapons	nd nd nd nd nd nd

* Italics: addition of a phenotypic trait and/or candidate genetic sequence from another source than Varki & Altheide (2009).

The genomic comparison shows that it is likely to be rather impossible to elucidate which genes (and other genomic but non-protein-coding regions) play a role in the behavioural phenotype and cultural expressions of the human species. A point of oversight that Varki mentions is the poverty of information regarding the 'phenome' of the compared species, meaning 'the body of information describing an organism's phenotype under the influences of genetic *and* environmental factors' (Varki & Nelson 2007, my emphasis). It should also be noted that these are early results. Findings might be due to incomplete coverage of the chimpanzee genome, or intra-specific variation in the genomes of different individuals, for example differences in gene copy number (Bradley, 2008).

What nevertheless becomes obvious is that an idea of evolution depending only on spontaneous single base pair mutations that cause changes in protein functionalities that can sometimes confer a relative fitness advantage is simplistic and incomplete. Linking candidate genes to phenotypic traits is only one way to understand human evolution, a top-down approach. Using bioinformatics tools, it has become possible to take a bottom-up approach to lay bare all the genomic differences, without any understanding yet of their meaning. Important differences in the DNA sequence are those that cause protein changes, regulatory changes, or changes that occur due to gene loss and/or gene duplications (Bradley 2008).

Protein changes can occur when mutations in the base sequence of a gene are positively selected for and get fixated in the population that way. With the use of bioinformatics genomes can be compared and scanned for genes that have undergone accelerated evolution. The genes found are involved in smell, hearing, and include candidate genes for controlling skull and brain size (A*spm* and *Mcph*1), genes involved in skin biology, and a gene (*Fox*P2) (see also Table 1) that, when mutated, is associated with speech disorder and speech understanding (of which it must be noted that this protein is a transcription factor that regulates the transcription of several other genes). Other genes with links to different phenotypic traits that cannot be discerned by the eye, and for that reason may have escaped Sloterdijk's attention (and a listing in Table 1), are those involved immune defence, cell signalling, and amino acid metabolism. Changes at this level can have an impact on the development of other ('downstream') phenotypic traits, which could mean that these other traits were not directly selected for, but are a by-product of change at the more basic level. It is interesting to note that the number of positively selected genes in general is smaller in humans than in chimpanzees, despite a higher non-synonymous base pair substitution rate in humans (Bakewell 2007). Even genes for the brain seem not to have undergone accelerated evolution. On the contrary: chimpanzees accumulated more changes in brain-specific gene sequences (Shi et al. 2008).

Another genetic factor of influence on a resulting phenotype is the level of gene expression. Cells in different bodily tissues contain the same DNA; therefore, the different phenotype of the different tissues must depend on differential DNA expression. Microarray studies can compare the expression levels of thousands of genes from samples of human and chimpanzee body tissues at the same time. One result shows an upward regulation of genes expressed in the human brain. But it must be noted that human and chimpanzee gene expression profiles also differ much in the liver, so the meaning of found patterns is not straightforwardly clear (Bradley 2008). Besides an upward regulation of gene expression levels there is also evidence that the brain 'transcriptome' (the brain's complete set of RNA molecules) is remodelled during brain development after birth. These changes result in a developmental retardation, or 'neoteny', when compared to brains of other primates (Somel et al. 2009).

Still another way of looking for important differences is to look closely at conserved regions in the genome, thought to 'have undergone purifying selection' which might indicate functional importance, meaning that any mutation in that sequence would only lessen functionality. Of the 35,000 regions of DNA sequence conserved in mice, rats and chimpanzees, 49 were markedly different in humans and were called HARs (Human Accelerated Regions). Only two of the HAR regions code for proteins. The other 47 are likely to be regulatory regions: non-protein coding stretches of DNA to which , for example, transcription factors could bind to activate a cascade of genetic transcription. One of the regulatory regions, HAR1, may play an important role in the development of important neurological pathways. (Pollard 2009; Pollard et al. 2006). HAR2 may be involved in thumb and wrist development; of the other HARs not much is known yet. Interestingly, HAR1 has no protein-coding sequence but only makes RNA transcripts. This fits in with the growing appreciation in biological science that species-specific changes may be more

easily conferred through changes of regulatory RNA molecules, which could affect many targets on the route from genotype to phenotype (Varki & Nelson 2007).

A third of the gene duplications in the human genome seem to be human-specific. An example is Duf1220, of which humans carry 212 copies and chimpanzees only 37, and which is expressed at high levels in brain regions associated with higher cognitive function (Bradley 2008). One could think of such groups of duplications as gene 'families', in which new copies could presumably be free to acquire new functions, providing a strong driver for evolutionary change (Varki & Nelson 2007). Non-protein coding DNA duplications can also be relevant, for example, the large number of transposon *Alu*-elements: repetitive pieces of DNA that duplicate and move randomly to places in the genome. Jumping *Alu*-elements are likely to have altered functional genes and their regulatory elements, providing an important source of novel variety that can play a role in adaptive 'plasticity' (Fedoroff 1999).

Besides duplication, gene loss can have dramatic effects on phenotypes and fitness (Bradley 2008; Wang et al. 2006). Besides losing genes involved in taste and smell perception, loss of a hair keratin protein gene, and the inactivation of a gene expressed in the jaw muscles of mammals represented in table 1, there is an interesting amount of loss of function mutations in genes involved in pathogen resistance.[15]

In the reviews used here another type of epigenetic regulation is being ignored that currently is getting more and more attention. The regulated addition of acetyl molecules[16] to histones, the protein material in which DNA is packaged, creates room for the molecular machinery that transcribes DNA into RNA to bind. The integration of epigenetic chromatin tags into a fine-tuned transcriptional response is called the 'histone code' (Levenson & Sweatt 2005). The attachment of

small methyl molecules to DNA directly, on the other hand, can ‘silence’ a gene by keeping it from being expressed. A study in adults born shortly after a winter of hunger in the Second World War showed that DNA-methylation patterns can change during the development of a foetus. This epigenetic adaptation is triggered in the foetus by stress signals coming from the mother’s body (Heijmans et al. 2008). The acquired pattern can be inherited by the offspring of this child, and persist for a few generations, even when the stressful conditions no longer exist. Variations in chromatin acetylation and DNA-methylation can influence rates of mutation, transposition, and recombination of DNA. This can provide a bias for change in the DNA sequence (Jablonka & Lamb, 2008). It may provide an explanation for the tendency of the Human Accelerated Regions (HARs) to have biased patterns of DNA nucleotide substitutions (Berglund et al. 2009).

Interpreting the genomic data in the light of anthropogenetic mechanisms

So do the data from genome-wide comparisons fit with Sloterdijk’s theory? Do they provide evidence that the four interferential ‘anthropogenetic mechanisms’ are in play in human evolution, and if so, to what extent? Let’s start with Sloterdijk’s first anthropogenic mechanism, ‘insulation’ from natural selection or ‘group-internal criteria’, which supposedly ‘freed the evolutionary plasticity’ of humans. From the genomic data it is obvious that the phenotypic differences between humans and chimpanzees are, at first sight, much more striking than their genotypic differences: 96% of their DNA sequence is identical. And for all the human phenotypic variation that is going on, the chimpanzees have more variation in their genes, and show more evidence of positive selection pressures on gene variants. ‘Phenotypic plasticity’ therefore does not necessarily translate to variability in protein-coding sequences. There are also other ways to vary genetically: the same unchanged genes and/or other genetic sequences may be copied, or no longer used at all, or ‘redeployed’ somehow to perform different functions. As biologist and philosopher Lenny Moss has put it, at issue is not whether changes in genome structure are

relevant, but rather in what way changes in genome structure help open up new 'phenotypic possibility spaces' (Moss, 2006). Insulation from natural selection pressures could allow the opening up of new possibilities of a biological response to novel external cues – those of the 'sphere'. This creates new selection pressures – the process of variation, selection and reproduction does continue. It is the result of this process that is reflected in the performed genomic comparison. It is not hard to interpret found differences from an anthropotechnological perspective: changes in genes for milk and starch digestion, jaw musculature and taste perception seem aligned with changes in the way humans grow and prepare their food, and changes in genetic regions involved in wrist- and thumb development can be linked to tool use. Changes in brain size and development could be explained by the increased cognitive demands of living in a techno-cultural environment. The comparison, however, is not complete: it ignores genetic changes underlying differences in immune defence, cell signalling and amino acid metabolism, which may have profound consequences on the development of other 'downstream' traits, and could offer an alternative explanation for found differences. Also, the comparison (apart from the added non-protein coding HARs) says nothing of differences at the epigenetic level, a level now considered crucial to explaining phenotypic outcome and perhaps the reason for the lack of candidate genetic sequences for many of the traits on the list.

According to Sloterdijk's second anthropogenetic mechanism it was anthropotechnology that 'unburdened' the human body of many of the demands a natural environment would have made on it, and that allowed the 'drift' towards esthetic and cognitive traits that later met present group-internal criteria. In time these criteria caused the spontaneously emerging traits to come to fall under 'spherical' selection pressures, which could ultimately lead to these traits finding some type of genetic fixation. This idea follows the logic of the 'Baldwin effect', an idea proposed by James Mark Baldwin in 1896. Mutant variants would not have to precede selection, but could also follow it. In new conditions, phenotypical adaptations would allow many members of a

population to survive. Mutants and/or new gene combinations that arise later would be selected as they stabilize, refine, and extend the phenotypic adaptation; this would optimize the process of adaptation (Kirschner & Gerhart 2005). Baldwin's view can be broadened to include the organism's capacity for non-adaptive responses to environmental stress.[17] Taken together, the adaptive and non-adaptive responses encompass the entire range of phenotypes the organism could generate from its single genotype. So whereas the first step in adaptation may consist of adaptive and non-adaptive responses, subsequentially, if the new circumstances are recurrent, selection can drive gene frequency changes that increase fitness and heritability. But the phenotypic change need never be under genetic control alone. It could have a heritable and an environmental dependence (West-Eberhard 2003).

In his third anthropogenetic mechanism Sloterdijk speaks again of a 'drift', driven by the initial response to increase brain size, towards 'neoteny'. McKinney (1998) contests this and claims that human beings do not show arrested development of the brain, but rather over-development.[18] In domesticated dogs, where arrested development of the brain does take place during late fetal development, one can speak of juvenalization (compared to wolves, dogs stay juvenile all their life), not in humans. The 'fetal image' given off by the lack of body hair and the 'baby face' is just a coincidence. But human babies, compared to other primates, are born three months too soon. The evidence of Somel et al. (2009) that the brain transcriptome is remodelled after birth, resulting in a developmental retardation, speaks in favour of Sloterdijk's mechanism. After birth the human brain continues to develop for a far longer time than in other primates in the prolonged period of youth and adolescence. One would expect to find genetic evidence changes in genes involved in controlling hormone levels or of genes involved in now retarded and prolonged development, but under the heading of 'life history' we find no differences in genetic sequences linked to neoteny. But perhaps one should not expect genetic evidence if neoteny is more about

changes in gene regulation and in regulation at the level of RNA and protein activity that have an impact on hormone production than about changes in singular genes.

Crucial to understanding the fourth mechanism is the notion that the 'sphere' is kept up by culture and technology, and the fact that the evolutionary process has changed for humans – having transferred some emphasis away from their bodily evolution to the evolution of their anthropotechnology first, and the body second. Also, consider the resultant (partial) uncoupling from the old concept of fitness: large numbers of offspring are no longer a driver for the direction this evolution will take. Rather than fixating adaptations genetically through higher numbers of offspring, the transient character of the artificially supported and quite sudden increase of the human population plus the exceptional cultural and ecological changes give rise to an increase in the rate and effectiveness of adaptation (Hawks et al. 2007). In our evolutionary state of disequilibrium, many avenues can be explored. What is striking about the human organism, in this scenario, is the rate at which our 'evolvability' evolves. It is this type of adaptation that Sloterdijk urges us to consider when he speaks of 'plasticity' and 'freedom' at a genetic level. Biologists should not (only) be looking at the positively selected genes of a previous age, but at our reservoir of genetic variety and at biological ways that have evolved to access that resource. It is the evolution of new kinds of adaptability in the context of the 'sphere' that Sloterdijk wants to emphasize.

Discussion

Triggered by Sloterdijk's suggestion that all anthropotechnology, directly or indirectly, is gene technology, this study set out to see if an impact of our 'anthropotechnological' evolutionary history can be found in our genetic 'record'. The twentieth century has been called 'the century of

the gene': all attention was focused on finding and understanding protein-coding genes, and on understanding evolution in terms of spontaneous variations in these genes and the selection of gene variants that provided an advantage, however small, in terms of fitness. And a few of such genes have been found in the performed genomic comparison: genes correlated with increased skull size, jaw musculature, skin and hair, the capacity to digest lactose, and a gene that is involved in our capacity for speech. Clearly, these genetic differences fit in with Sloterdijk's idea that they are adaptations to a special, anthropotechnological environment. But just as with other adaptive explanations, these findings only provide corroborative evidence of Sloterdijk's hypothesis, not proof. Taking visible human traits as a starting point, mutations in genes involved in molecular traits like immune defence, cell signalling, and amino acid metabolism are neglected in Sloterdijk's theory. Perhaps they could be made to fit, but if not, these fundamental changes may have had a profound impact on the development of other, 'downstream' traits and could provide an alternative explanation for them.

What is more interesting, though, is that the approach of comparing DNA sequences shows the limitations of the traditional, 'gene-centred' view of evolution in terms of spontaneous mutations in protein-coding sequences and the subsequent selection of the 'fittest' variant. Half of the genetic differences found in this study are due to gene duplications. The gene copies start out selectively neutral and can eventually perform new functions or shut down. This raises new questions: how and why do these duplications take place? Is it a spontaneous or a directed process? Sloterdijk would suggest that, in an environment where the body is unburdened in many ways, such genetic copying and exploration is supported by a less stringent selection on parts of bodily functioning (like having fur, or tasting and smelling) that allows such 'drift' in the genome, '*liberating our evolutionary plasticity*'. So instead of only providing a new selective environment, the anthropotechnological 'sphere' could also have this rather different and unexpected impact on the entire genome. The same logic may hold for the presence of thousands of transposon *Alu*-

elements that cause the transference of pieces of sequence throughout the genome. These are quite different mechanisms from spontaneous point mutations, and provide a lot of variation, presumably often selection-neutral variation, that could also provide novel material for evolutionary processes of the Baldwinian type. Still considered unthinkable until late in the previous century (e.g. Gould, 1980), the idea of considering genomes as being complex, integrated, and dynamic is picking up now (Shapiro 2005).

Comparing known genes is a top-down approach. A bio-informatically informed bottom-up approach makes clear that there is a lot of significant variation to be found outside of the genetic sequences that code for proteins. It resulted in finding the positively selected group of HAR genetic sequences that mostly code for RNA molecules only (that perform a function in gene expression regulation and are not translated into protein). These regulatory RNA molecules do not feature in Sloterdijk's theory, but this is not surprising, considering he wrote his material around the start of this century. Of importance is that both the theory of Sloterdijk *and* the used comparative approach also ignore many other extra-genomic epigenetic factors, like DNA-methylation and chromatin acetylation. They are clearly a crucial key for understanding the link between genotype and phenotype. Projects mapping human epigenetic variety have recently started: the 1000 Genomes project, the Encyclopedia of DNA Elements (ENCODE), the Epigenetic Network of Excellence, and the Alliance for the Human Epigenome and Diseases (AHEAD).

At the level of epigenetics adaptation takes place in a fast 'mode' (Gluckman et al. 2009). Epigenetic patterns are sensitive to the experiences of an organism during its lifetime, and can be inheritable. Considering that our 'sphere' continues to evolve at an accelerated pace, it seems safe to assume that this impacts on our epigenetic flexibility. As epigenetic patterns may bias genetic change, it becomes easier to see how our anthropotechnological environment could have very

profound effects on the course of our organismal evolution. With an integrated, dynamic genome and the responsive capacity of the epigenetic level in mind, the original research question would need to be recast: Has anthropotechnology supported new routes of (epi-)genetic exploration unique to the human species? Or is the human (epi-)genome, in that respect, not fundamentally different when compared to other animals?

Adaptation can take place at yet another level that falls outside of the scope of this study. It is also in the anthropotechnology itself that information about the generation of a well-adapted human being is preserved. Focusing on 'hard' anthropotechnologies, philosopher Bernard Stiegler considers the use of technology as a sort of external memory that is unique to humankind (Stiegler 1994). He sees it as a third memory of 'organized anorganic matter' or 'epi-phylogenetic memory' that exists next to our genetic and epigenetic inheritance.[19] Obviously, the technological artefacts can evolve much faster than humans can. Humans may be the source of 'mutations' (variations) in these artefacts, but in the co-evolution of human and technology it is the technology that 'leads', evolving outside of human biology (Lemmens 2008). This logic also holds for 'soft' anthropotechnologies (like cultural rules of behaviour).

Conclusion

As our evolution takes place at different levels and at different rates, understanding itour evolution turns out to be far more complex than previously thought. The impact of anthropotechnology on our evolution cannot be assessed by genomic comparisons only. Although some evidence for an impact of an 'anthropotechnological' evolutionary history was found in our genes, the research highlighted the limitations of a genetic comparative approach. Evolutionary significant differences cannot only be found in changed genes, but also in changed genome architectures and dynamics, epigenetic patterns, and 'epi-phylogenetic memory'. This paper has

considered some ways in which changes at one level may impact on other levels. It strongly challenges old-fashioned ‘gene-centered’ views and the inclination in our society towards ‘genetic reductionism’.

Sloterdijk’s central observation that the creation of ‘spheres’, starting with the creation of the first stone tools and houses, has ‘*freed the evolutionary plasticity of the inhabitants of these weird spaces*’ and provided a starting point for the creation of humanity, turns out to be an observation of great interest. Philosophical anthropological work like Sloterdijk’s, which admittedly has its own flaws, nevertheless provides a perspective that cannot be dismissed and from which one can arrive at new research questions.

There is reason to believe that the impact of anthropotechnology on our evolution, relatively subtle (although still rather impressive) in the last two million years, has been growing and will continue to increase. A better understanding of this phenomenon, obtained by interdisciplinary research, will be necessary to guide us, as we reach an increased awareness of our own roles and responsibilities in this process of change.

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Endnotes

1 A term coined by Richard Dawkins in 1976, the ‘extended phenotype’ originally refers to the effect that a gene may have on an organism's environment through that organism's behaviour.

2 Sloterdijk is inspired by sociologist Dieter Claessens (*Das Konkrete und das Abstrakte*, Frankfurt am Main, 1980) when he redefines the socio-techno-cultural environment of humans as ‘Brutkasten’ or ‘Treibhäuser’.

3 By ‘*world*’ Heidegger does not mean the natural environment (or the artificial environments that Sloterdijk calls ‘spheres’) but the appearance of a new way of *being* in the human incomparable to the mere survival of animals. This transformation has completely changed man’s perspective on everything. The human is an arrival at the ‘*Lichtung*’ (‘an open place in the woods’) where ‘being’ and ‘what is’ are closely related.

4 For the mechanism of ‘group-internal criteria’ Sloterdijk builds on elements from the ‘*Umweltslehre*’ of the philosophical biologist Jakob von Uexküll. The environment is not just perceived as objects by the animal, but a world in which the animal gathers and construes the information that is salient or meaningful to him. (*Streifzüge durch die Umwelten von Tieren und Menschen, Bedeutungslehre*, Hamburg, 1956). Also, he refers to biologist Hugh Miller’s theory of insulation from natural selection pressures from ‘*Progress and decline: the group in evolution’ (*1964). Both works show that this mechanism is already relevant in the evolution of certain animals.

5 According to John Hawks (2007), 7% of human genes show quite recent ‘accelerated selected variants’.

6 It was Paul Alsberg who first suggested that man had freed himself from imposed natural and limiting obligations of bodily adaptation through his superior artificial tools in: *In quest of man: a biological approach to the problem of man’s place in nature*, Oxford-New York, 1970 (first published in 1922).

7 The unprecedented capacity to perform the unusual, coordinated, exceedingly rapid, sequential series of the movements of throwing and swinging has become part of the normal human developmental sequence. The theory that Sloterdijk invokes here is the ‘chimpanzee-thrower-scavenger-warrior’ model of human evolution of Eduard Kirchmann (*Das Zeitalter der Werfer*, Hannover, 1999).

8 Genes underlying traits that are no longer necessary might accumulate spontaneous mutations that, in time, could inactivate them and cause ‘gene loss’. Since there is no selection pressure to act on them, this has no consequences for the relative fitness of the organism, and the process falls under ‘neutral evolution’.

9 Not often mentioned in these discussions is the fact that in the past 20,000 years and particularly the past 10,000 years, body size shrank a little bit, and brain size shrank quite a lot (Zorich, 2008), about 8-10%.

10 Psychologist Nick Humphrey (“The social function of intelligence.” *Growing points in ethology*. Eds. P.P.G. Bateson & R.A. Hide, Cambridge, 1976), primatologist Robin Dunbar (*Grooming, gossip, and the evolution of*

language, Cambridge, 1997), and evolutionary and developmental psychologist Andrew Whiten ("The evolution of animal 'cultures' and social intelligence," Philosophical Transactions of the Royal Society B 362: 603-620, 2007) suggest a 'Machiavellian intelligence theory' in which the primate brain expanded to cope with the social complexity in groups that, with humans, grew to unusually large numbers. Neurobiologist William Calvin ("The unitary hypothesis: A common neural circuitry for novel manipulations, language, plan-ahead, and throwing?" in: K. R. Gibson & T. Ingold, eds., *Tools, Language, and Cognition in Human Evolution*, Cambridge, 1993), neurologist Frank Wilson (*The hand: how its uses shape the brain, language, and human culture* ,New York, 1999), and Eduard Kirschmann (1999, see note 10) see more in the theory of the 'throwing planner', suggesting that the tasks of producing sequences of gestures let to the development of a mind that formed the basics of a capacity for grammar and language that exploded the capacity for culture when further explored. Science writer Marek Kohn and archaeologist Steven Mithen ("Handaxes: products of sexual selection?" *Antiquity* 73 (281): 518, 1999) have suggested that sexual selection played an important role in the time of the Achuelan hand axe 1.5-0.5 million years ago. Women judged a potential mate by his virtuosity demonstrated by a beautiful hand axe. Others have subsequently suggested that big-game hunting itself was sexually selected. Susan Blackmore (*The meme machine*, Oxford, 1999) further develops Richard Dawkin's controversial idea of 'memes', 'units of culture' that are abstractly comparable to genes as 'units of biology'. In this theory memes, once present, compete to colonize brain space and thus drive human brain expansion.

11 For the notion of 'neoteny' of 'fetalization' Sloterdijk refers to paleoanthropologist Louis Bolk (*Das problem der Menschwerdung*, Jena, 1926) and to biologist Adolf Portmann (*Das Tier als soziales Wesen*, Zürich, 1953).

12 Ait Varki is currently co-director of The Museum of Comparative Anthropogenics, a catalogue (now under construction) of comparisons between humans and other hominids.

13 Interestingly, so far, *no* differences in cell biology are noted. This is in alignment with the view of systems biologist Mark Kirschner that the core processes of the living cell have been strongly conserved throughout evolution to provide the stable basis for adaptive explorations of organisms (Kirschner & Gerhart, 2005).

14 Geneticist Dean Hamer spoke of a 'god gene' when in an analysis of DNA from 1000 individuals he found a correlation between their personal scores on 'spirituality' and the VMAT2 locus (*The god gene: how faith is hardwired into the genes*, New York, 2005). However, this locus does not turn up in this comparison to the chimpanzee genome, which suggests that chimpanzees have the same gene as well, making it unlikely for the gene to be responsible for spiritual feelings. Twin studies also do not show a genetic basis to spirituality.

15 Conversely, it must also be said that many known human genes are missing from the chimpanzee genome, for example genes on the Y chromosome that are likely to play a role in sperm production.

16 Besides acetylation, chromatin histone proteins can also be methylated, ubiquilated, or phosphorylated.

17 For example, fruit flies respond to temperature stress by eye enlargement (Kirschner & Gerhart, 2005).

18 The human prefrontal cortex shows a relative growth of over 100%. So even if there is a delay, it is a delay without a growth rate reduction, and therefore the brain ends up with more neurons and more complexity.

19 As an example Stiegler uses the handmade stone axe: the information on how to use and how to make stone axes is laid down in the organization of its matter and so each (surviving) stone axe can serve as a model for future generations (of people and axes).