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Genetic assimilation in the evolution of hominin neuroplasticity

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Abstract
The discovery and popularisation of the study of different evolutionary processes outside the current evolutionary framework provide researchers of different areas with new tools to make progress in their field. In this work, I explored how genetic assimilation may have played a role in human evolution, more specifically the evolution of our brains. The results indicate that human brain evolution concerning neuroplasticity fits within a context of genetic assimilation. However, further studies in comparative genomics and neurogenetics are needed to fully understand how the evolutionary changes happened at the genetic level.

Keywords
genetic assimilation, neuroplasticity, human evolution, extended evolutionary synthesis

1. Introduction

Learning the evolutionary past of our species provides us the knowledge to realize who we are, by helping us to understand our uniqueness and the origin of these unique traits that make our species special. One of these traits is the enhanced ability of our brains to change and adapt as result of our interaction with the environment. This increased neuroplasticity in human beings is indeed an exceptional feature, for it is related to our enhanced learning capacities, and further, to cultural evolution.

The core of current evolutionary explanations is the theoretical framework forged on the 1930s and 1940s called the Modern Synthesis. Considerable debate has been present in the current literature about the limits of the Modern Synthesis, some of them revisit old controversial concepts developed on evolutionary theory with new arguments and examples. One of these concepts is genetic assimilation,

In the light of the current increase in the interest for the study of genetic assimilation, the goal of this article is to present the results of the investigation about the possibility of an explanation for the origins of human neuroplasticity using the mechanisms genetic assimilation.

In order to achieve this goal, first I explore the debate about genetic assimilation and describe such a process and its mechanisms. Then I analyze the theories about hominin evolution and the environment in which such evolution took place. The third step was to review the research on neuroplasticity and delineate how this trait manifests

in humans. At last to characterize the interaction between neuroplasticity and genetic assimilation.

The analysis of human brain evolution by the process of genetic assimilation seems promising, it seems that further research on this topic may provide answers to other big questions on human evolution, e.g. the rapid rate of human evolution. This investigation also encourages the research of human evolution under the lens of other evolutionary processes outside the modern synthesis, which can also be fruitful for a better understanding of the origin of the uniqueness of the human being.

2. Genetic assimilation

During the 1930s and 1940s, many scientists were longing to find an explanation of how works on evolution could be unified with discoveries in ecology, paleontology, systematics, developmental physiology etc. The combined work of these scientists would be later called the modern synthesis of evolution by Julian Huxley in his book *Evolution: the modern synthesis*. This synthesis would be the framework for much of the further research on evolutionary theory (Huxley 2010). The result of the synthesis is well described as:

> The major tenets of the evolutionary synthesis, then, were that populations contain genetic variation that arises by random (i.e. not adaptively directed) mutation and recombination; that populations evolve by changes in gene frequency brought about by random genetic drift, gene flow, and especially natural selection; that most adaptive genetic variants have individually slight phenotypic effects so that phenotypic changes are gradual (although some alleles with discrete effects may be advantageous, as in certain colour polymorphisms); that diversification comes about by speciation, which normally entails the gradual evolution of reproductive isolation among populations; and that these processes, continued for sufficiently long, give rise to changes of such great magnitude as to warrant the designation of higher taxonomic levels (genera, families, and so forth) (Futuyma 1986: 12).

The influence of the modern synthesis on the understanding of evolution is undeniable, however many scientists suggest a revision or extension of this theory (Laland et al. 2014). Figure 1 illustrates the relations between features of Darwinism, the Modern Synthesis and a proposed integrated synthesis, or extended evolutionary synthesis.

Although, not all concepts suggested to complement the evolutionary framework are new. Phenotypic plasticity, for instance, was first discussed many years ago. This idea however embraces many processes, and one of these processes has received much attention in the last years, as the number of articles including this topic exponentially increased. This process, which was first observed in the 1940s, is genetic assimilation.

The process of genetic assimilation was first investigated in a genetic context in the 1940s by independent works of Waddington (1942) and Schmalhausen (1949), however it was not until 1953 that the term was used for the first time. Waddington formulated that,

> if an animal subjected to unusual environmental conditions develops some abnormal phenotype which is advantageous under those circumstances, selection will not merely increase the frequency with which this favorable result occurs, but will also tend to stabilise the formation of it, and the new development may become so strongly canalised that it continues to occur even when the environment returns to normal. For a series of events of this kind, the name "genetic assimilation" may be suggested. (Waddington 1953: 125).

Waddington put forward the hypothesis for genetic assimilation and provided evidence for it, however, he did not provide a clear account of which mechanisms were behind this assimilation. Braendle and Flatt (2006) suggest that the phenotype of the wild-type through generations

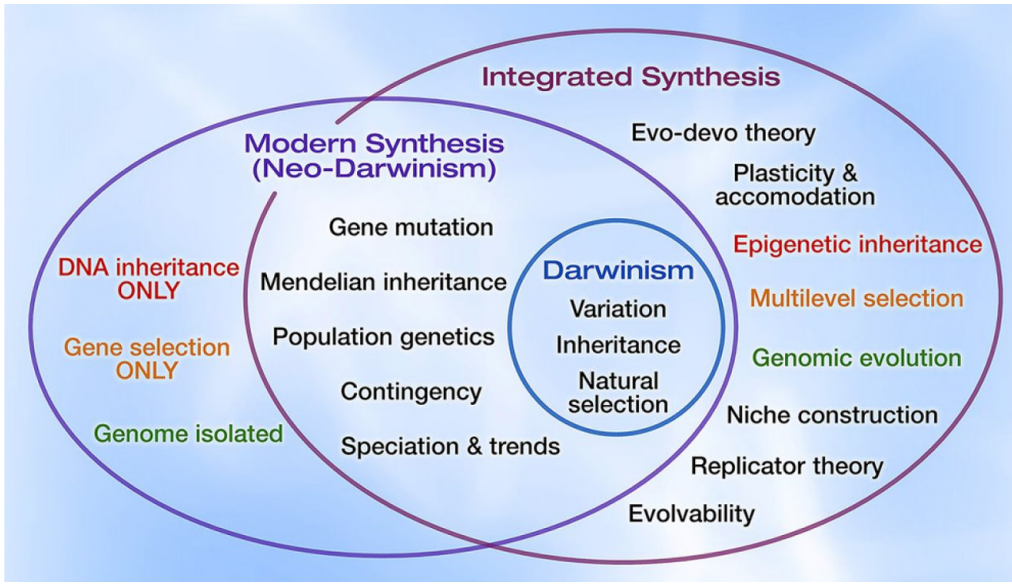

Fig. 1. Diagram illustrating definitions of Darwinism, Modern Synthesis (neo-Darwinism) and Integrated Synthesis. The diagram is derived from Pigliucci and Müller's (Pigliucci, Müller, 2010) presentation of an Extended Synthesis. All the elements are also present in their diagram. The differences are: (1) the elements that are incompatible with the Modern Synthesis are shown coloured on the right; (2) the reasons for the incompatibility are shown in the three corresponding coloured elements on the left. These three assumptions of the Modern Synthesis lie beyond the range of what needs to extend or replace the Modern Synthesis; (3) in consequence, the Modern Synthesis is shown as an oval extending outside the range of the extended synthesis, which therefore becomes a replacement rather than an extension (Noble 2015: 8)

of natural selection becomes relatively invariant to minor perturbations caused by environmental differences or mutations in genes that are, in this given environment, not expressed during development. Therefore many genetic changes can accumulate in an individual without producing a phenotype, and being so invisible for natural selection. This is called cryptic genetic variation. (Jablonka, Lamb 2005)

This variation is revealed then when the individual is exposed to exceptional mutations or an unusual environment that will change the epigenetic regulation of those genes and the development will be pushed away from the canalized pathway. In this condition, new phenotypes are produced from those many genes that were hidden and now they can be subject to natural selection.

Pigliucci, Murren and Schlichting (2006) posit genetic assimilation in other words, however with the same meaning of those above. They consider that this process occurs when there is a shift on the reaction norm that underlies a phenotype's expression in a population (Fig. 2).

Pfennig and Ehrenreich (2016) propose that studies regarding the molecular basis of gene expression variation and genotype-environment interaction can provide valuable ideas for the possible cause of genetic assimilation, and that genetic variants that affect gene regulation contribute as the mechanisms of this phenomenon (Pfennig, Ehrenreich 2014). One way in which gene regulation can be related to genetic assimilation is when a secondary pathway of regulation may evolve in which the changes in the plastic

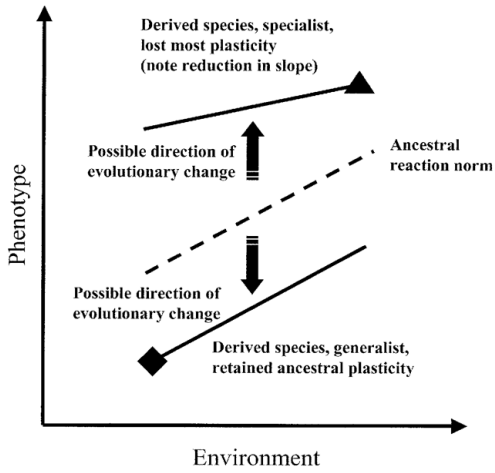

Fig. 2. A scenariogram illustrating a common pattern of differentiation of phenotypic plasticity that may be caused by ongoing genetic assimilation: the lines represent population or species reaction norms for an ecologically important character expressed in response to the variability of a given environmental factor. The ancestral reaction norm is depicted as a broken line. After speciation (or separation of the original population) the two taxa evolve an increasing degree of adaptation to their particular environment, which at least in some cases (upper reaction norm) is accompanied by a reduction of the degree of plasticity. See text for a discussion of some actual examples following this general pattern (Pigliucci, Murren 2003: 1460)

phenotype become more robust to the environment.

A final consideration left to be made is about the rapidity of this process compared to standard evolution by allelic substitution only (Pigliucci, Murren 2003). Waddington (1953) itself realized that the induced phenotype rapidly spread through the population in his experiment. Behera and Nanjundiah (2004) using a computational model argued for this speed up in evolutionary change brought about by genetic assimilation and phenotypic plasticity.

Understanding new evolutionary concepts, processes and the mechanisms behind them is essential to develop a better explanation of the origin of our species, especially when those processes can explain rapid evolutionary change, as it is believed that the evolution of our own species was rapid, particularly considering our brain (e.g. see Dorus et al. 2004).

3. Hominin evolution

During the last 2 million years of hominin evolution many changes happened in the brain, and many were the evolutionary processes that led to the evolution of homini from arboreal creatures to conscious individuals that can intelligently explore and manipulate their surroundings. Figure 3 illustrates the changes in cranial capacity, and thus brain size across hominin evolution.

As stated by Falk (2016), many attempted to solve the problem about the ultimate cause of our species having a big brain, the list, he says, includes: language, tool production, warfare, hunting, labour, Machiavellian intelligence, food gathering, and social intelligence.

Bailey and Geary (2009) propose that among those explanations there are three classes of models for hominin brain evolution, which are not mutually exclusive, the climatic, ecological and social models. The results of their assessment of these models suggest that multiple factors played a role, but the core selective pressure being of social character. Dunbar's (2009) social brain hypothesis became the current paradigm about social models of brain evolution, Dunbar proposes that large brains reflect the computational demands of the complex social systems that our antecessors were living.

Ash and Gallup's (2007) study goes however in a different direction, they compare environmental hypotheses of human evolution and propose that a significant and substantial proportion of variation in brain size correlated with paleoclimatic changes in temperature.

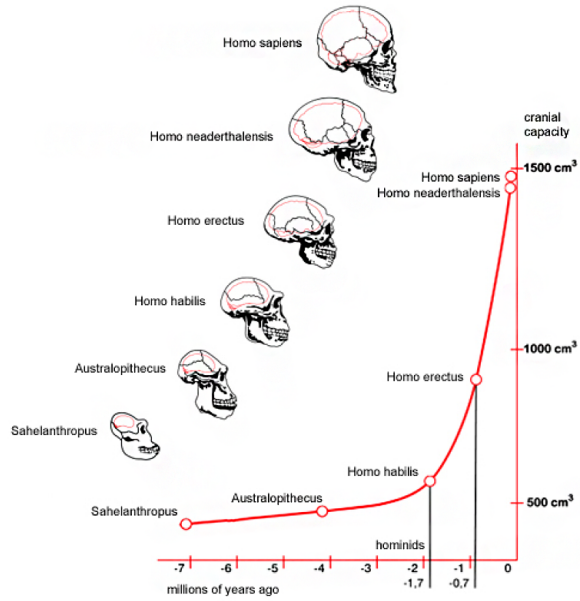

Fig. 3. Change in brain cavity size among hominids, showing a general trend towards larger brains in human evolution (Web-01

The last of the suggested models of hominin brain evolution by Bailey and Geary (2009) is the ecological model, which focus on the selective advantages of hunting and other adaptations that enable efficient extraction of biological resources. It is supported by findings that species with complex foraging or predatory demands have larger brain volumes and that changes in tooth morphology and tool sophistication with the emergence of australopithecines and in later hominids are also consistent with coevolutionary change in hunting efficiency, diet, and brain volume.

Although these three models are essentially different from each other, there is one factor that is common for all of them, they "highlight the adaptive advantages of the ability to anticipate and mentally generate strategies to cope with variation and change." (Bailey, Geary 2009: 68). Therefore in all of those hypotheses, it is assumed that individuals faced novel situations, variation in their surroundings and change in habits, and that these changes were the motor for hominin evolution.

4. Neuroplasticity

Neuroplasticity is a term that encompass many processes, however, in general, it can be regarded as the ability to make adaptive changes related to the structure and function of the nervous system (Zilles 1992). In other words, Pascual-Leone et al. state that:

> Plasticity represents an intrinsic property of the nervous system retained throughout life that enables modification of function and structure in response to environmental demands via the strengthening, weakening, pruning, or adding of synaptic connections and by promoting neurogenesis. (Pascual-Leone et al. 2011: 302)

It is not the case, though, that the brain of every individual is plastic in the same way, it is also not the case that this plasticity is the same during an individual's life (Fig. 4). Many studies have been conducted to understand how some lifestyle choices influence individual differences in neuroplasticity. Pittenger and Duman (2008) argue that chronic stress can hinder

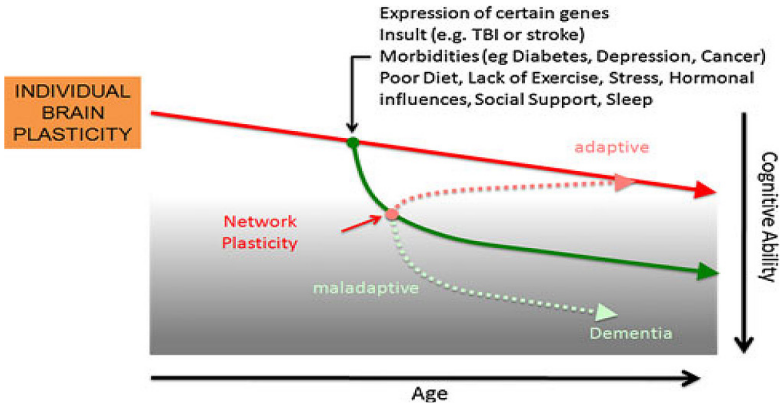

Fig. 4. Schematic representation of the influence of genetic or environmental impacts on brain plasticity. Alteration of local plasticity will trigger secondary adaptive responses across diffuse neural networks that may prove ultimately adaptive or maladaptive for the individual. Depending upon the amount and scope of such secondary responses, initial insult effects may be alleviated or heightened (Pascual-Leone et al. 2011: 308)

neuroplasticity. Gorgoni et al. (2013) in a similar fashion explore the decrease in synaptic plasticity presumably caused by constant sleep deprivation. On the other hand, mindfulness and meditation are argued to influence an increase in structural changes of the brain (Lazar et al. 2005).

Not only specific lifestyle choices were compared to assess individual differences in neuroplasticity, but also more general environmental features that are applied to a different range of scenarios. Exposure to more social and novel environments are two of these. Animal models provide evidence that sociality and novelty seems to favor neurogenesis and synaptic plasticity.

Lu et al. (2003) developed an experiment in which twenty-two-day-old rats were housed in isolation or in groups for 4 or 8 week to investigate the effects of social environments on learning and memory, neurogenesis, and neuroplasticity. The results demonstrated that more social environments can increase neurogenesis and synaptic plasticity in adult hippocampal regions, which is associated with alterations in spatial learning and memory.

A different method was used by Van-Elzakker, Fevurly, Breindel and Spencer (2015) to understand how novelty affects neuroplasticity, in this experiment they exposed rats to an environment with varying amounts of novelty and examined Fos protein expression within subregions of rat hippocampal formation, a protein that supposedly is an indicator of recent increases in neuronal excitation and cellular processes that support neuroplasticity (Sheng, Greenberg 1990). The results argue for a positive correlation between environmental novelty and Fos expression in rat's hippocampus, and therefore an increase in neuroplasticity.

The method from VanElzakker et al. (2015) to analyze neuroplasticity leads us to a different approach to this phenomenon, i.e., how genetic, epigenetic factors and thus protein expression are involved in the plasticity of an individual's brain and its individual differences.

Teles, Cardoso and Oliveira (2015) review the genetic underpinnings of neuroplasticity, while Woldemichael, Bohacek, Gapp and Mansuy (2014) and Felling and Song (2015) draw perspectives on the way epigenetic processes may support brain plasticity in relation to, amongst other things, drug addiction and cognitive dysfunctions, and Fuchsova, Julia, Rizavi, Frasch and Pandey's (2015) research the way that gene expression and neuroplasticity interact in depressed

subjects. Further studies are needed to reveal the epigenetics of novelty and sociality that causes the increase in brain plasticity in individuals subject to those environments.

Comparative studies by Gomez-Robles, Hopkins and Sherwood (2013) and Gomez-Robles, Hopkins, Schapiro and Sherwood (2015) argue for a higher plasticity in humans and that this this anatomical property of increased plasticity, may underlie our species' capacity for cultural evolution (Wexler 2011). This increased capacity of the brain to reorganize, and the possibility of creating new neurons on the hippocampus allow the humans to have a higher adaptability to its environment (Fig. 5). Pascual-Leone et al. posit:

> The world we live in changes rapidly. Afferent inputs and efferent demands to the brain shift quicker than the time needed to implement genetic or even epigenetic changes. Brain plasticity can be conceptualized as nature's invention to overcome limitations of the genome and adapt to the rapidly changing environment (Pascual-Leone et al. 2011: 302).

Potts (1998) proposes that complex morphological structures and flexible behaviors – allowing for novel responses to newly encountered selective pressures – its the key adaptation of the hominin lineage, emphasizing the evolutionary emergence of traits providing selective advantage to hominins in unstable conditions, without invoking changes in the reaction norm or the need for genetic polymorphisms. Given what was already discussed, neuroplasticity underlies this flexibility.

Neuroplasticity, then, is an intrinsic property of an animal's brain that facilitate its flexibility to a range of environments. During the fast brain evolution, not only size evolved, but also, as discussed, the plasticity of our brains contribute for the properties that define us as humans: our behaviour flexibility and our capacity for creating a complex culture. Evolutionary approaches to neuroplasticity use to take into account the dogmas of the modern synthesis. In the next chapter it will be discussed in a different fashion, genetic assimilation will be incorporated in the discussion of the evolution of our brain.

5. Discussion

As presented before, there are many ways to look at the phenomenon of genetic assimilation, some approach it to understand the canalization of a trait, i.e., the complete lost of dependency on its environmental

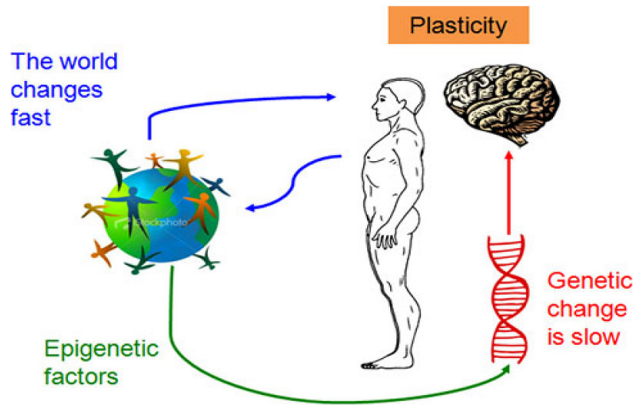

Fig. 5. Schematic representation of the concept of plasticity. Brain plasticity allows for rapid adaptation to environmental changes that occur quicker than genetic or epigenetic response times (Pascual-Leone et al. 2011: 303)

trigger. That accounts for cases of complete assimilation. Pigliucci and Murren (2003), as previously discussed, approach it by another angle, they consider genetic assimilation to occur when a shift on the reaction norm that underlie a phenotype's expression in a population, which is accompanied by a lost of plasticity, what in extreme cases leads to a total canalization.

However, the approach closest to the one that will be used in this discussion is the one of Jablonka, Ginsburg and Dor:

> Genetic assimilation is a process whereby selection for the developmental capacity to respond adaptively to a new persistent environmental stimulus (for example, a new chemical, a heat shock, or a new predator) leads to the construction of a genetic constitution that facilitates such an ontogenetic adjustment. It is based on pre-existing heritable differences among individuals in their responsiveness to changed conditions. For example, individuals who can learn more readily how to avoid a new type of predator would have a selective advantage, and hence, over time, the genetic constitution of such individuals would become more common. Eventually, the behavioural trait, which was originally learned after many trials, appears with a briefer induction and far less learning; in extreme cases, it appears after just a single exposure to the stimulus. The trait is then said to be genetically assimilated. (Jablonka et al. 2012: 2153).

After this brief clarification we can, in a similar fashion to Jablonka et al., apply it to understand the evolution of human neuroplasticity. First of all it is necessary to understand Neuroplasticity as a plastic phenotype underlined by a network of epigenetic and genetic factors and that the expression of this phenotype (i.e. the amount of changes in the brain) depends on environmental stimuli.

Secondly, it is worth remembering that genetic differences cause dissimilar "starting points" for different individuals and distinct lifelong "slopes of change" in plasticity (Fig. 6). In accordance with Jablonka's definition, it accounts for the pre-existing heritable differences in responsiveness to different conditions.

Another important factor for the occurrence of genetic assimilation is a new persistent environment, that inflict changes in the developmental trajectory of a species and on the expression of a plastic phenotype in the adult individuals leading to a higher adaptability to this new environment. Studying our evolutionary trajectory we see that the biggest environmental changes leading to hominin evolution were a changing environment demanding individuals to process novel information and a much more complex social systems requiring a higher level of social interaction (Bailey, Geary, 2009; Ash, Gallup 2007).

The evaluation on the causes of individual differences in neuroplasticity present on the last chapter pointed already the influence of these two environmental factors – novelty and sociality – in the increase of expression of genes related to neuroplasticity and adult neurogenesis, and also the influence of an increased neuroplasticity to the fitness and adaptability of our species to a different range of environment.

Summing up these three factors described in the last paragraphs it is possible to sketch a scheme for how genetic assimilation may have played a role in the evolution of the human brain. Rewriting Jablonka's statement about genetic assimilation in relation to neuroplasticity given these factors we reach the following scheme.

The environment approximately 2 million years ago suffered some changes leading early hominins to face increased novel stimuli and social interaction. This new environment containing these two properties induced an increase of neuroplasticity in the individuals of early hominin species, and this increase of neuroplasticity consequently enhanced the adaptability of these individuals to this environment.

This increase in neuroplasticity, however, did not occur in the same amount for all the individuals, some individuals' brain

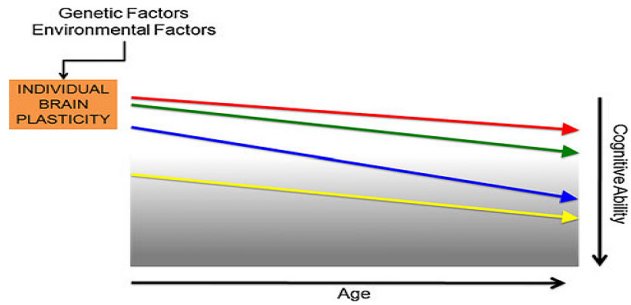

Fig. 6. Schematic representation of individual plasticity across the lifespan. Although mechanisms of plasticity show a downward trend over the course of a typical lifetime, this trend will manifest differently according to initial "baseline" levels, genetic factors, and environmental influences. Therefore, one may conceptualize each individual has a unique "slope of plasticity" across the lifespan (Pascual-Leone et al. 2011: 303)

reacted more appropriately to this new environment by having a higher increase in neuroplasticity than the brain of others. Since a higher increase in neuroplasticity led to a higher adaptability, these individuals were selected in opposition to individuals whose brain did not respond to the environment in the same way.

In the words of Pascual-Leone et al. (2011), we can consider that the individuals with a more favourable "slope of change" given this new environment were selected, what according to Pigliucci and Murren's approach to genetic assimilation, can be considered that there was a selection for the reaction norm that underlie the expression of this phenotype (neuroplasticity). Hence, this "slope of change" became more common across the population, and a higher expression of neuroplasticity began to necessitate less of an enriching environment.

Although in this scheme we can see that it was possible that genetic assimilation may have occurred in the evolution of hominin neuroplasticity, a complete canalization of this trait did not occur, since in modern humans' neuroplasticity still is a plastic phenotype, however it is clear that some kind of robustness of the expression of this trait was assimilated, since, e.g., humans reared in non enriching environments, such as, maltreatment during development, subjection to stress and sleep deprivation, still present a higher level of cognitive adaptability than other apes, like chimpanzees or bonobos, reared in socio-communicatively rich environment (Russell et al. 2011).

Another side of evolutionary explanation also must be assessed in order to understand the trajectory of our species, that is the genetic and epigenetic changes that led to this evolution. In the framework of genetic assimilation, exposure to a new environment may change the epigenetic regulation of some genes, revealing therefore genetic variation that was not expressed and not subject to selection, this genetic information that was hidden would then contribute to the development and adaptability of the organism and thus influence in the survival and reproduction of the individual.

It is now easy to link with certainty genetic change to alterations in brain phenotype. In the review from Vallender, Mekel-Bobrov and Lahn (2008), they argue that in almost no case there are experimental data linking sequence changes of a gene to alterations of brain phenotype and also that those explanations are rarely in specific terms, most of them just draw general remarks about the involvement of a gene in brain evolution. They also present some ideas of how to design experiments to demonstrate

a definitive causal relationship between the sequence evolution of a gene and the phenotypic evolution of the human brain, that would involve, e.g., introducing human genes into nonhuman primates and vice versa, or placing genes from humans and other primates into mice and examine the effect on phenotype.

Another complication for understanding the involvement of the environment in a genetic assimilation model for human brain evolution, is that few is known exactly about what is the epigenetic changes in the expression of certain genes related to neuroplasticity according to the exposure to different environments. Fuchsova et al. (2015) study is an example of how this interaction can be assessed.

In order to build a genetic background for the evolutionary hypothesis that genetic assimilation played a role in the evolution of hominin neuroplasticity, it is still necessary to understand other questions about the human genome, and comparatively, the genome of other hominid species. Especially the answering of two questions would be the most fruitful, first of all, how novelty and sociality changes the gene expression in humans and other hominids causing them a higher neuroplasticity, and second, the genome-wide changes that led to the evolution of the modern human brain.

In spite of the difficulties in the formation of a complete understanding of the mechanisms of genetic assimilation during hominin evolution, there are some studies that lead to exciting outcomes to this paradigm. As discussed before, evidence indicates that changes in reaction norms may be driven by variation in signalling pathways that mediate the relationship between genotype, environment and phenotype (Ehrenreich, Pfennig 2016). At the same time, Vallender et al. recognize that,

> changes in gene expression might have played an important part in the emergence of the human phenotype. In particular, it has been argued that small changes in non-coding regulatory elements could strongly impact the spatial and temporal expression patterns of key developmental genes, which could have profound phenotypic effects. (Vallender et al. 2008: 6)

Finally, they argue that several genome-wide analyses have been performed to systematically identify cis-regulatory regions of many brain-development genes might have experienced positive selection and accelerated rates of change during human evolution (Vallender et al. 2008). Combined to Ehrenreich and Pfennig's apprehension that cis regulatory variants have the potential to rapidly canalize the expression of individual genes, we may have a prospective explanation of how genetic assimilation may have occurred during hominin evolution.

It is not clear exactly the genetic underpinnings of hominin brain evolution, it is also not clear the approach to understand these genetic variations, and also to which variations we should be looking to assess neuroplasticity. It was only with recent technological advances that it was possible to read and analyze the sequences of genes across species, developing therefore the field of comparative genomics, and to understand the genetic basis of neurobiology. Further studies in these areas will certainly fill some of the gaps to develop a full theory of genetic assimilation and human brain evolution.

6. Conclusion

The study of the living conditions during hominin evolution, the apprehension of the influences of the environment in the individual differences of neuroplasticity and the exploration of the phenomenon of genetic assimilation lead us to believe that it was possible that during hominin brain evolution, phenotypic plasticity, and therefore some kind of canalization, may have had an impact, and also may be the explanation of the, widely accepted, rapid pace of evolution that our ancestors were subjected to.

Although an explanation based on genetic assimilation may be plausible given the factors previously mentioned, the genetic underpins of the changes leading to the appearance of our species are still uncertain given the infancy of research programs responsible to investigate these problems, e.g., comparative genetics and neurogenetics. Further development of the technologies used in these areas, and the maturation of the research programs will certainly contribute to insights on human evolution.

Another factor that hinders the expansion of new approaches to human evolution is a misunderstanding on the role of phenotypic plasticity in the whole evolutionary theory, and therefore a fear of an attempt to overthrow the Modern Synthesis. This neglect inhibits the development of a deeper clarification of the mechanisms of genetic assimilation and the encouragement for its application to different areas of knowledge, e.g., psychology and anthropology.

In this sense this work was planned and completed to encourage the exploration of novel concepts of evolutionary theory in the understanding of human evolution, human mind and human nature. Not only the modern synthesis, or more boldly, genetic assimilation, can guide research about human evolution, the whole spectrum of the proposed extended synthesis can be evaluated, debated and if possible applied to answer the fundamental questions of how we are connected to the nature and at the same time what is about our species that makes us so unique.

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Asymilacja genetyczna w ewolucji neuroplastyczności homininów

Streszczenie

Odkrycie i popularyzacja badań różnych procesów ewolucyjnych poza obecnymi ramami ewolucyjnymi zapewniają naukowcom reprezentującym różne dyscypliny wiedzy nowe narzędzia do osiągania postępu na polu ich własnych dyscyplin. W niniejszym opracowaniu przebadano jaką rolę mogła odegrać asymilacja genetyczna w ewolucji człowieka, a dokładniej w ewolucji naszego mózgu. Wyniki wskazują, że ewolucja ludzkiego mózgu w zakresie neuroplastyczności przystaje do kontekstu asymilacji genetycznej. Konieczne są jednak dalsze badania w zakresie genomiki porównawczej, by w pełni zrozumieć zmiany ewolucyjne, jakie dokonały się na poziomie genetycznym.

Słowa kluczowe

asymilacja genetyczna, neuroplastyczność, ewolucja człowieka, rozszerzona synteza ewolucyjna