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MOLECULAR LAMARCKISM: ON THE EVOLUTION OF HUMAN INTELLIGENCE

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In modern times, Lamarck's view of evolution, based on *inheritance of acquired traits* has been superseded by neo-Darwinism, based on *random DNA mutations*. This article begins with a series of observations suggesting that Lamarckian inheritance is in fact operative throughout Nature. I then launch into a discussion of human intelligence that is the most important feature of human evolution that cannot be easily explained by mutational selection. Thus, we are smarter than demanded by our evolutionary experience as hunter-gatherers. The difficulty lies in the inability of neo-Darwinism to satisfactorily answer the following question: How can a large energy-costly set of genes, each member of which has little apparent benefit when first created individually, all gather into a permanent existence within a short time period in each and every member of a small population (that was dispersed and geographically isolated over a huge planet) who had a low reproductive output, a low rate of beneficial mutations, and a low level of genetic contact? The article concludes with a speculative but far-reaching epigenetic theory of intelligence that does not require DNA mutation as the exclusive source of evolutionary change. Instead, *cranial feedback* relating brain chemistry, as affected by brain activity including education, with the genome. When it comes to the fast rate of evolution, and the dissemination of the intelligence trait worldwide, cranial feedback could make all the difference.

KEYWORDS: Cranial feedback, epigenetics, evolution, human intelligence, Lamarck.

INTRODUCTION

Jean-Baptiste de Lamarck, half a century before Darwin, expressed the view that certain acquired traits are passed on to future generations. Robyn Lindley in her book "Soma" writes: "There is now extant scientific evidence suggesting that nature has evolved a number of molecular mechanism for us to consciously direct

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our own evolutionary future. We ignore this at our peril." (p. IX). Why, then, am I writing about Lamarckism, a subject of past and current ridicule? The answer is simple: I am backed by observations that overwhelmingly support a departure from strict random-mutation/selection as the core of our evolutionary belief system. Yet Richard Dawkins (2010), a prime spokesman for neo-Darwinism, has stated that it is an established, unarguable fact that all life on this planet is shaped by Darwinian theory. Although recent decades have seen many supplemental offshoots to this theory, in the end these mechanistic variations reduce to basic neo-Darwinism. Only the Lamarckian concept stands as a truly divergent view of evolution.

In 1809 Jean-Baptiste de Lamarck published his *Philosophie Zoologique*, which elevated him to the status of a great evolutionist. Evolution is popularly attributed to Darwin, but this is grossly unfair to Lamarck who was the first to devote an entire book exclusively to the subject. True, Lamarck never documented his ideas with exacting observational data as did Darwin. And Lamarck's *inheritance of acquired traits* seems (incorrectly as we will see) to lack the power and generality of Darwin's natural selection. Yet the reality of evolution (including that of humans) was championed by Lamarck before Darwin was born.

Examples of "inheritance of acquired traits" can be taken from Darwin himself. In the "Descent of Man," Darwin advised young women to learn as much as they can prior to starting families, the expectation being that this would help endow the future children with useful skills. In 1873 Darwin published a note in *Nature*, entitled "Inherited Instinct," which describes a dog's violent antipathy toward a butcher who had mistreated the animal. According to Darwin, the antagonism had been transmitted to at least two generations of the dog's offspring. Darwin wrote that many special fears, and tastes, which must have been acquired at a remote period, are now strictly inherited." Darwin paying lip-service to the Lamarckian mechanism is an interesting historical note but, more to the point, there exist actual experiments testing the mechanism, and these are presented below. The listed experiments and observations, many of them classics in the field, all suggest that a Lamarckian inheritance is operative in Nature. Taken collectively, a large body of experiments offers a neo-Lamarckian view of evolution that is too compelling to ignore. As will be argued at the end of the article, even human intelligence has Lamarckian ("epigenetic") origins.

EXPERIMENTS AND OBSERVATIONS

William McDougall (1927) published a paper entitled "An Experiment for the Testing of the Hypothesis of Lamarck." He reported that the first generation of rats made an average of 165 mistakes during their first run through a maze. After some practice the rats learned to carry out the task perfectly. The offspring of these trained rats, however, made an average of only 20 mistakes on their first run as if they had inherited the acquired maze-running ability. It was concluded that Lamarckian transmission is a *real process in nature*. Others have disagreed and speculated that the effect derives from parent-to-young training (Sonneborn, 1931). In any event, this is one of those tantalizing experiments that merits more thorough study. It is conceivable, for example, that McDougall's results could be explained

by "trail marking" pheromones deposited by the first generation along the favorable route. A second maze for the offspring experiments, identical in design but pheromone-free, could be used to exclude this possibility. The point here is that Lamarckism is a *testable* concept.

There are numerous examples of a so-called "sire effect" involving heritable genetic transfer. For example, Sobey and Connolly (1986) found that when male rabbits ("bucks") with an acquired immunity to the *Myxomatosis* virus were mated with non-immune female rabbits ("does"), the immunity was passed on to the offspring. An acquired trait was clearly inherited. This "sire effect" also manifested itself when a non-immune buck was mated with a non-immune doe that had previously been mated to an immune buck. Some of the offspring were again born with an immunity to the virus although, presumably, the sperm from the first mating with the immune buck were long gone. Somehow genetic information from the immune buck, deposited into the doe, manifested itself much later in the offspring from the second mating.

Prior to pupating, the willow-moth caterpillar crawls near the tip of a leaf and draws the leaf, beginning with the tip and ending near the stem, around its body. The rolled leaf is kept in place with a web. Fifty years ago a scientist by the name of Harry Schroeder wondered what would happen if the tip of the leaf were removed (Taylor, 1983, pp. 48–49). He found that the caterpillars solved the dilemma by rolling the leaf from side-to-side rather than between the termini. More interesting, Schroeder discovered that 4 of 19 descendants of the side-rolling caterpillars also rolled from the side even when exposed to normal, uncut leaves. It appears as if an acquired behavior had been inherited.

Anway, Cupp, and Uzumcu (2009) reported experiments in which pregnant rats had been transiently exposed to vinclozolin, a fungicide known for its hormonal effects. The male young experienced reduced sperm cell number and viability, resulting in a reduction in fertility. These effects were transferred through the male germ line to nearly all males of the subsequent four generations.

A listing of Lamarckian-type experiments must include those of Gorczynski and Steele (1980). To understand these experiments, one should be aware of P. Medawar's Nobel Prize work carried out three decades previously. Medawar showed that foreign cells injected into a newborn mouse will permit, later in life, acceptance of a graft composed of the same foreign cells. Thus, Medawar was able to graft onto a white mouse a black patch from another mouse after first subjecting the white mouse, while newly born, to the black cells. In other words, early injection of black cells caused white mice to become non-immunogenic toward black-cell grafts later in life.

Gorczynski and Steele (1980) found that 50% of white offspring from graft-tolerant males were also tolerant to black grafts even though the newly born white offspring had, unlike their father, never been exposed to black cells. The second generation of untreated white rats was tolerant to black grafts in 20–40% of the cases. It was concluded that immunity factors in the black cells had been transferred to the germ line (perhaps via viruses) or, stated more simply, an acquired tolerance had been inherited. It should be stated that others have had difficulties reproducing this work, and the resulting debate is still unresolved.

Geneticist T. Sonneborn removed by microsurgery a piece of the cortex (outer surface) of paramecium, a one-celled animal covered by cilia (small hairs) (Beisson & Sonneborn, 1965). The researcher then reinserted the piece after first rotating it 180° from its original position. It was obvious that the piece had been rotated because the paramecium now had a segment of cilia pointing in the "wrong" direction. Remarkably, the offspring of the paramecium also had an inverted row of cilia. The acquired trait had ostensibly been inherited in true Lamarckian fashion.

The parathyroid gland helps maintain calcium levels in the blood. When the gland is removed (a "parathyroidectomy"), calcium levels decline. Fujii (1978) carried out parathyroidectomies on pregnant rats. Their newborn offspring experienced little decline in calcium during the first 24 hours of life even though parathyroidectomies had been carried out on them at birth. In other words, parathyroid removal from the mother rat protected the newborn from the effects of a similar operation. In a control experiment, mothers were not subjected to the operation whereas the offspring were. None of these offspring showed the protection evident in the previous experiment. In the final and most informative experiment, a brother and sister with a parathyroidectomized mother, but who were allowed to keep their thyroids, were mated. The progeny of such unions produced newborn rats with a protective response upon having their parathyroids removed. The effect persisted for four generations, the obvious implication being that an acquired trait, namely protection against parathyroid removal, is inheritable.

J. A. Arai and colleagues (2009) exposed young mice to 2 weeks of an enhanced enrichment program including access to novel toys and elevated levels of social interactions. The program significantly improved the memory and the long-term ability to learn. Importantly, the benefits were inherited by offspring even though the offspring themselves had not been exposed to the enrichment program. Proper controls had been carried out. Thus, offspring of "enriched" mothers were split into two groups, one of which was given to "enriched" foster mothers and the other to "non-enriched" foster mothers. The type of foster mother was found to make no difference. Both groups of offspring profited equally from the stimulating environment experienced by their biological mothers prior to their birth. This seems to be a clear case of heritable adaptation generated by an environmental change.

Victor Jollos (1921) in Germany found that *Paramecium aurelia* developed specific resistance on exposure to arsenic, high salt levels, heat, and antiserum to surface antigens. These resistances (called "Dauermodifikations" or enduring changes) could be transmitted for hundreds of generations, eventually disappearing. Resistance to antiserum may be transmitted via the cytoplasm, but a detailed understanding of the effect at the molecular level was never clarified. Another possibility with the arsenic exposure, for example, is that Jollos was dealing with paramecia having arsenic-resisting genes, already in the genome, amplified by the arsenic. In either case, the environment was dictating the genetics, the essence of Lamarckism.

There have been many other investigations similar to that of Jollos. For example, Sir Cyril Hinshelwood, a Nobel Prize–winning physical chemist, experimented with bacteria that had been grown on sublethal levels of toxic drugs

(Dean & Hinshelwood, 1963). Most, but not all, of the bacteria survived, and the survivors were then repeatedly transferred to fresh media containing the drugs. Hinshelwood observed that the bacteria gradually adapted to the drugs to an extent depending on the number of serial passages to which the bacteria had been exposed. After a sufficient number of passages, 100% of the bacteria survived the drugs. These resistant bacteria were then grown for several generations on drug-free media. When the bacteria were transferred to drug-containing media, they all survived, indicating that the original resistance had been maintained during growth and multiplication in the drug-free media. Hinshelwood concluded that he was observing a heritable adaptive change not unlike that proposed by Lamarck.

Hinshelwood's work drew widespread dismissal because his opponents argued that the adaptations had arisen from spontaneous mutations, in a Darwinian-type mechanism, rather than from heritable environmentally induced effects. Indeed, mutations are frequently invoked as an argument against inherited environmental effects even those originating from diet and other factors usually considered non-mutagenic. Although it is difficult to outright eliminate contributions from mutational events, several features of Hinshelwood's data definitely favor a Lamarckian over a neo-Darwinian mechanism: (a) The doses were sublethal, so this is not a case where all the bacteria are killed off except for a few resistant mutants that continue to multiply. (b) Drugs of diverse structure all manifested the effect, and none of the drugs is known to be mutagenic. (c) Rather than an "all or none" behavior characteristic of many mutations, there was an almost continuous increase in resistance as the number of passages on drug-containing media progressed. (d) Resistance was expressed more quickly throughout the populations than would be expected from rare mutational events. (e) Unlike most mutational behavior, the bacteria regained their original drug-sensitive phenotype after they had been grown for many generations on drug-free media. Gradual reversibility is more adaptive in origin than mutational.

Dias and Ressler (2014) have recently reported in *Nature Neuroscience* on male mice that had been trained to associate, Pavlov-style, the odor of acetophenone with mild foot shocks. The offspring of these mice with unexposed females were fearful of the odor although they had never encountered it previously. The fear response was passed on to the next generation even if they were conceived by artificial insemination using sperm of sensitized mice. As is generally true, it is unclear exactly how information is transferred between generations.

Nature itself has provided evidence that heritable transmission of traits arises from effects other than alterations of DNA nucleotide sequences (i.e., mutations). For example, cells in human embryos differentiate into a variety of phenotypes such as nerve, skin, blood, and bone. Since all these cell types in a given human have identical DNA sequences, there must be a form of cellular inheritance that depends on the interaction of the cells with their environments as opposed to classical DNA-based inheritance. The fact that cells of the gut and bone marrow perpetuate themselves for thousands generations shows that the traits acquired by the differentiated embryonic cells are enduring.

Even given the possibility that one or more of the above sample experiments might be inadequately verified (or even incorrectly interpreted), there is such a

backlog of diverse data suggesting the heritability of acquired traits that it is impossible to ignore the Lamarckian construct. Obviously Lamarck had no idea of the genetics behind the inheritance of acquired traits, just as Darwin had no idea of the genetics behind his survival of the fittest. In the subsequent discussion I will often equate "Lamarckian inheritance" with the more modern term, "epigenetic inheritance" (Jablonka & Lamb 1995, 1998). Accordingly, epigenetics is to Lamarckism as neo-Darwinism is to Darwinism. In other words, epigenetics provides a molecular grounding to the inheritance of acquired characteristics. One might well use the term "neo-Lamarckism" instead of "epigenetics" except that the latter carries less emotional and historical baggage with it.

It should be stated forthwith that epigenetics is not in direct conflict with natural selection. Both models invoke the idea that favorable traits (whether acquired or mutational in origin) can be passed on preferentially to the offspring, thereby perpetuating the trait ("natural selection"). But epigenetic inheritance does provide an additional source of variation, derived from environmental conditions, which is not included in neo-Darwinian theory and the many current off-shoots based on it. The difference between the two constructs is critical. One creates change in response to external stimuli, the other creates change according to random alterations in DNA sequencing. Let us now consider molecular aspects of transgenerational epigenetic inheritance.

EPIGENETICS ("NEO-LAMARCKISM")

Epigenetics is already a well-developed area of biology and only rudimentary aspects can be considered here (Burggren, 2014; Eakin, 2014; Felsenfeld, 2014). The term "epigenetics" (meaning "outside conventional genetics") refers to the generation of heritable variations that originate purely from environmental effects, but classical mutations in DNA sequences play no role. Thus, an environmentally induced phenotype can transmit its respective phenotype to its descendants even when the stimulus that originally triggered the phenotypical variation is no longer present. As alluded to above, this occurs when, for example, a human's fibroblast divides in culture to give a fibroblast, and a keratinocyte divides in culture to a give a keratinocyte.

Perhaps the most straightforward mechanism for inheritance of an acquired trait involves a positive feedback model. Imagine an environmental perturbation that "turns on" a previously silent gene, thereby producing (directly or indirectly) a transcription product that preserves the newly created activity of that same gene. If even low levels of the regulatory product are passed on to daughter cells, the activity of the gene will be inherited. Examples of such self-sustaining regulatory loops acting at the transcriptional and post-transcriptions levels are known. Such behavior must be classified as an epigenetic or neo-Larmarckian adaptation in that an acquired trait has been passed on from generation to generation in the absence of any mutational modifications of DNA sequences. Other transgenerational epigenetic mechanisms have been proposed including noncoding RNA, specific DNA methylation, and chromatin effects (Bonasio, Tu, & Reinberg, 2010). The latter two will be discussed briefly.

Methylation

DNA methylation involves an enzyme-catalyzed insertion of a methyl group into cytosine or, less commonly, into adenine (two of the four DNA bases). Methylation is an important component of many cellular processes, particularly epigenetic signaling that locks genes in an "off" position. For example, DNA methylation suppresses the expression viral genes that have become incorporated into a host's genome. Abnormal hypermethylation can lead to certain cancers owing to silencing of tumor-suppressor genes. DNA methylation directs cell differentiation during embryonic development as well as the ability of these differentiated cells to reproduce with high fidelity. How does this work? Assume that a replicating DNA chain has a methylated cytosine (^{Me}C) with a guanine (G) neighbor (i.e., a ^{Me}C•G pair within the DNA chain). DNA replication produces a second chain that incorporates an non-methylated G•C pair (i.e., ^{Me}C generates G, and G generates C in concert with the usual C/G affinity).

It is at this point that a methyl is introduced into the new G•C unit to give a G•^{Me}C pair, and in this manner the methylation pattern of the original DNA stand is maintained. The important point here is that this mechanism of information transfer occurs independently from an actual sequence mutation. Modification of the DNA sequence occurs only after the DNA molecule has been synthesized. If some sort of environmental perturbation causes a change in the rate or location of methylation, a new inherited phenotype is possible. Lamarck's theory is, in summary, founded on chemistry. Note that a significant number of genes in sperm are known to escape epigenetic reprogramming, which scrubs the DNA free of methylation (Greer et al., 2011).

Chromatin

Chromosomes are not merely strands of DNA. Chromosomes are an elaborate complex of DNA, protein, and RNA called chromatin. Thus, DNA is wrapped around a core of so-called histone proteins to form nucleosomes, the basic subunit of all chromatin. In addition there are a great variety of non-histone proteins that bind directly to the DNA and that include enzymes (e.g., RNA polymerase) as well as proteins that control higher-order structure (e.g., organization within the chromosomes).

The whole business is extremely complex. Suffice it to say that DNA-associated proteins affecting the maintenance of gene activity can be carried from one cell generation to the next. Inheritance of the appropriate state of DNA activity by daughter cells is not fully understood. It is not hard to imagine, however, that environmental changes can induce, and in some cases direct, changes in the copying system. Epigenetic variants lead potentially to adaptive progress at a rate far greater than that accompanying random and generally harmful DNA mutations. This claim arises from the fact that, whereas a given mutation is seldom reproduced concurrently in multiple cells or organisms, this is not true of epigenetic variations induced by environmental changes. The speed and efficiency of epigenetics are the very same attributes that appealed to Lamarck when developing his idea that acquired characteristics can be inherited.

It is quite possible that new entirely mechanisms of epigenetic information transfer await future discovery.

EVOLUTION

Although the idea that evolution and epigenetics are related is by no means new, it is still in a state of infancy. For example, Diez, Roessler, and Gaut (2014), in a paper entitled "Epigenetics and Plant Genome Evolution," wrote that epigenetics was envisioned as a topic to inform evolutionary theory, but the interplay between epigenetics and evolution has received little attention. While Duncan, Gluckman, and Dearden (2014), in a paper entitled "Epigenetics, Plasticity, and Evolution. How do We Link Epigenetic Change to Phenotype?" wrote that while we are beginning to understand how these [epigenetic] mechanisms have roles in human biology and disease, we have little understanding of their roles and impacts on ecology and evolution. Hernando-Herraez et al. (2013) in a paper entitled "Dynamics of DNA Methylation in Recent Human and Great Ape Evolution" wrote that the dynamics of DNA methylation changes between humans and their closest relatives are still poorly understood. And finally C. C. Ledon-Rettig (2013) wrote in a paper introducing a symposium that the role of epigenetic variation and inheritance in natural populations remains poorly understood. The point of citing these quotes is to show that evolution via epigenetics, while the focus of increasing attention, remains an undeveloped field (Donohue 2014; Handel & Ramagopalan, 2010) and is thus legitimately open to the grounded theorizing that will appear here next.

HUMAN INTELLIGENCE

This section deals with the application of neo-Lamarckism to the evolution of human intelligence. The topic was selected for two reasons: (a) The origin of our intelligence is one of the great unsolved mysteries in biology; (b) The origin of human intelligence has been a particular interest to me, leading to a book entitled *The Thin Bone Vault. The Origin of Human Intelligence* (Menger, 2009). The following text has been adapted from the book. As will be seen, the arguments are based mainly on common knowledge culminating with three speculative but far-reaching axioms. A vast library is available to those desiring more traditional views of evolution (Ehrlich, 2000; Hawkins, 2004; Jablonka & Lamb, 2005; Margulis & Sagan, 2002; Pigliucci & Muller, 2010; Richards, 1987; Richardson, 2000; Rose, 2005; Skoyles & Sagan, 2002; Sternberg & Kaufman, 2002; Stringer 2012). Again let me emphasize that there is no dispute here with natural selection; the difference lies only in the additional environmental source of heritable variation. Yet this difference, as we will see, has critically important implications.

If our ancient ancestors owned a modern and highly energy-expensive but largely underutilized intelligence, then a neo-Darwinian explanation for the human brain fails, and fails badly, because random natural selection provides no foresight, no plan. Neo-Darwinian evolution could not have foreseen the eventual need for the human brain to, for example, write computer programs. If, on

the other hand, intelligence evolved only in modern times, then neo-Darwinism must be side-stepped with equal determination because natural selection claims that organs of complexity, such as the brain, evolved in minute steps over excruciatingly long time periods. Either way neo-Darwinism faces a serious predicament.

Here is a summary of the main difficulty with a neo-Darwinian explanation of human intelligence: *How can a large set of energy-costly genes, each single member of which had seemingly little benefit when first created, all gather into a permanent existence within a short time period in every member of a small population (that was dispersed and geographically isolated over a huge planet) who had a low reproductive output, a low rate of beneficial mutations, and a low level of genetic contact?*

I now expand briefly on each of these evolutionary parameters to help the reader grasp the magnitude of the problem:

1. "C*omplex set of genes"*: Fourteen genes control eye color in fruit flies. Many hundreds of genes are thought to impact human odor detection. It is not known how many genes operate in concert to create neural networks in the brain, but it must be a large number.
2. "*Expensive traits"*: About 20% of our energy consumption is devoted to the brain. Any such energy-costly trait would get diminished or outright eliminated if it were not fully utilized.
3. "*Little immediate benefit when first created"*: Written vowel-centers in the brain; the ability to solve differential equations; and the hand–eye–ear–brain coordination needed to play the violin were, for example, of no direct benefit to our hunter-gatherer ancestors who lived prior to the advent of agriculture 10,000 years ago. Yet currently the traits are commonplace.
4. "*Permanent existence"*: Our permanent acquisition of intelligence is self-evident.
5. "*Short time period"*: Modern humans (Cro-Magnons) appeared only abut 40,000 years ago. It is not clear whether humanoids prior to the Cro-Magnons even had a vocal apparatus to speak.
6. *"Every human"*: Uniform intelligence distributions are found throughout the world. Whatever mechanism is proposed for the trait's evolution, the mechanism must explain a "perfect mixing" (meaning that our large and complex set of intelligence genes have become distributed to everyone). Neo-Darwinism has not satisfactorily explained what competitive interactions gave rise to a uniform worldwide spread of multiple mutations among populations that had presumably never come into genetic contact.
7. "*Small population"*: It is estimated that 25,000 years ago the worldwide population was only about 3 million. In the midst of the ice age, the number of humans may have totaled only in the thousands and were in perilous danger of going extinct.
8. "*Widely dispersed"*: Distributing a mere 3 million humans across all continents except the Americas and Antarctica must obviously have limited reproductive interactions, thereby rendering "perfect mixing" of multiple

intelligence genes, formed individually across the globe, a challenging prospect for neo-Darwinism.

9. "*Low reproductive output*": According to fossil records, perhaps 50% of the population was dead before the age of 14. Disease, starvation, cold weather, homicidal raids, and infanticide all took their toll. Early death in a small population restricts, of course, the dissemination of genes.
10. "*Low mutation rate*": Humans are generally harmed, not benefitted, by mutational assaults. For example, mutations induced by X-rays or nuclear radiation often lead to cancer and other maladies. Even if a rare beneficial mutation does appear, it can often be removed by our well-developed repair mechanisms, thus limiting the potential for DNA-based adaptation and, by the same token, restricting neo-Darwinian evolution.

None of the parameters, be they genetic or demographic, is favorable to the elements of neo-Darwinism.

THE PROPOSAL

I propose that, when *Homo erectus* departed from Africa and expanded into Europe and Asia, humans were already endowed with silent intelligence-related genes that were buried among a vast number of other unexpressed genes. This immediately explains why all humans are similar in intelligence—useful DNA combinations were present from the beginning of early humans. The presence of large numbers of masked genes in our early DNA library was fortuitous. Neo-Darwinism is, of course, likewise predicated on fortuity, and in this sense the two models are analogous. Only much later were genes unmasked by environmental or cultural ("epigenetic") changes. "Junk" DNA, comprising the great majority of human DNA, may be regarded as remnants of these early times.

Note that the intelligence genes, being initially silent, cost little by way of energy and therefore survived rapid elimination that would normally accompany expensive but unused traits. As *Homo erectus* evolved into *Homo sapiens*, masked intelligence genes (among our DNA's huge collection of hidden genes) were being continuously unmasked. In other words, concurrently with their cultural advances, humans were taking more and more advantage of their long-held, but underutilized, mental capacity. This is purely a neo-Lamarckian mechanism devoid of neo-Darwinian mutations. Unlike what happens in neo-Darwinism, the development of human intelligence can be comparatively rapid because it occurred independently among multiple individuals worldwide who were exposed to common epigenetic stimuli. The perplexing problem of explaining worldwide uniformity among rare beneficial DNA mutations as required by neo-Darwinism is thereby precluded.

The preceding mechanism proposes that improvements in intelligence are related to environmentally based gene unmasking. Thus, when prehistoric humans learned new mental-based skills, there were chemically based epigenetic effects that facilitated the learning process in the next generation. In this manner, acquired neural connections associated with new skills did not need to be totally re-established, over and over again, with each new generation. Note that neo-

Darwinism and neo-Lamarckism embody selection processes as a common thread. Where they differ critically is in the mechanism of macro-evolutionary change prior to selection: mutation versus gene unmasking, respectively.

This leads to a "*cranial feedback*" theory for the evolution of intelligence based on three axioms:

Axiom #1. Mental activity stimulates the production of chemical messengers in the brain.
Axiom #2. Brain and germ cell tissues interact by means of circulating chemical messengers such as peptides and nucleic acids.
Axiom #3. A silent gene related to mental acuity within the germ cell is turned on by the brain's messenger and passed on to the next generation.

Although the three axioms are speculative, each of them has experimental support described by Menger (2009), and none of them violates current biochemical precepts. To give one example, axiom #2 is supported by the observation that circulating nucleic acids can induce soma-to-germ genetic transformations (Anker & Stroun, 2012). The main point here is that a newly "acquired" mental competence appeared on the scene as a result of environmental stimuli and not as the result of random, disperse, and rarely beneficial DNA mutations. Thus, cultural factors, including but by no means limited to universal education, should continue to activate a genome poised to expand our intellect. We are inherently smarter now than in prehistoric times because of an epigenetic awakening of our genes.

Brief mention should be made of the classic "Weismann barrier," which has been used to counter Lamarckian constructs. The Weismann barrier postulates that hereditary information passes from the reproductive cells (germline cells) to the body cells (somatic cells), but the reverse *never* occurs (Sabour & Schöler, 2012). The essence of the preceding article is now stated simply in a few words: The Weismann barrier can indeed be crossed. When this happens, the oft-compared Nature and Nurture mechanisms fuse into a single entity.

SUPPORTING EVIDENCE

The most important question in science, "What is the evidence?," must, of course, be applied to the cranial feedback theory of human evolution. Although hard evidence is as yet lacking, there are numerous observations pointing directly to its plausibility. Some of these are now cited:

1. It has been shown that IQ, worldwide, has increased substantially in recent years, a phenomenon referred to as the "Flynn effect" (Flynn 1984). In as much as IQ measures intelligence, people are getting measurably smarter in recent decades owing in part, one surmises, to improved and more widespread education.
2. Just recently Rachel Yehuda at Mt. Sinai found that genetic changes from trauma suffered by Holocaust survivors are capable of being passed on to their

children (Yehuda et al., 2014). One can ask for no clearer sign that life experiences of humans, not just their genetic makeup, affect subsequent generations.

3. British and Swedish researchers studied carefully documented historical records from Överkalix, an isolated community in northern Sweden (Pembrey et al., 2006). They found that the paternal grandfathers' food supply (i.e., whether or not there was a famine during their pre-puberty period) was closely linked to the mortality of the grandsons (but not the granddaughters). It was concluded that the environment might be able to modify the germline. In other words, exposure of a group of men to a particular diet had a profound effect on the viability (disease resistance, etc.) of men living two generations downstream. Since diet is normally considered an environmental factor, inheritance of the viability trait must be Lamarckian (i.e., acquired) rather than the result of diet-induced neo-Darwinian-style mutations. It is not clear at this point for how many generations the diet effect persists.
4. In a paper entitled "Obesity and Bariatric Surgery Drive Epigenetic Variation of Spermatozoa in Humans" (Donkin et al., 2016) it was shown that obese and lean men have different epigenetic patterns in their sperm. When obese men underwent bariatric surgery to reduce weight, their sperm's methylation patterns trended toward the configuration of lean men. Clearly, genetic changes can be reversible depending on the environment. This work suggests that obese men might want to lose weight before conceiving a child and, thereby, impart to their offspring favorable DNA methylation. One is reminded here again of Darwin's advice to young women to learn as much as they can prior to raising families.
5. Cambodian people traumatized during the Khmer Rouge genocide tended to have children with depression and anxiety. Children of Vietnam War veterans in Australia have higher suicide rates than the general population. Obviously, the trauma's effect derives in part from social factors including how the parents might have treated their children. But a recent study in *Nature* finds that stress in early life alters the production of small RNAs, called microRNAs, in the sperm of mice (Hughes 2014). Both depressive behaviors and glitches in metabolism persist in the progeny. Human sperm likewise experience subtle environmentally based changes in sperm micro-RNAs that set the stage for a huge plethora of other effects.
6. A team of researchers from Spain, France, and the United States reported evidence for down-regulation of gene expression following a period of "mindfulness meditation" (i.e., eyes closed, sitting on crossed legs, focusing on breath) (Creswell et al. 2012; Kaliman et al., 2014). Thus, epigenetic changes in several genes were observed following meditation, changes that were not seen in a group engaged in quiet activities other than mediation. Meditators were able to recover faster from stressful situations such as public speaking and carrying out mental calculations. Richard J. Davidson, one of the authors, wrote that our genes are quite dynamic in their expression and these results suggest that the calmness of our mind can actually have a potential influence on their expression.

If diet, trauma, obesity, and meditation can impact our genes, is it far-fetched to postulate that brain chemistry can do likewise?

RELEVANCE TO EDUCATION

The human race will, averaged over the entire population, become smarter and smarter until some unknown inherent ceiling is reached. And this process should be aided, in part, by educational systems that challenge children to learn, think, and create. Of course, intelligence, even genius, can appear from all quarters, educated or otherwise, because an enhanced mental potential can also have non-epigenetic origins (e.g., a favorable combination of parents' genes). Clearly, epigenetics supplements, not supplants, classical genetics. But epigenetics has important advantages over classical genetics in that epigenetics responds directly to specific needs and, compared to mutational events, epigenetic changes can occur more quickly.

If education is one of the many mechanisms that catalyze an epigenetic release via the cranial feedback mechanism, then there exists an important conclusion that society should keep in mind: Comfort can be taken in knowing that teachers are molding the intelligence not only of the current generation but of future ones as well. Since universal education has just begun, relative to our evolutionary timescale, its consequences will likely become more evident as time passes. In any event, neo-Lamarckian effects are yet another reason for establishing high-quality educational systems as a prime goal for all of humanity, and this is a good thought with which to end this article.

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