



Human nature or human natures?

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ABSTRACT

Most evolutionary psychologists share a belief in one key concept: the environment of evolutionary adaptedness (EEA), i.e., the ancestral environment that shaped the heritable mental and behavioral traits of present-day humans. It is usually placed in the African savannah of the Pleistocene, long before our ancestors began to spread to other continents some fifty thousand years ago. Thus, later environments have not given rise to new traits through genetic evolution.

This belief rests on two arguments: 1) such traits are complex and therefore evolve too slowly to have substantially changed over the past fifty thousand years; 2) because the same time frame has seen our species diversify into many environments, recent traits should tend to be environment-specific and hence population-specific, yet such specificity seems inconsistent with the high genetic overlap among human populations. Both arguments are weaker than they seem. New complex traits can arise over a relatively short time through additions, deletions, or modifications to existing complex traits, and genetic overlap can be considerable even between species that are morphologically, behaviorally, and physiologically distinct.

There is thus no conceptual barrier to the existence of EEAs in post-Pleistocene times. Such a paradigm could shed light on such research topics as the visual word form area, reproductive strategy, predisposition to violence among young men, and personality traits. Eventually, a multi-EEA model may dominate evolutionary psychology, perhaps after an interim period of accommodation with the current model.

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1. The current model

In evolutionary psychology, one key concept has been the 'environment of evolutionary adaptedness' (EEA). This is the ancestral environment that presumably made us what we are today. It is usually placed in the African savannah of the Pleistocene, long before modern humans began to spread to other continents some fifty thousand years ago. Proponents notably include John Tooby and Leda Cosmides:

It is no more plausible to believe that whole new mental organs could evolve since the Pleistocene—i.e., over historical time—than it is to believe that whole new physical organs such as eyes would evolve over brief spans. It is easily imaginable that such things as the population mean retinal sensitivity might modestly shift over historical time, and similarly minor modifications might have been made in various psychological mechanisms. However, major and intricate changes in innately specified information-processing procedures present in human psychological mechanisms do not seem likely to have taken place over brief spans of historical time.

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... For these and other reasons, the complex architecture of the human psyche can be expected to have assumed approximately modern form during the Pleistocene, in the process of adapting to Pleistocene conditions, and to have undergone only minor modifications since then [1, p. 34].

Recently, Tooby and Cosmides have softened their stand: “Although the hominid line is thought to have originated on edges of the African savannahs, the EEA is not a particular place or time” [2, p. 22]. It is a composite of whichever selection pressures brought each adaptation into existence [2]. There are thus potentially as many EEAs as there are adaptations, and some may be later than others.

1.1. Problem of complexity

How much later? Tooby and Cosmides consider one limiting factor to be complexity. The more complex the adaptation, the more genes it involves, and the more time needed to make all of the right changes to all of the right genes. Therefore, recent evolution has created only simple traits, and certainly nothing as complex as mental or behavioral ones [1].

This argument assumes that complex traits evolve directly from simple origins. Actually, they almost always arise through additions, deletions, or modifications to existing complex traits. As Henry Harpending and Gregory Cochran point out:

Even if 40 or 50 thousand years were too short a time for the evolutionary development of a truly new and highly complex mental adaptation, which is by no means certain, it is certainly long enough for some groups to lose such an adaptation, for some groups to develop a highly exaggerated version of an adaptation, or for changes in the triggers or timing of that adaptation to evolve. That is what we see in domesticated dogs, for example, who have entirely lost certain key behavioral adaptations of wolves such as paternal investment. Other wolf behaviors have been exaggerated or distorted [3, p. 10–11].

1.2. Problem of apparent panmixia

There is another argument against recent evolution of mental or behavioral traits. Because the past fifty thousand years have seen our species diversify into a wide range of environments, recent traits would tend to be adaptive in some environments but not in others. And their underlying genetic variants would tend to proliferate in some populations but not in others. Yet such population specificity seems impossible. At almost any genetic marker (blood types, serum proteins, enzymes, mtDNA, etc.), a typical gene varies much more within than between human populations. And this is true not only for large continental populations but also for small local ones. The geneticist Richard Lewontin concluded that 85% of our genetic variation exists only among individuals and not between ‘races’ [4].

For Tooby and Cosmides, these findings render “implausible the notion that different humans have fundamentally different and competing cognitive programs” [5, p. 30]:

Human groups do not differ substantially in the types of genes found, but instead only in the relative proportions of those alleles. . . . What this means is that the average genetic difference between one Peruvian farmer and his neighbor, or one Bornean horticulturist and her best friend, or one Swiss villager and his neighbor, is 12 times greater than the difference between the “average genotype” of the Swiss population and the “average genotype” of the Peruvian population (i.e., the within-group variance is 12 times greater than the between-group variance) [5, p. 35].

This is true but does not mean what one might think. The same genetic overlap exists not only between populations of one species, like our own, but also between related species, like canids. “[U]sing genetic and biochemical methods, researchers have shown domestic dogs to be virtually identical in many respects to other members of the genus. . . . there is less mtDNA difference between dogs, wolves and coyotes than there is between the various ethnic groups of human beings, which are recognized as belonging to a single species” [6, p. 32–33].

Many other examples could be cited. In the deer family, we see more genetic variability within some species than between some genera [7]. Some masked shrew populations are genetically closer to prairie shrews than they are to other masked shrews [8]. Only a minority of mallards cluster together on an mtDNA tree, the rest being scattered among black ducks [9]. All six species of Darwin’s ground finches seem to form a genetically homogeneous genus with very little concordance between mtDNA, nuclear DNA, and morphology [10]. In terms of genetic distance, redpoll finches from one species are not significantly closer to each other than are redpolls from different species [11]. Among the haplochromine cichlids of Lake Victoria, it is extremely difficult to find interspecies differences in either nuclear or mitochondrial genes, even though these fishes are well differentiated morphologically and behaviorally [12]. Neither mtDNA nor allozyme alleles distinguish the various species of *Lycaedis* butterflies, despite clear differences in morphology [13]. An extreme example is a dog tumor that spreads to other dogs through sexual contact: canine transmissible venereal sarcoma (CTVS). It looks and acts like an infectious microbe, yet its genes would reveal a canid and conceivably some beagles may be genetically closer to it than to Great Danes [14].

In sum, total genetic variation poorly mirrors genetic variation in adaptive traits, be they morphological, behavioral, or physiological. Keep in mind that a new species typically arises when a founder group buds off from a parent population and enters a new environment with new selection pressures. The new selection pressures, however, will leave most of its genome unchanged. In some cases, this is because the gene itself has little adaptive value (e.g., most genetic markers), often

being no more than 'junk DNA'. In other cases, the gene's variants are equally adaptive in a variety of organisms. Many blood polymorphisms span not only different species but even different genera. In terms of the ABO system, for instance, a person may have more in common with some apes than with other people [12].

Of course, once the two populations have become reproductively isolated, they no longer accumulate the same mutations and will drift apart at all gene loci, including the many that weakly respond to natural selection. But this process is slow. For example, redpoll finches diverged into two species some fifty thousand years ago and have distinct phenotypes, yet their mitochondrial DNA shows a single undifferentiated gene pool [11]. The past ten thousand years have seen dogs diverge into distinct breeds, which nonetheless cannot be told apart by genetic markers. In fact, greater mtDNA differences exist within the single breeds of Doberman pinscher or poodle than between dogs and wolves [6].

One might argue that humans have artificially created dog breeds by using limited criteria that involve a small set of genes. This objection is not wholly true. Many breeds, such as dingoes, originated in prehistory long before kennel clubs. More to the point, if one argues that artificial selection acts on relatively few genes, it does not follow that natural selection acts on the whole genome. In fact, we are still looking at a small set of genes, a larger one than what dog breeders use, but still much smaller than the genome. It is no surprise, then, that human populations overlap so much genetically. They began to move apart only some fifty thousand years ago.

2. Building a new model: gene-culture coevolution

So let us reframe the question. How much do human populations differ from each other because of real adaptive differences due to natural selection? The jury is still out but an answer is taking shape. A team led by anthropologist John Hawks estimates that natural selection has altered at least 7% of our genome over the last forty thousand years. This period saw modern humans spread from Africa to other continents, thus forming the different populations we know today. Furthermore, again according to Hawks et al., natural selection has been altering our genome at an accelerating rate, particularly after agriculture replaced hunting and gathering less than ten thousand years ago. The rate of genetic change may have then risen over a hundred-fold [15].

We still poorly understand these recent changes to the genome. John Hawks sees adaptations to new ecological and cultural environments, specifically to colder climates, to an agricultural diet (cereals, milk, etc.), to diseases associated with the spread of agriculture (smallpox, malaria, yellow fever, typhus, cholera), and to forms of "communication, social interactions, and creativity" [15]. Indeed, given that the human genome has changed mainly over the past ten thousand years, we are probably looking at adaptations to new cultural environments.

John Hawks is not the first to give culture a role in human evolution. So have such people as Pierre van den Berghe, Charles Lumsden, E.O. Wilson, Robert Boyd, and Peter J. Richardson [16–18]. This paradigm, usually called gene-culture coevolution, has nonetheless remained marginal partly because of the influence of John Tooby and Leda Cosmides and partly because of two obstacles to research:

1. The linkages between genes and culture tend to be remote, indirect, multiple, and complex. Some are fairly straightforward, like the one between lactose tolerance and milk consumption, but such linkages are atypical.
2. With a few minor exceptions, gene-culture coevolution is specific to humans. Cross-species comparisons, so common elsewhere in evolutionary study, are of little help [16].

These obstacles are not insuperable. To some degree, they reflect a wish to study humans with the same research tools we use to study other species. But there is no reason why we cannot develop other tools or borrow them from psychology, sociology, and anthropology. Methodology alone should be no barrier.

There remains, however, a conceptual barrier: the single-EEA model and its dominant status within evolutionary psychology. This barrier can be negotiated at some cost of theoretical dissonance, for example by claiming to believe in a single EEA while arguing for population-specific 'fine-tuning.' Such dissonance could settle into a relatively stable regime of data interpretation, at least over the short term. Over the longer term, however, efforts to reconcile data with theory will likely become more and more perfunctory and eventually cease altogether. Only then, and long after it has ceased to guide research, will the single-EEA model disappear from scientific discourse. Only then will its theoretical impossibility become apparent to many researchers and most educated laypeople.

For now, this conceptual shift will be driven not so much by overt changes in scientific discourse as by an accumulation of data that cannot easily fit the single-EEA model. Such data are accumulating in several fields of research, including four to be discussed here: a) *ASPM* and the visual word form area; b) reproductive strategy; c) predisposition to violence among young men; and d) personality traits. In all four fields, the data increasingly challenge the existence of a single species-wide human nature.

2.1. *ASPM* and the visual word form area (VWFA)

ASPM is a gene implicated in the regulation of primate brain growth. In humans, a new variant arose some six thousand years ago, apparently somewhere in the Middle East. It then spread outward, becoming more prevalent in the Middle East (37–52% incidence) and Europe (38–50%) than in East Asia (0–25%) [19].

This pattern matches the spread of alphabetical writing [20]. Writing emerged near the end of the fourth millennium BC in the Middle East, initially with pictorial characters that represented ideas rather than sounds. This ideographic system gradually became phonetic in the third to second millennia BC and ultimately developed into an alphabet that spread to Europe, North Africa, and the Indian subcontinent. An ideographic system independently emerged in East Asia, perhaps in the second millennium BC, but never gave way to an alphabet, except in Korea some six hundred years ago.

Although an alphabet has fewer characters and is easier to learn, it demands more from short-term memory. Longer character strings must be remembered and more transformations performed in mental space. In contrast, ideographic systems seem to evoke meaning faster, apparently because the mind encodes the characters visually and maps them onto meanings directly [21–24].

In antiquity, alphabetical script demanded even more mental effort. Reading would cause fatigue because characters formed a continuous stream with little or no punctuation. Writing was no less tiresome because speech had to be transcribed manually in real time. Texts also needed frequent copying, likewise by hand, given the deterioration of parchment and papyrus in warm climates. Today, all of these tasks are much easier or simply unneeded. Reading is facilitated by word, sentence, and paragraph breaks. Stenography is seldom done in real time, thanks to dictaphones and other recording machines. Copying has been largely automated by printers, scanners, photocopiers, fax machines, cut-and-paste functions, and so forth.

Thus, ancient reading and writing tested the limits of human ability, particularly for the various scribes who processed texts day in and day out, i.e., clerks, stenographers, copyists, secretaries, notaries, and calligraphers. Their skills were honored in the eulogy ‘Praise of the scribe’ (*Book of Sirach*, 2nd century BC):

Many will praise his understanding; it will never be blotted out.
His memory will not disappear, and his name will live through all generations.
Nations will speak of his wisdom, and the congregation will proclaim his praise.
If he lives long, he will leave a name greater than a thousand [25].

In the ancient world, ‘leaving a great name’ did not mean being written about by historians but rather having many illustrious children to carry on the family name long after death. Scribes thus seem to have enjoyed high reproductive success. In addition, their offspring often entered the same profession, thereby creating a quasi-hereditary caste as seen in the many Mesopotamian scribes who claimed descent from the editor of the *Epic of Gilgamesh* [26]. Such individuals may have been vectors for genetic factors that facilitate reading and writing, including perhaps the new *ASPM* variant.

Of course, not all offspring could become scribes, given the limited number of positions in the public administration and as personal secretaries to the wealthy. Their talents were put to other uses, thereby raising the intellectual tenor of society. Indeed, ancient philosophers used analytical tools that scribes had earlier developed for legal and administrative texts: statement of the problem; presentation of the argument and counter-argument; review of the literature and inventory of relevant facts; quotations from authorities; and so on [27].

All of this assumes the existence of heritable aptitudes for reading and writing. Such an assumption is at odds with the view, held by many psychologists, that cognition displays heritable variation only for general intelligence (commonly referred to as *g*). This view helped dry up interest in *ASPM* when its variants showed no significant correlation with IQ or brain size [28,29].

Recently, however, it has been found that *ASPM* variants correlate not with total brain size but with growth of specific brain tissues, especially within the cerebral cortex. After examining *ASPM* variants in different primate species, the authors of a comparative study concluded: “different brain parts still have their own evolutionary and functional differentiation with unique genetic bases” [30, p. 6]. Researchers have also found a specific brain region for processing of alphabetical characters. Named the visual word form area, it seems to be a patchy population of neurons in the left posterior occipitotemporal sulcus [31]. Its very existence raises a question:

... why is there a reproducible cortical site responsive to visual words? Reading is a recent cultural activity of the human species. The 5400 years that have elapsed since its invention are too short to permit the evolution of dedicated biological mechanisms for learning to read [31, p. 473].

How did this specialized brain area evolve so rapidly? This would be puzzling if adaptations came together from scratch, but evolution seldom works this way. Typically, they arise from adaptations that served another purpose. The VWFA may have evolved from neurons that assist face recognition, and such neurons actually do exist here and there in this brain area [31].

A second puzzle is of the chicken and egg sort. If this brain area is crucial for reading and writing, how did humans first become literate? This, too, would be puzzling only if the VWFA were required for any degree of literacy. In fact, lesion studies have shown that some ability to read and write can return after loss of the VWFA [32]. Humans may have thus acquired reading and writing by initially pushing the limits of mental plasticity, perhaps by using face-recognition neurons. Natural selection would have then favored individuals who could perform the task better, the eventual result being a specialized brain area. In short, the VWFA did not arise to make literacy possible. It arose to make literacy easier.

A third puzzle: How could the VWFA exist universally within our species when human populations differ so much in writing system (phonetic or ideographic), in degree of literacy, and in historical experience with writing? Actually, this brain area does vary within our species. An MRI study indicates that Chinese subjects process ideographic characters not only in the VWFA but also in other regions, notably the left middle frontal cortex [33]. Such a finding again suggests that ideographic writing does not impose the same cognitive load that phonetic writing imposes [21–24].

In the future, researchers may extend MRI studies to populations that have acquired reading and writing only in recent times, thus allowing us to retrace the evolution of the VWFA from a group of neurons that originally served another use. This brain area should also be investigated in relation to *ASPM* variants. Does the most recent variant improve VWFA functioning under premodern conditions of reading and writing? Subjects could, for instance, be coded for their *ASPM* variant and then tested for ability to transcribe speech or text over a long time.

Finally, genetic researchers could team up with historians and archaeologists to study the prevalence of various *ASPM* variants over time and space in response to cultural change, such as the advent of alphabetical literacy, the rise of a caste of scribes who specialize in reading and writing long texts, and the growing need by the public to read short texts (signs, notices, graffiti).

2.2. Reproductive strategy

It has been argued that children develop differently if the biological father is absent and a strange male is present (e.g., a stepfather). In both sexes, sexual activity will begin earlier with less stable pair bonds. Sons will show hypermasculine behavior, such as aggressive acting out, boasting, and risk-taking. Daughters will reach puberty earlier and judge potential mates by current appearance and status in the male hierarchy rather than by steadfastness and ability to support a family. It has thus been hypothesized that an early sensitive period allows certain environmental cues, like father presence, to define reproductive strategy later in life [34–38].

To study the effects of parental absence, the psychologist Michele Surbey recruited a large sample (1247 daughters) and measured several possible confounding factors: family size, birth order, weight, height, body mass index, and socio-economic status (SES). On none of the factors did father-absent daughters significantly differ from father-present ones. Nonetheless, they had sexually matured four to five months earlier than daughters with both parents and seven months earlier than mother-absent daughters [35].

One may still ask whether father absence was a proxy for some other confounding factor. How well had SES really been controlled? The subjects were apparently university students and likely shared the SES of their mothers. But what do we know about their absent fathers? And did the above factors fully capture the differences between father-absent daughters and father-present ones? Could the two groups still differ physiologically and, perhaps, genetically?

To control for genetic background, a team led by Jane Mendle examined the daughters of twin mothers where one mother was still living with the biological father and the other was not. For each pair of mothers, the daughters of one had the same average age of menarche as did the daughters of the other, despite different parental environments. Age of menarche was also the same when all daughters living with stepfathers were compared with all daughters living with biological fathers, as long as mother's age of menarche was controlled. Thus, early menarche was predicted as strongly by a step-uncle's presence as by a stepfather's. "It does not seem necessary for a child to experience the direct environmental influence of a stepfather to exhibit an accelerated age of menarche—as long as she is genetically related to someone who does have a stepfather" [39, p. 539].

These findings have support from other authors. A longitudinal study found that a daughter's pubertal development was better predicted by her mother's age of menarche than by family environment [40]. A common genetic causation is also suggested by a class of X-linked androgen receptor alleles that is linked in males to aggression, impulsivity, sexual compulsivity, and lifetime number of sex partners and in females to paternal divorce, father absence, and early menarche [41].

Mendle et al. also found a weaker correlation between father absence and early menarche in their Australian sample than in the American samples of other researchers. Were ethnic differences responsible?

The wholly Caucasian population of our Australian sample may explain our failure to replicate the strong father-absence association observed in more ethnically diverse American samples. Given that African American and Latina girls experience menarche on average 6 months prior to Caucasians (Herman-Giddens et al., 1997), it may be that the previously established associations between early menarche and lack of a traditional two-parent family structure are affected by racial differences in family structure [39, p. 539–540].

Why did the other studies fail to control for ethnicity? Apparently because SES controls were felt to be sufficient. This does seem true with respect to the Hispanic-American/Euro-American difference in age of menarche, which disappears when samples are controlled for socio-economic status [42]. On the other hand, since Hispanic-American SES correlates with European ancestry, this control would bias the sample population toward individuals who genetically resemble Euro-Americans.

In the future, researchers will probably try to confirm Mendle et al.'s findings by studying twins reared apart. Efforts should also be made to elucidate the common cause behind father absence and early menarche. Is it a gene that influences both behavior and physiology? Or has a certain kind of social environment favored early maturing girls precisely because the father will probably not stay around and provide for them while they are still dependent?

We may eventually see father absence and early menarche not as cause and effect but rather as aspects of a reproductive strategy of low paternal investment and high maternal investment, i.e., the man maximizes his number of mates while minimizing assistance to each of them and their children; conversely, the woman provides for herself and her children without male assistance. Such female autonomy is not possible in all environments, particularly those where a woman

cannot gather or grow food year-round. The best strategy will then be to extract enough resources for herself and her offspring from a male provider. Although either strategy is initially pursued for lack of better alternatives, natural selection will over time strengthen whichever genetic predispositions favor the optimal one.

Reproductive strategy is no less an adaptation to our environment than lung capacity or body shape. Since this behavioral adaptation varies among human populations, as evidenced by the relative importance of monogamy versus polygyny or of paternal investment versus maternal investment, humans should also vary in such aspects as father presence and age of menarche.

2.3. Predisposition to violence among young men

Aggressive/antisocial behavior seems to be moderately to highly heritable among males. A heritability of 40% was estimated through meta-analysis of 51 twin and adoption studies [43]. A later twin study found a heritability of 96%, the subjects being 9–10 year-olds from diverse ethnic backgrounds [44]. This higher figure reflects the closer ages of the subjects and the use of a panel of evaluators to rate each of them. In the latest twin study, the heritability was 40% when the twins had different evaluators and 69% when they had the same evaluator [45].

This heritable predisposition is more adaptive in some societies than in others. Among the Yanomamö, a horticulturalist people of Amazonia, significantly more children are fathered by men who have committed homicide than by those who have not [46]. Among the Ache, a hunter-gatherer people of Paraguay, 'homicidal' men do not have more offspring but more of their offspring survive, either because strong fathers can better protect their children or because some other factor makes both father and offspring healthier than average [47].

In contrast, aggressive males are penalized in societies where central authority has monopolized the use of violence. The historical economist Gregory Clark argues that such societies have lower rates of violent death for all causes, including war. He points to England, where imposition of central authority led to a steady fall in male homicide from the twelfth century to the early nineteenth. Meanwhile, there was a parallel decline in blood sports and other forms of exhibitionist violence (cock fighting, bear and bull baiting, public executions) that nonetheless remained legal throughout this period. Clark ascribes this change to the reproductive success of upper- and middle-class individuals who differed statistically in their behavioral predispositions from the much larger lower class, including predispositions to violence. Although initially a small minority in medieval England, such individuals grew in number and their descendants gradually replaced the lower class through downward mobility. By the nineteenth century, their lineages accounted for most of the English population [48,49].

This pacification of society did not occur uniformly throughout England. Endemic violence persisted until the 18th century in the northern border regions, where any encounter with non-kin, however innocent, could turn violent. "In a world of treachery and danger, blood relationships became highly important. Families grew into clans, and kinsmen placed fidelity to family above loyalty to the crown itself" [50, p. 628]. Disputes would grow into long-running feuds if not settled through payment of blood money [50].

What path will this research topic take in the future? Researchers will probably look for genetic markers that correlate with male predisposition to violence. In this, as in most mental and behavioral traits, there has probably been an accumulation of small heritable effects at many gene loci, since genes may intervene at many stages between ideation and actual behavior. To go from one stage to the next, for instance, a person must cross a threshold whose properties are genetically influenced. The threshold may float up or down in response to various cues in the environment, but these cues will feed into mental algorithms that are genetically encoded in whole or in part.

Since these diverse loci largely undergo similar selection pressures, we need data from only a few to chart the 'genetic pacification' of various human populations. Different loci may nonetheless display somewhat different patterns of geographic variation, since triggers for violent behavior are often culture-bound. In patriarchal clan-based societies, the trigger may be loss of face or a crime against the honor of female kin. In bands of hunter-gatherers, it may be rivalry for key resources. Generally speaking, genetic pacification should be most advanced where the State has monopolized the use of violence and where a breach of this monopoly will harm one's reproductive success through ostracism, imprisonment and/or execution, poor mating prospects, and fewer chances for wealth accumulation. Conversely, it should be least advanced where the State is weak, where men must defend themselves as individuals, and where prestige goes to the 'big man' who dominates social relations through charisma and intimidation.

2.4. Personality traits

Genes moderately influence variation in most personality traits. This has been the finding of studies on twins [51–54], separated twins [55–57], and adoptive families [58,59]. A study of 65 monozygotic twins reared apart and 54 dizygotic twins reared apart estimated heritabilities ranging from 26 to 62%. Heritability is 50% or more for schizophrenia, hypomania, religious fundamentalism, psychoticism, family problems, and phobias [57]. Risk taking is 20% heritable for experimental lottery choices and 35–54% for symptoms of pathological gambling, this trait being associated with alleles at two genes for dopamine and serotonin neurotransmission [60,61]. Deceitful behavior is 13% heritable when the twins have different evaluators and 65% when they have the same evaluator [45]. These substantial heritabilities do not necessarily mean that natural selection has been too weak to push one allele to fixation over all others. In fact, the likeliest model seems to be one of balancing selection due to environmental heterogeneity [62,63].

Do these traits vary among human populations? Some anthropologists, notably Ruth Benedict, used to argue that ‘modal personality’ differs from one population to another for such traits as shame, grief, jealousy, and self-restraint [64]. This paradigm, called ‘culture and personality,’ was influential until the 1960s but has since fallen into disfavor. Today, there is a subfield called psychological anthropology that focuses more on mental health than on broader personality issues. Many mental illnesses are in fact population-specific, often exclusively so [65,66].

Evolutionary psychology could revive the ‘culture and personality’ paradigm by reinterpreting it in terms of Baldwinian selection: 1) a culture requires adherence to a task, norm, or behavior and individuals comply by pushing their envelope of mental plasticity, i.e., they consciously mold themselves into the desired phenotype within the limits of their capacities; 2) this new cultural environment selects for genotypes that more easily produce the desired phenotype, with the result that a heritable basis will increasingly support what first came about through conscious effort; and 3) there is thus a shift in the mean genotype and its envelope of mental plasticity.

Such selection may explain differences in modal personality among human populations. Because these populations have different cultural environments, which require different approaches to social relations (high-trust vs. low trust, assertive vs. timid, etc.), each of them should develop its own mix of personality types.

3. Conclusion

This is a sample of research topics that will further develop as evolutionary psychologists move beyond the paradigm of one EEA in the Pleistocene. There will undoubtedly be other topics. All of them, however, flow from two classes of evidence that cannot easily fit into the existing paradigm.

First, humans vary in genetic predispositions for a wide range of mental and behavioral traits. Second, some of these predispositions vary not only from one individual to another but also statistically from one population to another. Any trait that shows some heritability may, in fact, differ statistically among human populations. To be sure, one might alternatively argue that all humans share a common mental and behavioral genotype that adaptively responds in different ways to different post-Pleistocene environments. Whatever the merits of such an idea, it would still depart from the single EEA of Tooby and Cosmides, which implies that few mental or behavioral adaptations of any sort have emerged since the Pleistocene.

Despite the evidence in its favor, the multi-EEA model is still widely dismissed as implausible. The situation resembles that of the late 19th century when geologists like Lord Kelvin believed the earth to be less than a hundred million years old—an estimate that left far too little time for the evolution of current life forms and that troubled even the most convinced evolutionists, including Darwin. Today, we face a belief that new mental or behavioral traits could not have arisen during the time of modern humans. On the one hand, such traits are believed to evolve slowly over long spans of time. On the other, our presumed panmixia would have largely prevented adaptations to the many ecological, social, and cultural environments that humans have entered over the last fifty thousand years.

Neither argument is true, no more than the alleged young age of the earth. Sooner or later, the single-EEA model will be abandoned, perhaps after an interim period of *de facto* abandonment. When and how remain a matter of conjecture. In all likelihood, we are looking at the medium to long term. The psychologist Jonathan Haidt predicts that this shift, which he calls a “game changing” scientific event, will begin between 2012 and 2017 [67]. Much will likely depend on the eloquence, positioning, and influence of the individuals who argue the pros and cons of this debate, particularly John Tooby and Leda Cosmides. As indicated by their latest writings, they themselves may play a role in broadening the EEA concept [2].

This debate will play out within a larger one of environmental determinists versus biological determinists, i.e., nurture versus nature. At present, the latter support the single-EEA model and its claim that all human populations share the same genetic constraints on behavior. With the entry of multi-EEA proponents into the debate, the two-way contest will become a three-way one that may make single-EEA proponents more acceptable to foes of biological determinism, perhaps to the point of fostering an informal alliance between the two camps.

A further complication is that all three camps will have different positions of strength in different forums of debate. Peer-led discourse may help the established camps hold their own in conventional forums (conferences, journal articles) while multi-EEA proponents gain ground elsewhere (Internet discussion groups, blogosphere). These divergent outcomes might settle into a long and even stable equilibrium of theoretical dissonance, i.e., researchers will support the multi-EEA model while construing their findings as support for ‘fine-tuning’ of a common human nature. The equilibrium will be all the more stable and long-lasting if environmental determinists and single-EEA proponents come to an accommodation and present a united front.

Such a united front could win over public and elite opinion by portraying its opponents as pessimists. If many deviant behaviors, like male violence, result from innate predispositions and are ‘deviant’ only in relation to the current host society, the range of possible societal responses will be narrower and the public mood less hopeful. This is so even if the pessimism is warranted. For instance, when people believe that illness is caused by spirits and can be cured by magic, they are more optimistic about the prospects for treatment than when they believe the cause to be physiological. All too often, modern medicine can offer only palliative care [68].

Will this debate be influenced by technological developments? Haidt points to the Human Genome Project and the growing volumes of data on the way genes vary among human populations [67]. Probably just as critical, though less commented on, is the increasing use of MRIs to image how the brain responds to different stimuli, such as alphabetical

characters versus Chinese ideographs [33]. There is also the democratization of research via online databases, which is broadening a research community that hitherto encompassed only salaried professionals and graduate students with special-purpose funding. Finally, the Internet is altering the information life-cycle (idea creation → elite awareness → popular awareness) by speeding up the flow of ideas and creating alternate paths for publication and peer review [69,70].

Nonetheless, if we look at the current evidence for multiple EEAs, it has largely been produced through low-tech methods that go back half a century if not further, e.g., twin studies, adoption studies, longitudinal research, cross-cultural analysis, polymorphism genotyping, charting of allele frequencies. The limitations seem to be more conceptual than technological. People will seek evidence for the multi-EEA model once they begin to question “the belief that genetic change happens at such a glacial pace that there simply was not time, in the 50,000 years since humans spread out from Africa, for selection pressures to have altered the genome in anything but the most trivial way” [67].

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