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DRAFT

Historical Evidence and Human Adaptations

Abstract:

Phylogenetic information is often necessary to distinguish between competing evolutionary scenarios. Recently, evolutionary psychology has acknowledged this, and has claimed that such evidence can be and has been brought to bear on adaptive hypotheses involving complex human traits. If this were possible, it would be a valuable source of possible tests for hypothesized adaptive traits in humans, especially given that such hypotheses generally cannot be tested by the sorts of phenotypic manipulations used to test adaptive hypotheses in other species. However, I argue that in practice evolutionary psychology has failed to use phylogenetic information in a meaningful way, and further the structure of the Hominidae family makes such a research program nearly impossible. While phylogenetic information can be useful for testing adaptive hypotheses in humans, these generally involve traits that are (a) not widely shared in the species and (b) of a much lower order of complexity than the sorts of traits evolutionary psychology has been interested in. With respect to complex psychological traits, the human species lacks sufficient genetic variation, and the closest extant relatives to the human species are too phenotypically different from humans, for such methods to provide meaningful tests.

Historical Evidence and Human Adaptations¹

I. Introduction: Adaptations, Adaptationism, Evidence, and Humans

The primary focus of the current debates surrounding ‘adaptationism’ seems to be resolving into questions about the sorts of evidence necessary for an adaptive (or a non-adaptive, for that matter) hypothesis to be considered well-supported (see e.g. Griffiths 1996, Brandon and Rauser 1996, Rose and Lauder 1996 and cites there, Pigliucci and Kaplan 2000 and cites therein); what requirements are accepted as legitimate will obviously have serious implications for the success of evolutionary

psychology meeting its stated goal of providing strong empirical support for the adaptive hypotheses put forward² (see Lloyd 1999, Lewontin 1998). There are a number of different kinds of evidence that can be brought to bare on hypotheses surrounding how and why a particular phenotypic trait (or traits) arose, spread within a population, and has been maintained within that population. These include laboratory and ecological field studies of the consequences of phenotypic manipulations of that trait (or those traits), laboratory evolution experiments designed to test adaptive and non-adaptive hypotheses (such as those involving constraints, or the robustness of proposed pathways), optimization analyses of various sorts, and analyses of the phylogeny of the populations involved; in general, more than one type of evidence will need to be gathered for an adaptive (or again, a non-adaptive) hypotheses to have been successfully tested against reasonable competitors (see Pigliucci and Kaplan 2000)³.

In this paper, I want to explore the extent to which the various techniques used to test adaptive hypotheses in non-human populations can and have been successfully applied to testing adaptive hypotheses in the context of human populations. Currently, evolutionary psychology has been putting forward the boldest such hypotheses; however, other disciplines, such as medical genetics, have also put forward hypotheses involving adaptive scenarios in the human case. Comparisons between these domains will reveal that evolutionary psychology's focus on complex psychological traits as well as on supposedly universal human adaptations is deeply problematic from the standpoint of putting forward testable hypotheses. Approaches less focused on these arenas however, might prove to be more promising.

II. Testing Adaptive and Non-Adaptive Hypotheses in Humans: The Impossible and the Difficult

Unfortunately, many of the tests common to testing adaptive hypotheses in other organisms are of little use in testing purported human adaptations. Obviously, experimental manipulations designed to reveal fitness consequences of modifications of putatively adaptive traits, both in laboratory and in natural populations (see Sinervo and Basolo 1996 and cites therein) fall into this category, as except in the most limited of ways, using these techniques on humans would be ethically impossible. Partial exceptions are provided by 'natural' experiments, in which phenotypic traits are 'manipulated' accidentally (either because of e.g. traumatic injury or genetic mutations); although in these cases it is very hard to control for variables. In the case of hypothesized human *psychological* adaptations, the manipulations necessary to properly test adaptive hypotheses by phenotypic manipulation might well be beyond the current state-of-the-art in e.g. neuropsychology, even if they weren't ethically impossible. Similarly, laboratory evolution experiments designed to for example falsify alternate hypotheses (such as those involving constraints and allometry) (see Schlichting and Pigliucci 1998) or to test the repeatability of adaptive pathways (see Rose, Nusbaum, and Chippindale 1996, and cites therein) can be rejected as all-but impossible in the human case.

'Transplant' experiments, a variation of experimental manipulations wherein supposed adaptations to local conditions (e.g. hypotheses about the significance of particular ecotypes) are tested by physically *moving* the organisms in question to other locals and observing the fitness consequences (see Reznick and Travis 1996 and cites therein; see Wilson 1994 and cites therein), can only be 'done' with humans in a very limited way (e.g. following people or peoples that move), and generally with a self-selecting and likely atypical sample. Again, properly controlling for the effects of e.g. being moved

is obviously difficult to do in the human case. While in principle possible, little research on human adaptations has been done using these techniques (more on this topic below).

Optimization arguments that take historical contingencies into account (see for example Sober 1996, Orzack and Sober 1996, Lauder 1996, Seger and Stubblefield 1996, Dennett 1995 for discussion) seem a more promising avenue for research into human adaptations, as they primarily involve *observation* rather than manipulation. Similarly, techniques making use of broadly historical evidence about the genesis and spread of the trait in question should be one of the more plausible approaches to testing adaptive hypotheses in human. Indeed, this latter kind of evidence, has been explicitly mentioned as relevant by prominent evolutionary psychologists; Miller, for example, has noted that “examining the distribution of traits across related species with known phylogenies” can be used to “discern when and where evolutionary innovations occurred” (1998 117) (see also Buss 1999 54-64).

While these techniques are not obviously impossible in the way that most phenotypic manipulations of humans would be, there are still important questions about how successful one should expect such techniques to be at developing evidence that can be used to support or dis-support contemporary adaptive hypotheses in humans. In the case of some possible human adaptations, especially those that resemble the sorts of adaptations common to ecotypes, these techniques have been applied with at least moderate success, and adaptive hypotheses have been well-supported. However, despite the claims of some of evolutionary psychology’s proponents, I argue here that such evidence is rarely available in the case of purported human psychological adaptations. The very limited information available on the environments in which key aspects of human evolution took place make optimization techniques difficult to apply to the human case in practice; further, while in some cases phylogenetic information about Hominidae may provide evidence relevant to adaptive hypotheses in humans, nature and history have ‘conspired’ to make the task much more difficult with humans than it is in many other species (see also Sterenly and Griffiths 1999). These difficulties, combined with the well-understood difficulties involved in testing adaptive hypotheses in humans (difficulties that stem from the ethical limitations on human experimentation), continue to doom many of the hypothesis of interest to evolutionary psychology to having to make do with inadequate evidence. Oddly, perhaps, other sorts of hypotheses involving psychological traits that might be more amenable to the sorts of tests actually available in the human case seem not to have attracted attention of the evolutionary psychology community.

III. Human Adaptations: Discovery and Evidence

In the case of *physical* adaptations in humans, there is reasonable agreement regarding, at least, what needs to be explained, and how well supported current explanations are. For example, while human bipedalism seems by most people to be a fact in need of explanation, there seems general agreement that no current hypothesis (adaptive or otherwise) is so well supported by the evidence as to exclude all reasonable competitors⁴. The massive increase in brain size during human evolution is in a similar position – there is agreement that it stands in need of explanation, but no agreement on what that explanation is. While many possible explanations have been proposed, none are generally considered well supported, nor is there agreement on a plausible research program for solving this problem.

Famously, the high prevalence of alleles associated with sickle-cell anemia (the HbS alleles) in

certain populations is a fact in need of explanation; unlike the other cases, however, there is reasonable agreement on what the correct explanation is. The sickle-cell allele is an adaptation for partial malaria resistance; the alleles associated with sickle-cell anemia in homozygotes are associated with resistance to malaria in heterozygotes. Evidence for this adaptive hypothesis comes from many sources; the alleles associated with sickle-cell anemia are relatively prevalent only in populations that have lived in areas with serious malaria problems (mosquito infested areas); further, work done on the history of populations vis-a-vis migrations, prevalence of HbS allele, and the prevalence of malaria show a clear relationship; finally, reasonable estimates of the fitness of the HbS versus 'normal' alleles in various environments correlate fairly well with historical distributions (see Griffiths et al 1996, Das 1995 and cites therein). There are still any number of questions to be answered in the sickle cell case, and some fascinating work being done on the extreme heterogeneity of the clinical manifestation of sickle-cell disease⁵, but the basic adaptive claim seems reasonably well supported. While there has been some research suggesting that other diseases caused by single recessive genes (such as CF) may be the result of alleles with similar adaptive roles (in the case of CF, possibly resistance to typhoid; see Pier et al 1998), as yet none have gathered evidence strong enough to have been generally accepted. Indeed, a promising research avenue involves attempts to explain peculiar genetic variations through the testing of particular adaptive hypotheses more generally, such as the work being done attempting to link the prevalence of the apparently recent CCR5^{Δ32} 'HIV resistance allele' in populations of European descent to outbreaks of Black Death in Europe (see Stephens et al 1998).

Other phenotypic traits in humans for which adaptive explanations have been offered and seem reasonably well-supported include skin color (fair skin for diets poor in vitamin D in locals with low levels of sun-light), and some variations in the size and shape between populations in radically different climates (see for example Cavalli-Sforza and Cavalli-Sforza 1995, Lewontin 1995). There are undoubtedly others. What is startling, though, is that even basic phenotypic features (such as our large brains, bipedalism, opposable thumbs, relative-hairlessness, etc.) that are generally agreed to have arisen after the lineage that gave rise to humans diverged from those lineages that gave rise to the great apes have in general *not* been explained by hypotheses (adaptive or otherwise) that have gained wide acceptance (see Tattersall 1998 for a review). This is certainly *not* a feature of adaptive explanations shared with out species; for many species with species-typical phenotypic traits there are adequate adaptive explanations for these traits. Why then have acceptable explanations even for such banal human traits as our bipedal stance, been so hard to come by in the human case?

IV. Phylogenetic Histories and Bad Luck

The basic difficulty with testing adaptive hypotheses for widely-shared human traits stems from an unfortunate feature of our phylogenetic history. Our closest living relatives are the great apes; however, as these things go, they aren't really all that close. It is widely agreed that the most recent common ancestor we share with the great apes was at the very least 6 m.y.a., and possibly rather longer (see Tattersall 1995 218, Goodman et al 1998). Nor do those apes themselves represent a particularly diverse range of species; only the two extant species of chimpanzee share a relatively recent common ancestors with another extant species (Tattersall 1995 218, Goodman et al 1998); although some have suggested that DNA evidence suggests that orangutans may be best thought of as

representing two distinct species, having diverged perhaps 1.7 m.y.a. (see Gragneux et al 1999). There are, then, perhaps 7 or so species in the 'Hominini' tribe, which (perhaps) shares a common ancestor on order of 10-14 m.y.a. or so, and perhaps another few species of 'lesser apes' in the Hominidae family, sharing a common ancestor with the great apes on the order of 18 m.y.a. or so (see Goodman et al 1998, Gragneux et al 1999).

The problem with such a sparsely populated clade is that the basic techniques that permit one to figure out how putative adaptations relate to selective regimes are difficult if not impossible to assess without reasonably close relatives. To get good evidence that a trait was subject to strong selective forces, and figure out what those forces were (and hence what the trait might be an adaptation for), requires comparing phylogenetic histories of the populations and traits in question to ecological field studies⁶ of the fitness consequences of the traits in questions (see for example Griffiths 1996, Larson and Losos 1996, Sinervo and Basolo 1996, Leroi et al 1994), as well as doing for example Lande-Arnold regression analyses done on the phylogenetic changes (see Lande and Arnold 1983; see Schlichting and Pigliucci 1998 11-12, 166ff and cites therein for recent advances in these techniques). These techniques are only plausible if there are available variations on the traits to test against the known histories of the traits.

This evidence is not at all impossible to come by in other species, even for 'species typical' adaptive traits. For example, Sinervo and Basolo (1996) describe the research that was done to test the hypothesis that the 'sword' of the sword-tailed fish (*Xiphophorus*) is an adaptation to female preference for male fish with swords. Phenotypic manipulations, mainly the addition or subtraction of swords, were used both to measure the strength of the female preference for swords (and hence the fitness consequences of being 'sword-ed') in current populations, and, in combination with phylogenetic information about *Xiphophorus* and related genera (such as the closely related *Pirapella*), to try to unravel the history of the trait and preference. The studies suggest that the female preference for swords arose prior to the swords, that is, that the primitive trait is a female preference for swords but sword-less males. This, they suggest, supports the hypothesis that the swords are an adaptation to female preference (Sinervo and Basolo 1996 173-175). These tests were possible *because* a reasonable phylogeny exists for fish of these types; the genus *Xiphophorus* itself comprises several dozen species, the males of some of which have sword-tails (of varying lengths), and some don't, but where the females all have a preference for swords. The females of some closely related genera (such as *Pirapella*) share the preference for swords, even where none of the males of species of those genera share the trait; since evidence suggests that sword-less-ness is the primitive trait, the adaptive hypothesis is well-supported (see Sinervo and Basolo 1996 172)⁷. While Sinervo and Basolo point out that many questions still remain to be answered, this sort of research into putative adaptations addresses many of the problems with 'adaptationism' that Gould and Lewontin pointed towards (Gould and Lewontin 1979)⁸.

The problem is that this sort of evidence just isn't available in the case of supposedly universal human adaptive traits; we have no extant relatives which are suspected of sharing similar selective regimes and that can therefore be used to test the fitness consequences of the supposed adaptations. If all or most of the estimated dozen or so extinct hominid species (comprising, perhaps two or three genera) still existed, phylogenetic studies would certainly be easier, and might well be useful for distinguishing between competing hypotheses about the spread and maintenance of phenotypic traits of interest⁹. Unfortunately for testing adaptive hypotheses in humans, all the other hominids are extinct¹⁰,

and so comparisons between the groups, with special attention to the fitness consequences of differences in key traits, are impossible¹¹.

The best place to look for testable human adaptations, then, is either for traits widely shared in the hominoid (or larger) clade (hence, not uniquely human traits) *or* for traits that are of local adaptive significance (such as the HbS mutation or the CCR5delta32 mutation). However, there is very little systematic diversity within the genus *Homo*, either at the level of phenotype or at the genetic level. Indeed, current DNA evidence points humans having far less genetic diversity even than the other great apes, despite the relatively old age of our clade (Gagneux et al 1999 5081). This of course limits the extent to which adaptive hypotheses for uniquely human traits, even those involving possible local adaptations, can be adequately tested.

The limited genetic diversity of the *Homo* has another problematic implication for those attempting to explain uniquely human traits as adaptations. It has been suggested that the most likely explanation for the low level of genetic diversity in *Homo* is that at some point, the “lineage leading to humans must have experienced a lower genetic effective population size” though just how this played out remains unclear (Gagneux, et al 1999 5080). If this is so, the implications for non-adaptive explanations of widely shared human traits are intriguing: small effective population sizes could easily result in the fixing particular phenotypic traits through non-adaptive forces such as drift (see e.g. Futuyma 1998). Again, this recommends a cautious approach towards attempts to argue towards universal uniquely human adaptations.

V. Giving Up On Universal and Unique Features: What’s Left For Evolutionary Psychology?

Insofar as evolutionary psychology’s goal is to explain ‘human nature’ as a collection of adaptive traits unique to humans (see Cosmides, Tooby, and Barkow 1992 5), the unfortunate structure of our clade, combined with the ethical constraints on human research, seem very problematic. Given this, evolutionary psychology’s fascination with explaining supposedly species-typical traits¹² is especially problematic. However, evidence relating to other related goals is more plausibly obtainable.

Wilson notes that evolutionary psychology’s obsession with a universal human nature has blinded the research program to possible adaptive phenotypic variation between human populations; he argues that there are both theoretical and empirical reasons to be suspicious of evolutionary psychology’s claim that human psychological adaptations will be universal (Wilson 1994). More to the point, some of critical difficulties in gathering evidence relevant to claims regarding universal uniquely human adaptations don’t apply to gathering evidence relevant to more local, population-level adaptations. For example, when population-level psychological adaptations are suspected, adoption studies might provide ‘natural’ transplant experiments. While obviously not as good as adoption studies, studies of immigrants to new locals could also shed some light, at least of a negative sort, on hypothesized population-level adaptations. Of course, gathering such evidence would not necessarily be easy. Not only would it require cross-cultural field work, but it would also require careful work on adoption and immigration practices in order to attempt to deal with the large number of confounding (cultural) factors¹³.

Another approach is to deal not with hypothesized adaptive traits that are uniquely human, but rather with adaptive traits shared by at least some of our close relatives. Some promising work has

been done in this area; for example, as Sterelny and Griffiths point out, it seems like it should be possible to do good work on the origin of those emotions widely shared within the Hominidae lineage (1999 chapter 14). While little of that work has been done, hypotheses about the origins and adaptive significance of some aspects of our emotions might be tested by comparisons to related species which share problems (and solutions?) of varying similarity. Similarly, the work done regarding testing theories regarding sperm competition, while not yet wholly adequate to reveal anything of interest about the human case, show at least a good start (see Futuyma 1998 359, 588-589 for an introduction to some of this research)^{14, 15}.

Evolutionary psychology has not yet developed the tools necessary to uncover our “shared human nature” (if such there is – see Dupré 1998) any more than physical anthropology has been able to uncover the specifics even of such clear human adaptations as our bipedalism. It is obvious that our brains were subject to selective pressures during our evolutionary history; it is not at all obvious what those pressures were (see note 9). Rather than over-reaching by attempting to uncover the historical causes of psychological features shared by all and only humans, evolutionary psychology might be better off attempting to develop ways of adequately testing possible adaptive traits of a less ambitious nature, either because they are widely shared outside our species, or shared only by specific ‘eco-types’ within our species.

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NOTES:

1. I'd like to thank David Buller for his help in developing these ideas via a long email exchange; the section on the unhealthy conviction held by many prominent evolutionary psychologists that their work should be focused on finding universal human adaptations owes much to his advice and help with citations.
2. Kitcher put this point quite well back in 1985, with respect to the relationship between Gould and Lewontin's famous paper and sociobiology (see Kitcher 1985, Chapter 7). As is so often the case with research programs involved in human genetics and evolution, however, very few within the research programs took notice. Lewontin's 1974 paper regarding the profound uselessness of broad-sense heritability for understanding the relationship between genetic and phenotypic variations in humans is another example of this.
3. Sometimes, a single sort of evidence will be adequate to for example confirm that selection was involved, but rarely will any single form of evidence tell you *why* selection was involved. As evolutionary psychology is generally interested in *specific* adaptive hypotheses, rather than the hypothesis *that* a particular trait is an adaptation for something or other, in general the requirements will be of the stronger sort.
4. The 'primitive condition' in this case was clearly not bipedalism, so it is a change that stands in need of some explanation. Adaptive hypotheses include at least: freeing the hands, either for more sophisticated physical labor or for child-rearing; better visual range in tall grasses; thermo-regulation by reducing the radiant heating of the body and increasing cooling potential; fooling predators evolved to focus on horizontal silhouettes; and others. One must be careful to distinguish a range of questions and possible answers, including: When and under what conditions did bipedalism first arise and spread? What was its spread helped and hindered by? What factors *created* by bipedalism then made continued bipedalism attractive? and others. See Tattersall 1998 110ff for discussion.
5. For example, some preliminary work may point towards the existence of 'co-mutations' inherited with the HbS allele that provide partial protection against some of the medical problems associated with sickle-cell disease, even in the homozygotic case (see Guasch et al 1999 and cites therein). More work obviously needs to be done on this subject, including when these (putatively) protective mutations arose compared to the HbS mutations.
6. Fitness consequences can also, of course, be evaluated via experimental studies; however, as noted above, these are usually impossible with humans for both ethical and practical reasons.
7. As an aside, Miller for example states quite boldly that sexual selection will usually generate traits not shared among species of a single genus, let alone between related genera (2000, 228); as the above shows, preferences pre-existing speciation events coupled with similar developmental possibilities can cause roughly the same 'adaptations' to sexual-selection to independently evolve several times.

8. Some of these questions include: when, precisely, the trait arose, when the preference arose, the relative strength of the pre-existing preference compared to contemporary preferences, and why such a preference arose in the first place (see Sinervo and Basolo 1996 175).

9. Estimates for the number of extinct hominid species vary widely; the number of relevant genera is if anything more controversial. Here I follow Tattersall 1995 (see esp 229-230). Also, if we think of these extinct species as falling into 3 or more separate genera, as Tattersall suggests and I follow, how exactly to refer to this clade is an interesting question. However, the nomenclature as well as the exact number of species and genera one settles upon matters less for the point I wish to make here than the recognition that extant humans represent a far less diverse group, biologically speaking, than our extinct lineage represents. This, I take it, is a far less controversial claim.

10. *Why all the other hominids are extinct* is of course an interesting question; suggestions range from other hominids being out-competed by *H. sapiens* for resources to *H. sapiens* having systematically slaughtered all the other hominids (see Tattersall 1995, 2000); other possibilities of course cannot be ruled out. It should be noted that those evolutionary psychologists convinced that the key innovations in the line that led to *H. sapiens* were the result of runaway sexual selection (e.g. Miller) will be at something of a loss to explain why this happened in the line leading to *H. sapiens* and not other hominid lines, and why all the other hominid lines went extinct.

Clearly our large brains evolved through natural selection of one sort or another, but the key innovation (if there was such a thing) that set *H. sapiens* apart from the other hominids may or may not have been related to brain-size or processing power simply construed (see Tattersall 2000 for a particularly bold hypothesis). Big (and metabolically expensive) brains may have evolved in the hominid line several times, or only once (see Tattersall 1995, 2000), and the uses to which they were put in our evolutionary history may well represent 'exaptations' or 'exadaptations' of the selective pressures they evolved in the face of.

11. Another difficulty is that despite many evolutionary psychologists' claims to the contrary, little is known about the details of the environmental (including social) conditions under which human evolution took place; without such details, analyzing the fitness consequences of traits is, of course, difficult. This difficulty is less important if one is concerned to show merely that there was directional selection (say) driving a trait towards fixation (say), and more important if one is interested in knowing *why* the trait was so-selected (what it is an adaptation for) (see note 3, above).

12. Cosmides and Tooby have been most active in arguing this position; see Tooby and Cosmides 1989, 1990, 1992, 2000. Buss makes the same argument in his (1995), as does Pinker in his (1999) and Symons (1995). Wilson (1994) argues against it, and while Miller (2000) seems to accept the argument with respect to 'ordinary' adaptations, he rejects it for those traits involved in sexual selection, which are his primary concern (one might even say obsession).

13. Indeed, while it should be possible in principle to test population-level adaptive hypotheses in the human case (through, e.g. natural 'transplant' studies, regression analyses done on historical changes in the phenotypes and between related populations, etc.), it may in practice prove to be impossible, as there may often be not enough information available to adequately support either the acceptance or the

rejection of adaptive psychological hypotheses. However, insofar as this is a serious problem for population-level adaptations, it is even more of a problem for universal adaptations.

14. Miller, for example, claims that the size of testes in human males compared to other primates *shows* that human females were polygamous during key periods of our evolutionary history (indeed, he argues that the comparisons reveal that ancestral females had sex with “more than one male in a month fairly often”) (2000 231-232); this strikes me as rather too bold a conclusion to draw on the basis of the evidence reviewed – Miller for example cites no research as to the likely primitive condition re: testes size in the lineage that gave rise to the great apes (did the ancestral line start with relatively large testes by today’s standards and did the proportional size drop in only some lineages, or did they start small and go up in only some lineages? Or has the proportional size gone up and down within particular lineages several times?). But claims of this sort at least point towards a plausible research agenda, albeit not one that many evolutionary psychologists seem to be inclined to pursue.

15. Of course, in both the case of traits shared with other related species and those that vary between populations, care must be taken to distinguish homologous traits from those that are homoplasious. While both homologous and homoplasious traits can be used to test adaptive (and non-adaptive) hypotheses, they must be used in different ways to do so. More to the point, the specifics matter a great deal. For a trait widely shared in, say, the Hominini clade, it matters a great deal whether the trait arose once in the ancestral population and has been passed down unchanged or whether it arose separately several times, each time under similar pressures. Similarly, it matters a great deal to arguments regarding the status of the trait vis-a-vis it being a possible adaptation if those populations which do not share the trait *lost* that trait (their ancestors had it) or failed to gain that trait in the way in which related species did. So far, at least, evolutionary psychology has shown relatively little interest in these distinctions (see Dupré (forthcoming) for discussion).