

selves to the Pleistocene). There is the additional problem that environments and selection pressures that are no longer extant – “the Pleistocene world of nomadic foragers” (Symons 1989) evoked at several points by Andrews et al. – cannot be directly studied. As a result, arguments linking the demonstration of adaptation or exaptation to such contexts are inherently limited to plausibility arguments rather than direct empirical tests. Where is the empirical evidence that a “sweet tooth” was adaptive in the “calorically limited ancestral environment,” that ancient social contracts vulnerable to free riders were countered by evolved “cheater-detection modules,” or that men of the EEA gained fitness advantages in choosing mates with high waist-hip ratios? There is a large gulf between the rigorously collected and analyzed survey data or lab experiments of evolutionary psychology, and the just-so stories about the EEA that provide the “adaptive” component of “adaptive design” arguments.

Problems (2) and (3) are rather more complex (Smith 1998; Smith et al. 2001). The standard evolutionary psychology approach to revealing adaptive design is to construct an argument linking (a) ancestral selection pressures that pose (b) a specific adaptive problem with (c) a specialized cognitive module evolved for solving just that adaptive problem. The specialized module in turn implies: (d) a specific set of genes coding for its underlying neurological circuits. In principle, the argument is sound and provides a satisfying causal structure linking adaptations to selective pressures in a tight lock-and-key manner reminiscent of the releasing stimuli and fixed-action patterns of classical ethology. In practice, however, we can only guess at (a) and (b), and rarely have any evidence at all for (d). Cognitive psychologists themselves are bitterly divided about the degree to which (c) holds in higher-level forms of human cognition.

In addition, there is the problem of how to analyze an evolved mechanism (cognitive and otherwise) that generates facultative responses to a variety of “adaptive problems,” including ones that are relatively novel. Andrews et al. tackle this question by proposing that we distinguish cases of adaptation from exaptation. In essence, if we can build a case for the specificity of linked selective pressure, adaptive problem, and cognitive mechanism (elements [a]–[c] above), then we have a bona fide adaptation; but if the cognitive mechanism or adaptive problem is insufficiently specific, even if the effects of patterned behavior produced by this mechanism are adaptive, then we have an exaptation – in the case of learning, an exapted learning mechanism (ELM). But the distinction between these two options in any specific case is ambiguous at best, and a semantic issue at worst (Smith 1998). Andrews et al. discuss this issue, using the example of driving a car and optical flow, but ultimately waffle by concluding (see target article, Note 3) that it all depends on how the researcher “carves up” behavior. My point is that Andrews et al., and standard evolutionary psychology theory, are analytically hampered by their glorious vision of a set of tightly defined adaptive problems, cognitive modules, and one-mechanism-at-a-time adaptive responses. This may work fine for certain behavioral phenomena, but is inadequate for analyzing less tightly coupled systems.

One alternative to this “demonstrate special design, or else it’s just an ELM” approach is to analyze adaptive behavioral variation as forms of *phenotypic adaptation* that in any specific instance may draw on a variety of cognitive mechanisms and other adaptive processes (including cultural transmission and evolution). This is admittedly less precise but very often far more realistic. Abundant evidence suggests that humans are particularly well designed for engaging in processes of phenotypic adaptation, using what in behavioral biology are termed *conditional strategies*, or in psychological language might be labeled *broad-purpose learning mechanisms*. Humans also adapt to a considerable extent via processes of cumulative cultural evolution – a phenomenon on which Andrews et al. are conspicuously silent.

These various attributes of human behavioral adaptation presumably reflect a hominid evolutionary history characterized by severe environmental fluctuation (Potts 1998), and colonization of

an unprecedented variety of habitats (Foley 1996) via generation of novel technological and institutional responses. It is critical to remember that this behavioral diversity has been generated and maintained within a single evolving species, was essentially complete even before the origin and spread of agriculture (Kelly 1995), and was highly adaptive as measured by both environmental fit and reproductive outcomes. This suggests that human behavioral adaptive mechanisms are not limited to the highly specific and inflexible mechanisms discussed by Andrews et al. – the “stone-age minds” maladapted to post-Pleistocene conditions so often portrayed in the evolutionary psychology literature.

In sum, the research program advocated by Andrews et al. (and mainstream evolutionary psychology) looks far better in the abstract – as philosophy – than in practice – as a guide to either empirical research or theory building. By adopting a more realistic research strategy, evolutionary psychology could contribute a great deal to understanding behavioral adaptations and exaptations, but only in conjunction with other traditions such as behavioral ecology (Winterhalder & Smith 2000) and cultural evolution theory (Henrich & McElreath 2003).

Adaptationism and molecular biology: An example based on ADHD

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Abstract: Rather than starting with traits and speculating whether selective forces drove evolution in past environments, we propose starting with a candidate gene associated with a trait and testing first for patterns of selection at the DNA level. This can provide limitations on the number of traits to be evaluated subsequently by adaptationism as described by Andrews et al.

In a small example about Attention Deficit Hyperactivity Disorder (ADHD), Andrews et al. evaluated the hypothesis of Jensen et al. (1997) that “the presence of ADHD traits in *some* children” represents placement on the “response-ready” rather than the “problem-solving” end of a dimension of behavior that may have been beneficial in past environments but detrimental in current environments. Andrews et al. concluded from this: “Because ADHD appears to lack special design for response readiness, the evidence seems to be more consistent with the hypothesis that ADHD is a maladaptive spandrel that persists despite selection, not because of it” (sect. 5.2).

As specialists in the area of ADHD, we respectfully offer some technical objections, including (1) disagreement with the assertions that “symptoms or consequences of ADHD have been found to hold an advantage” (Goldstein & Barkley 1998) and that ADHD is a disorder characterized by “a group of individuals, all of whom share the same failure of adaptation” (i.e., a deficit in behavioral inhibition) (Barkley 2001) – which seems to suffer from the common error of inclusion (i.e., assuming that all individuals with a heterogeneous disorder exhibit a characteristic of the group); and (2) disagreement with the acceptance of the null hypothesis about adaptation, which may suffer from the use of multiple tests.

We have used a molecular biological approach (Swanson et al. 1998a; 1998b) that may (1) avoid the many (see Sergeant et al. 1999) and often petty (see Swanson et al. 1998c) disagreements about a complex clinical literature, and (2) limit the number of alternatives to be considered in a psychological approach. We are aware of views that complex behaviors may involve so many genes (Plomin et al. 1994) and require analyses of genotypes so expensive as to make our approach impractical. But these views are changing (see Pennisi 2002), and here we present a concrete example.

We started with a candidate gene, the dopamine receptor D4 (DRD4) gene (Swanson et al. 2000a). This gene is extremely polymorphic in the human population as a result of an “imperfect” 48 base-pair variable number of tandem repeat (VNTR) in exon 3 that codes for the third cytoplasmic loop in a DA receptor that has regional localization in brain regions (e.g., anterior cingulate gyrus) involved in control of attention and movement (see Posner & Raichle 1994). Two to eleven repeats (R) have been identified, but the most common alleles in the worldwide human population are the 4R (~68%) and 7R (~15%) variants. We found the 7R allele to be increased in ADHD samples (LaHoste et al. 1996; Swanson et al. 1998c), and many other investigators replicated this finding (see Collier et al. 2000 and Faraone et al. 2001). Next, we used genotype as an independent variable. In a study of ADHD children (Swanson et al. 2000b) we found that the 7R-present subgroup did not exhibit some characteristic signs of cognitive deficit (slow and variable responding) that were manifested by the 7R-absent subgroup, and in a reaction time study of normal adults, we found that the 7R allele may confer an advantage in conflict resolution (Fossella et al. 2002). Subsequently, we focused on variation in a nonclinical sample of 600 chromosomes by sequencing the exon 3 VNTR (Ding et al. 2002) and used established statistical methods from evolutionary biology to test the hypothesis of selection at the DNA level based on nucleotide motif, allele frequency, linkage disequilibrium, and patterns of variation predicted by the “Out of Africa” theory. Based on this, we proposed that the 4R allele was the ancestral DNA sequence and that the 7R allele originated about 50,000 years ago as a rare mutational event (requiring multiple recombinations, gene conversions, and single nucleotide mutations to be derived from the 4R sequence), yet rose to high levels attributable to positive Darwinian selection. With this in hand, we then sequenced DNA from a clinical sample to investigate why an allele with the “fingerprints” of selection is overrepresented in individuals with ADHD, which led to the suggestion that the 4R and 7R alleles probabilities are the result of “balanced selection” (Grady et al. 2002).

Given evidence of selection at the DNA (nucleotide) level, speculation about the traits (and the association with ADHD) can be limited and tested in the context of the type of program proposed by Andrews et al. One could speculate that individuals with ADHD and the 7R genotype had personality traits such as novelty seeking that drove migration out of Africa and enriched the 7R allele in groups that populated the Americas (Chen et al. 1999). Another possibility, based on Darwin’s (1874) hypothesis that selection will be the result of “any advantage which certain individuals have over others of the same sex and species solely in respect of reproduction,” is that individuals with ADHD and the 7R genotype may have manifested traits affecting sexual selection and mating behavior that resulted in increases in the 7R allele depending on the cultural milieu (Ding et al. 2002). Specific types of cultural selection in males favoring nurturing fathers (“dads”) or non-nurturing fathers (“cads”) could account for the higher prevalence of the 7R allele in some ethnic groups than in others (Harpending & Cochran 2002), and determining the allele frequencies of DRD4 7R in cultures known to exhibit variation in particular behaviors could be used to test this.

To follow this molecular biological approach and limit the number of traits to be evaluated in a psychological approach, we propose the following steps: (1) identify a candidate gene associated with a trait (or a condition such as ADHD); (2) evaluate the DNA nucleotide sequence of the gene to see if any allele has the “fingerprint” of selection; (3) identify the protein change that was produced by the sequence change from the ancestral coalescent DNA sequence; (4) determine the functional significance of the structural difference; and then (5) propose and test hypotheses about a restricted set of plausible traits that may be influenced by these differences in DNA variants and might have contributed to selection.

Adaptation for, exaptation as

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Abstract: The expression *exapted as* is offered as a substitute for the target article’s *exaptation for* and *exaptation to* on the grounds that *exapted as* is less likely to foster the pernicious intuition that natural selection designs for future consequences.

I am grateful for Andrews et al.’s clarifications concerning adaptationism, particularly for their point that the recognition of adaptations is secondary to and dependent on the recognition of exaptations. I also endorse their conclusion that Darwinian explanatory stories should more frequently contain fortuitous consequences as steps in the evolution of traits. Our own preliminary theory of the evolution of babies’ cries was a theory of that type, invoking the fortuitous consequences of the evolution of the speech apparatus in babies as a determinate of the form of babies’ cries and adults’ sensitivities to them (Dessureau et al. 1998; Thompson et al. 1996; 1998; see also, Falcon et al. 2002).

Because I think Andrews et al.’s article will have wide influence, I hope these authors will reconsider – perhaps even recant – their use of the expressions *exapted to* and *exapted for* in favor of the alternative *exapted as*. Different expressions afford different patterns of thinking. I fear that the expressions *exapted to* and *exapted for* will make much mischief in the years to come by fostering the idea that exapted traits are produced on account of their future utility.

Natural selection is a scientific metaphor or “model” (Thompson 2000; Thompson & Derr 1995; Williams 1966). It is used to explain how organisms, as they have descended through time, have come to more or less match to the demands of the circumstances in which they have lived. The model is akin to the process by which breeders eventually create organisms matched to their needs by breeding some members of a herd, flock, or stand of organisms instead of others in the same herd, flock, or stand. In the model, breeders choose organisms for their properties – high-butterfat cows over low, good-flying pigeons over bad, sweet-tasting corn over bland. These choices determine the breeding future of the individuals chosen or not chosen. In nature, the survival and breeding of organisms is taken to occur for the same reason: because something selected them on the basis of their properties.

Without both its parts, the formulation “Organism O has been selected for Property P” is dangerously incomplete. In the context of natural selection, it is meaningless to say what organisms we selected if we do not say what properties we selected them *for*. By itself, an individual-by-individual enumeration of the organisms that the breeder permitted to breed would tell us nothing much about how the selection regimen was going to change the flock. Furthermore, in an evolutionary context, to say that we selected organisms for P is useless if we fail to specify which sorts of organisms we obtained by that selection regimen. Selection claims display a sort of referential opacity. An organism that is selected for P may display a variety of other properties that the breeder (or nature) might not have selected, if he or she had the choice, because they had fortuitous consequences on the fitness of his/her stock (cf. Williams 1966). Understanding evolution as a historical process requires our knowing what sorts of other properties “came along” when we were engaged in our selection for P.

The philosopher of biology Elliot R. Sober (1984) has given much careful thought to this problem. He writes:

[T]he idea of selection for and against characteristic . . . [may be understood in terms of] a toy my niece once enjoyed playing with before it was confiscated to serve the higher purposes of philosophy. [The toy is a transparent plastic cylinder with three horizontal layers.] Each horizontal layer contains holes of the same size. The holes on each level are larger than those on the level below. The balls also vary in size. If the balls are at the top, shaking the toy distributes them to their various levels. This is a selection machine. Balls are selected for their smallness.