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What Genetics Can Tell Us about the Origins of the Modern Human Brain

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Today genetic methods are so powerful, and genetic databases so informative, that it is now possible to identify genes, and regions of the human genome, that have experienced positive Darwinian selection (i.e., adaptation) during the evolution of modern humans. What is more difficult than identifying the genes that experienced adaptation, is figuring out the basic functions of these genes. And more difficult still is figuring out just what the functional differences were between the ancestral form of a gene and the more adaptive form that replaced it. For adaptive traits that can be studied at the physiological or cellular level, it is sometimes possible to connect genetic changes to adaptive phenotypic changes. However for cognitive or behavioral adaptations, like many of these thought to be associated with the evolution of the modern human brain, it is acutely difficult to identify the genetic changes underlying the adaptations.

Because humans differ from other species in having high intelligence, language, and an array of behaviors that facilitate complex, culturally based societies, it seems reasonable to suppose that our species has experienced a lot of adaptive evolution that uniquely shaped our cognition, behavior, and sociality. If this is true then some of our genes are different from those of our distant ancestors in the way that they now encode such traits.

Evidence of a genetic basis underlying human cognitive and behavioral traits comes from several different sources. Certainly the observation that all humans (even infants) have capacities for reason and language that appear to differ sharply from those of other animals is strong prima facie

evidence of an evolved genetic basis for such abilities. A different kind of insight comes from cases when genes are disrupted. Many of our cognitive and social abilities can be strongly affected or eradicated by genetic mutations (McKusik 1998), suggesting (though not proving) that genes are responsible for some of our unique abilities. Another kind of evidence for a genetic basis of many behavioral and social traits comes from findings of behaviors that are not known in other animals but that seem to be common to all human societies, even those long separated from one another (Brown 1991).

Notwithstanding the uncertainty and complex debate that surrounds questions about the adaptive nature of particular cognitive traits (see, e.g., Hauser, Chomsky, and Fitch 2002, Fitch, Hauser, and Chomsky 2005, Jackendoff and Pinker 2005, Pinker and Jackendoff 2005), this review supposes that many of our unique abilities do have a basis in genetic adaptations. We can survey the issues that arise when trying to identify and study genes that carry adaptations for human cognitive and social traits, even if there is debate about the degree to which evolution by natural selection has shaped or caused these traits.

CONTRASTING ADAPTATION AT THE GENETIC LEVEL WITH ADAPTATION AT THE PHENOTYPIC LEVEL

At the DNA level a change in a species that is wrought by natural selection will be manifested as an alteration in one portion of the DNA sequence of the genome of each of the organisms of the species. At this level, adaptation begins with a mutation that alters the DNA sequence of a gene, proceeds through a process of natural selection, whereby the altered copy of the gene replaces other forms of the gene in the species; and is complete when all copies of that gene in the species bear the DNA sequence that resulted from the original mutation.

The contrast between this highly reduced view of adaptation and what is typically meant by "adaptation" as applied to phenotypic traits is considerable. A phenotypic adaptation is usually considered to be an inherited trait that is characteristic of a species and that came into existence through natural selection (Tooby and Cosmides 1992, Buss et al. 1998). The requirement of inheritance demands that a phenotypic adaptation has some basis in the DNA, but otherwise there is little need for any kind of one-to-one

correspondence between adaptations at the DNA level and at the phenotype level.

One phenotypic adaptation could be caused by one or more DNA adaptations. This will certainly be true of large phenotypic changes that are the result of many DNA changes over a period of time, but it can also happen through the simultaneous fixation of multiple DNA adaptations that contribute to the same phenotypic trait.

Similarly, one single DNA adaptation may affect a great many aspects of the phenotype (a process called *pleiotropy*). When a DNA mutation becomes fixed by natural selection it is because of the overall net effect of that change on the fitness of the organism throughout the life cycle, and not just because of its contribution to a salient trait that an investigator might identify. This last point concerning pleiotropy is especially relevant when considering traits of the mind or brain, for it is the brain tissue that expresses more genes than any other tissue. Adult human brain tissue expresses approximately 75% of all known genes at least to some degree (Franz et al. 2005). This means that for most genes a DNA adaptation is expected to lead to either an altered protein that is expressed in the brain (and typically other tissues) or an altered expression pattern in the brain (and typically other tissues). The point is somewhat rhetorical since many genes are expressed in contexts where they are not required, but it nevertheless serves to highlight the discontinuity between DNA adaptations and phenotypic adaptations. Those phenotypic adaptations for which a basis in DNA has been found are likely far more complex at the phenotypic level than may appear, simply because most genes are expressed in many tissues and many stages of the life cycle.

THE DIFFERENCE BETWEEN HUMAN TRAITS AND VARIATION FOR HUMAN TRAITS

When considering questions about genes for human traits it is important to highlight the distinction between traits for which humans differ from other great apes, and traits for which humans are themselves variable. To develop the point we can use language as an example and suppose that some fundamental aspect of language, which humans have and apes to do not, is an adaptation caused by natural selection since we last shared a common ancestor with chimpanzees (including both chimpanzee species, the common chimpanzee and the bonobo). But in addition to this human/ape difference,

we can also note that some humans are more versatile with language than others, and it is possible that some component of this variation has a genetic basis (i.e., it is possible that there is genetic variation that explains some of the phenotypic variation among people in language versatility). Then for the sake of the argument suppose for both of these cases (i.e., the human/ape difference and the differences among humans) that the differences in language ability are caused by genetic differences.

Both of these situations are interesting, and for various reasons we might want to find the genes underlying both kinds of genetic differences (i.e., the genes underlying the human/chimpanzee difference, and the genes that affect language and that vary in humans). However the contrast between these two situations is an important one for several reasons. First, the genes associated with the human/ape difference might rightly be said to be the site of adaptations that make us who we are—they set us aside from other animals. This is simply not the case for genes that are variable and that contribute to variation among people in some part of language, however interesting they may be. Second, there is no necessary association between that subset of our genes which experienced adaptations for language since our genetic separation from other apes and that hypothetical subset of genes which is variable for language among humans.

A third contrast that becomes apparent when considering these two categories of variation is a great disparity in our capacity to identify genes associated with a trait of interest. In the case of human/chimpanzee differences there are few tools available for discovering such genes. The geneticists' favorite method for identifying genes that affect a trait is genetic mapping. This method relies upon being able to compare large number of individuals with varying degrees of genetic similarity *and* varying amounts of sharing of the target phenotype. In the case of humans and chimpanzees, all humans differ from all chimpanzees to some degree at virtually all of our genes, and of course at all of the cognitive traits associated with humans' unique cognitive and behavioral traits. The massive correlation of all genes with all traits, when considering humans and chimpanzees, summarily halts any genetic mapping project that one might suppose, particularly given the ethical issues that immediately arise under some possible experimental designs. In contrast it is often possible, and increasingly less difficult, to map genes for traits that vary among humans, and today it is done routinely to identify genes that contribute to diseases.

Recent research on the microcephalin gene (MCPH1) highlights some of the issues that arise when considering human/chimpanzee differences and when considering variation among humans. Mutations at the MCPH1 gene are known to cause a harmful condition of reduced cranial capacity called microcephaly. The simple fact of such mutations suggests a role in brain development for the product of this gene, and it suggests the possibility that the gene might be among those at which humans have experienced adaptations affecting cognition. Evans et al. (2004a) and Wang and Su (2004) compared the DNA sequence of the human form of the gene with that of other primates, and showed that several branches of the gene tree had experienced accelerated rates of amino acid change—a clear sign of adaptation. However the branch of the tree that separates humans from chimpanzees did not have an unusually high rate. So on the basis of this evidence, this gene does not appear to be the site of a uniquely human brain even though it does appear to have undergone adaptation during primate evolution.

Another study of the MCPH1 gene looked within human populations, and in this case did find indirect evidence of different functional forms of the gene in human populations (Evans et al. 2004b). This finding was controversial, and it was argued by some to have been over-interpreted by the authors and possibly to be mistaken (Balter 2005). But suppose for the sake of the argument that it is true. Even if present-day people do vary for functional alleles at this gene, and even if there is evidence of cognitive differences associated with those alleles (although newer evidence seems to refute this, Mekel-Bobrov et al. 2007), the observation only concerns variation *within* our species. In other words, even if the finding were true it would not bear directly on the evolution of cognitive traits that separate us from apes.

THE AGE OF UNIQUELY HUMAN ADAPTATIONS

Based on fossil and genetic evidence the date of the last common ancestral species to humans and chimpanzees is in the range of 4 to 7 million years ago (Chen and Li 2001, Brunet et al. 2002, Hobolth et al. 2007). This age marks the lower temporal boundary of when adaptations could have occurred in the history of humans, and yet not be shared with related species. Of course, there were almost certainly a great many adaptations shaping cognition and behavior that happened in the ancestry of humans before the time of the last common ancestral species leading to humans and chimpanzees. But since

TESTING FOR GENETIC ADAPTATIONS

The test for adaptation that was used for the MCPH1 gene is based on the genetic code and the fact that in gene regions that code for proteins, some mutations will affect the protein by causing a change in the sequence of amino acids in the protein, and other changes will have no affect on the protein, because they are redundant within the genetic code. The former changes, called non-synonymous changes, are potentially more common (more random mutations will affect the amino acid sequence than will not), but because most non-synonymous changes are harmful (i.e., they cause reduced Darwinian fitness) these types of changes are relatively rare. In contrast the redundant changes, called synonymous changes, are likely to have little or no affect on gene function and to be selectively neutral. The synonymous changes are usually observed to be more common than non-synonymous changes when the DNA sequences of the same genes are compared in related species. For any pair of gene copies it is possible to calculate the ratio of the non-synonymous changes to synonymous changes, called the K_a/K_s or the D_N/D_S ratio. Both types of changes are calculated per position that could potentially have a change of that type. A ratio of 1 suggests that the sites that could have an amino acid change are evolving just as fast as the sites that can have synonymous changes and that are supposedly selectively neutral. It follows that a gene with a value of K_a/K_s that is statistically significantly greater than 1 can be inferred to have experienced a higher rate of amino acid sequence evolution than is expected based on sites at which mutations are neutral (Hughes and Nei 1988). In other words, a gene with a finding of $K_a/K_s > 1$ over some portion of its evolutionary history is considered to have experienced adaptation during that time. Genes with values of $K_a/K_s \leq 1$ may well have also experienced adaptive amino acid changes, but that cannot be detected on the basis of this simple kind of test.

such adaptations would have been present in the ancestors of humans and chimpanzees, they would also have been passed on to chimpanzees.

To assess the upper, recent time boundary of the interval in which uniquely human adaptations may have occurred we need to figure out when

was the last time it would have been possible for a beneficial mutation to arise and spread throughout the entire human population. This could possibly happen today because humans, in the past few hundred years, have been migrating at increasing rates. But before that it is likely that many human populations were isolated from one another for very long periods of time.

One way to assess the upper time boundary is to consider the age of fossils and artifacts associated with the earliest known modern humans. The oldest known skulls that look like those of present-day people, more than they resemble archaic hominins, have been found in Ethiopia with an estimated range of dates between 150,000 and 200,000 years (White et al. 2003, McDougall, Brown, and Fleagle 2005). Archaeological evidence from one of these sites reveals a Middle Stone Age culture and apparent modifications of some of the skulls point to mortuary rituals (Clark et al. 2003b). Given the skull morphology and the snippets of insight into the culture of these people there seems to be a fair chance that we would recognize in them many, if not all, of the mental traits we associate uniquely with humans.

From living humans we can also get a time point that brackets the age when all modern humans possibly were not isolated from one another (and thus could share in a common process of adaptation). It happens that Australia was populated by people perhaps fairly soon after some modern humans first migrated out of Africa, 40 to 50 kya (Bowler et al. 2003, Hudjashov et al. 2007). Genetic evidence from the mitochondrial genome and the Y chromosome suggests that Australians share alleles with New Guineans, and thus that genetically they are related to other Asians. This means that they are probably part of the same out-of-Africa migration event that lead to the peopling of Asia (Hudjashov et al. 2007). However, the genetic evidence also suggests a long-term isolation from other Asians. In other words, adaptations that are shared by all humans, including Aboriginal Australians, would have arisen not later than the time of the populating of Australia.

From these different reference points we can see that uniquely human adaptations would have occurred between about 6 million years ago and not later than the populating of Australia 40 to 50 kya. A somewhat different time range is arrived at if we wish to focus on adaptations that were also shared by the earliest known modern humans. In this case, uniquely human adaptations would not have arisen more recently than the age of the early modern skulls from Ethiopia, that is between about 150,000 and 200,000 years ago.

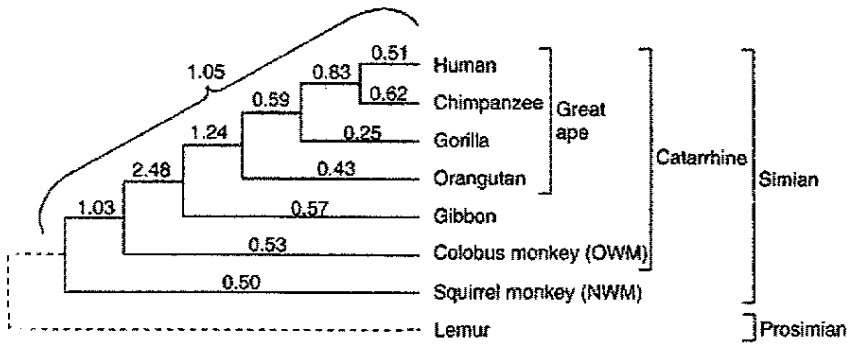
The draft sequence of the Neandertal genome suggests that we last ared a common ancestor with Neandertals between 270 and 440 kya, al- ough there is also a signal in the genomic data of limited gene flow from eandertals into modern humans (Green et al. 2010). These genomic data ovide an additional opportunity to estimate dates of genetic changes on e lineage to modern humans. However, our knowledge of Neandertal henotypes is limited to inferences from bones and archaeological evidence. hile these reveal Paleolithic cultures, many questions about Neandertal ulture and language remain open to speculation.

When the MCPH1 gene was examined in the Neandertal genome it as found to have the ancestral form of the gene (Green et al. 2010, Lari t al. 2010), which is not surprising given the recent estimated age of the ther form of the gene (Evans et al. 2005). However the finding is not con- istent with the possibility that had been suggested that the derived form of he gene that is found in many modern humans entered the population via ene exchange with Neandertals (Evans et al. 2006).

IT IS EASIER TO FIND ADAPTING GENES THAN IT IS TO KNOW WHAT TRAITS WERE UNDER SELECTION

Paradoxically, it is easier to identify genes that have undergone adaptation than it is to figure out what those adaptations are, or which phenotypic traits are involved. Consider again the MCPH1 gene and Figure 3.1 which indicates the branches on the primate evolutionary tree that show evidence of adaptation at this gene. This evidence comes from an analysis of the coding sequence of the gene and a finding of high ratios of changes that affected the MCPH1 protein, to DNA sequence changes that did not (see box). However, DNA sequences by themselves do not reveal the role that a gene plays in development. Even if a detailed role for a gene is known, DNA sequence evidence of adaptation is unlikely to reveal the nature of the trait differences between the old (before the adaptation) and the new (after the adaptive change) forms of the gene.

Now that both the human and the chimpanzee genomes have been sequenced it is relatively straightforward to align their genes and to assess the amounts of different types of changes at every gene. Roughly 10% of human protein coding genes are estimated to have undergone adaptive evolution at the amino-acid level since the time of common ancestry

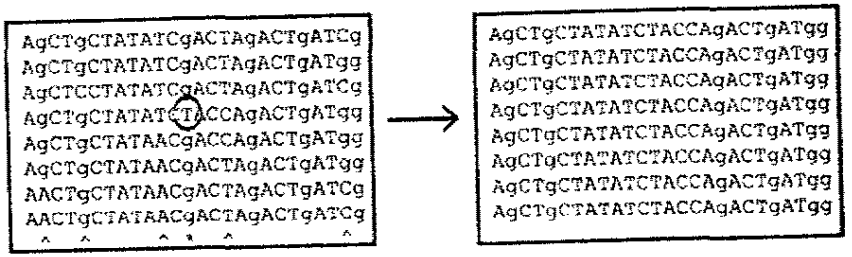


3.1. Evolution of microcephalin within primates. The Ka/Ks ratios of individual phylogenetic segments are indicated. The Ka/Ks of the entire lineage from simian progenitors to humans is also indicated. OWM: Old World monkey; NWM: New World monkey. (From P.D. Evans et al. *Human Molecular Genetics* 2004b 13:1139–145, fig. 1; used with permission.)

with chimpanzee (Clark et al. 2003a, Bustamante et al. 2005, Nielsen et al. 2005). Given that the human genome contains about 25,000 protein coding genes (International Human Genome Sequencing Consortium 2004), these findings implicate a couple of thousand genes as having been shaped by considerable adaptive evolution in the time since common ancestry with the chimpanzee. Also given that these studies report only those genes that show a clear statistical signal, it is likely that there are many thousands more of beneficial mutations that have occurred at individual genes but that went undetected given the statistical power of the tests being used.

But simply having found the genes that have experienced adaptations tells us nothing about the phenotypic traits that changed as those genes evolved. Humans can be expected to have experienced adaptations in the entire panoply of phenotypic traits, not just the minority of phenotypes associated with intelligence or language or behavior. The point is brought home particularly by the recent observation that chimpanzees have experienced more adaptation in protein coding genes than have humans, over precisely the same time period (i.e., since our common ancestor) (Shi, Bakewell, and Zhang 2006, Bakewell, Shi, and Zhang 2007).

Another approach to identifying genes that have experienced recent adaptation relies upon patterns of variation within a species to reveal the imprint of a mutation that has recently become fixed within a species. Figure



3.2. An illustration of selective sweep and genetic hitchhiking. On the left is a set of aligned gene copies. Base positions that are variable are indicated by a caret "^". At one base position, indicated by an asterisk, "*", one of the sequences has a base value that confers a selective advantage on individuals with that form of the gene. After the selected mutation becomes fixed in the population (right panel), all forms of the gene carry the selected form of the base and all of the sequence that was linked to that selected base. The effect is the removal of variation at other sites within the gene. In effect the sequences linked to the favored mutation have hitchhiked to fixation in the population by being physically linked to the selected base, and the gene has been swept clean of variation.

3.2 shows how a beneficial mutation (indicated on the left with a circle) can lead to a removal of variation in a population in the region of the chromosome that is near a gene. When the beneficial mutation replaces other forms of the gene in the population, all of the DNA sequence that was originally physically linked to that mutation also becomes fixed in the population. The next effect is a removal of variation that was present in the population before the selective event. This kind of removal of variation is called a "selective sweep" and the fixation of sequences linked to the beneficial mutation is called "genetic hitchhiking" (Maynard Smith and Haigh 1974).

The potential for selective fixations to remove variation from populations immediately reveals an approach to identify recent selective events by looking for regions of the genome that have unusually low levels of variation. To date, millions of variable positions in the human genome have been identified and this large database of single nucleotide polymorphisms (or SNPs) can be used to search for regions of unusually low variation (Williamson et al. 2007). This approach is particularly interesting because it is directly targeted at recent adaptations—regions of the genome that experienced adaptations and selective sweeps long ago are expected to have rebounded in the amount of diversity. In fact, it is possible to catch selective

sweeps "in the act" by identifying genomic regions that are significantly depauperate of variation in just one or some human populations. Recently, Williamson and colleagues (2007) used this approach and identified over 100 regions of the genome that appeared to have experienced a recent selective sweep. However the large majority of these affected only one or two of the three human populations considered. Only 21 genomic regions showed evidence of a sweep that affected all three populations (one each representing Europea, Asia, and Africa) in the study (Williamson et al. 2007).

A key limitation of using genetic polymorphisms to identify recent selective sweeps is that the analysis cannot offer information about the functional role of the adaptation that caused the sweep. Indeed, the situation is even worse when a gene is highlighted on the basis of high rates of amino acid polymorphism. In that case, at least the investigator knows what gene is involved, if not the functional differences that underlay the adaptation events. However, screens for apparent selective sweeps reveal only the general genomic regions that have low variation. If a region of low variation is long, as it may be if the selective sweep happened quickly, then there may be multiple genes contained within it, each of which could have been the one that experienced the beneficial mutation that caused the sweep.

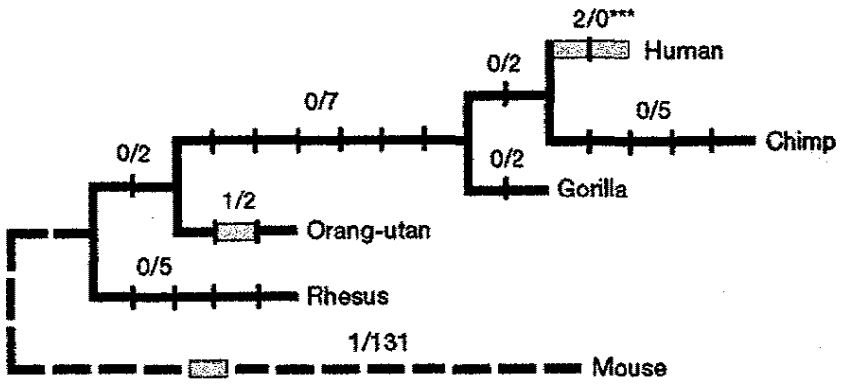
HOW CAN WE IDENTIFY THE PHENOTYPES ASSOCIATED WITH ADAPTATIONS IDENTIFIED IN THE GENES?

The classical way to do genetics is to start with a phenotype of interest and to try and find the gene(s) responsible. As described above, this requires some variation for the trait, in which case the situation lends itself to genetic mapping methods. However, it is not possible to map genetic differences between humans and chimpanzees for two reasons: (1) all humans differ from all chimpanzees at all genes *and* all traits that uniquely distinguish humans from animals, so that there is no scope for identifying particular associations between genes and traits; and (2) there are strong ethical prohibitions against developing methods that might overcome this.

The other modern (as opposed to classical) approach to connecting phenotypes to genotypes is to begin with the genes rather than the traits, and to try and figure out the function of genes and what phenotypes they affect. With the rise of molecular and cell biology, and with vast genetic and genomic resources for humans and many other organisms, it is today a simple

matter to begin an investigation of a particular phenotypic trait by starting with the genes that *might* be associated with that trait. The emphasis on "might" is used here to highlight the uncertainty of this approach. Investigators, knowledgeable of their phenotypes of interest, can come up with lists of genes that could possibly be involved with that trait. The sources of information used to make such connections are many and diverse, and ultimately dependent on the investigators' level of knowledge of their phenotype of interest.

A relatively straightforward example of this "candidate gene approach" is the FOXP2 gene, which encodes a transcription factor protein of 715 amino acids. Mutations in this gene have been shown to cause a speech and language disorder (Lai et al. 2001), suggesting that the gene might be a site of functional changes associated with the evolution of language in humans. Soon after the Lai et al. paper appeared, two groups of researchers compared the human form of this gene with those of other mammals and discovered that humans had two amino acid changes in this gene, whereas only one amino acid change had occurred in this gene since the common ancestry of apes and the mouse (see Fig. 3.3) (Enard et al. 2002, Zhang, Webb, and Podlaha 2002). In other words, the FOXP2 gene seems to evolve very slowly at the amino acid level and yet has had a relative burst of change on the branch leading to modern humans.



3.3. The gene tree for the FOXP2 gene is shown, with branch lengths among primates corresponding to the number of synonymous changes (dark bars) and non-synonymous changes (light bars). (Reprinted with permission from Macmillan Publishers Ltd: *Nature* 418 [2002], Enard et al., pp. 869–72, Fig. 2.)

From low levels of variation among humans at the FOXP2, it has been estimated that our current form of the gene became fixed in human populations within the past 200,000 years (Enard et al. 2002, Zhang, Webb, and Podlaha 2002). For insight into when the human form of the gene first arose, researchers turned to the Neandertal genome and found that both modern humans and Neandertals share the same form of the FOXP2 gene (Krause et al. 2007a).

The FOXP2 discoveries have triggered an explosion of research trying to discern just what it is that the gene does. Such studies include knocking out function in nonhuman model systems (French et al. 2007), more behavioral studies on individuals with mutations in FOXP2 (Liegeois et al. 2003, Hamdan et al. 2010), and studies of the location of FOXP2 proteins in cells in culture (Mizutani et al. 2007), among others. Recent studies of the affect of the human and the chimpanzee forms of FOXP2 on gene expression of neuronal cells in culture reveal that the human form of the gene alters the expression of over 100 other genes (Konopka et al. 2009).

CONNECTING MOLECULAR PHENOTYPES TO COGNITIVE AND BEHAVIORAL PHENOTYPES

Molecular biologists are rapidly discovering the genes that have experienced adaptation in the evolutionary history of modern humans. Yet they are limited in the tools they can use to identify what traits were affected by those adaptations. In the case of the FOXP2 gene we have a useful example of the panoply of methods that creative investigators are likely to employ. How long will it take to find the higher-level traits, above the level of gene expression, that are associated with the adaptations at these genes? What kind of traits will these be?

A mirror set of questions arise with regard to language, and other cognitive traits, for researchers who study cognition and language and other human behaviors. In areas of psychology, cognitive philosophy, and anthropology there are long-running debates about the nature of human cognition. For example, some scholars of language insist on a very limited role for Darwinian adaptation in the origins of human language (Hauser, Chomsky, and Fitch 2002, Fitch, Hauser, and Chomsky 2005), whereas others disagree strongly (Jackendoff and Pinker 2005, Pinker and Jackendoff 2005). Similarly, the focus in Evolutionary Psychology on identifying those com-

ponents of present-day human behavior that have evolved and that distinguish us from apes is controversial and generally lacking in consensus on just what components of human behavior can be singled out as indicative of adaptation (Buller 2005). A more hopeful approach links evolutionary changes in hominin cognition and behavior to the archaeological record and to such knowledge as is available about climate change, hunting patterns, social groups, and toolmaking; the chapters by Donald, Gärdenfors, Mithen, Nowell, and Richerson in this volume provide examples. Similarly, comparative studies of monkey, ape, and human behavior, as exemplified in the chapters by Chaminade, Seyfarth and Cheney, and Warneken in this volume, may help identify common adaptations shared by primate subgroups as well as species specific differences.

It is tempting to wonder, Who will get there first? Will molecular biologists, having identified the genetic targets of selection, find a way to get back to the phenotypic traits that were actually selected? Or will social scientists find a way to figure out what are the key components of cognition, language, and culture that are likely to have a basis in genetic adaptations?

4

The Primate Mind before Tools, Language, and Culture

ROBERT M. SEYFARTH AND DOROTHY L. CHENEY

Plato says in Phaedo that our "necessary ideas" arise from the preexistence of the soul, are not derivable from experience—read monkeys for preexistence.

We can thus trace causation of thought...it obeys the same laws as other parts of structure.

— Charles Darwin, 1838 [1987]: Notebook M

INTRODUCTION

Beginning with the arrival of the first stone tools, roughly 2.3 million years ago (Semaw 2000), the archaeological record provides a rich source of data from which to reconstruct the evolution of human mind and behavior. Supplementing these historical data, some living monkeys and apes, particularly chimpanzees, make tools (McGrew 1994, Matsuzawa 1994, Yamakoshi 2001) and exhibit a limited form of culture (Whiten et al. 1999), allowing these species to be used as points of comparison when developing theories about human cognitive evolution.

But what about the period before tools and culture appeared? Regardless of whether they were made by early hominids or modern chimpanzees, tools and culture did not emerge *de novo*—they were, instead, the product of minds that had been evolving for millions of years in response to a vari-