Probing Human Origins

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Please direct inquiries to:
American Academy of Arts and Sciences
136 Irving Street
Cambridge, MA 02138-1996
Telephone: (617) 576-5000
Fax: (617) 576-5050
E-mail: aaas@amacad.org
Visit our Website at www.amacad.org

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Introduction

As a social species, humans have unrivaled abilities to engage in symbolic thought and language and in moral, cooperative, and altruistic behaviors. Art, literature, music, mathematics, and science flourish only in human societies. Cultures and adaptive learned behaviors are socially rather than genetically transmitted from generation to generation and evolve in response to technological innovations. Understandably, humanists and social scientists are not captivated by the idea, increasingly prevalent in popular culture, that the DNA sequence of the human genome contains a book of instructions that defines being human. This DNA reductionist view of being human, with its corollary that genes are much more important than environments in dictating how different individuals act, is far too simplistic. A more defensible view is that during humankind's evolutionary history, genic changes occurred in ancestral genomes that were positively selected to help shape the distinctive human phenotype. This view complements rather than contradicts the view that our social environment guides how we act. Perhaps the major trend in humankind's evolutionary history has been the selection of genomes that gave their bearers the brain power to use learned, culturally transmitted behaviors to cope successfully with an increasing range of external challenges. Thus, paradoxically, because of humankind's genetic evolution, the future of the human species now heavily depends on its further cultural-social evolution rather than its further biological evolution.

The evolutionary origins of humans involved molecular-genetic, organismal-phenotypic, and cultural-social changes that increased adaptability to physical environmental changes. With the goal of exploring different facets of the complex holistic process of human origins, the American Academy of Arts and Sciences convened, at its House in Cambridge on July 6–8, 2001, a multidisciplinary study group of 35 senior and junior scholars. A National Science Foundation Biocomplexity Incubation Grant (entitled "Development of the Human Species and Its Adaptation to the Environment") provided the funding for this workshop-style conference. The group consisted of molecular biologists and geneticists, evolutionary developmental biologists, paleontologists, anthropologists, cognitive scientists, humanists, and computer scientists, all with the common interest of better understanding human origins. After the conference, members of the study group prepared papers based on their presentations and the subsequent discussions.

This volume contains five of these papers. Two of them—one by Gumucio et al. ("Primate Genomics: A Rich Resource for Functional Genomics") and another by Wildman et al. ("Functional DNA in Humans and Chimpanzees Shows They Are More Similar to Each Other Than Either Is to Other Apes")—illustrate how comparative primate genomic studies can help elucidate humankind's evolutionary history. In particular, such research can identify gene changes that were functionally significant and were favored by natural selection. The paper by Potts ("Complexity and Adaptability in Human Evolution") provides evidence that during the past 4.5 million years of humankind's history, increasingly variable conditions in physical environments selected for genomes that gave human ancestors adaptive versatility to endure increasing environmental instability and to invade new habitats. The paper by Richerson and Boyd ("Culture is Part of Human Biology") presents the thesis that the ancestors of modern humans had genomes that gave their bearers the capacity for socially transmitted, nongenetic, learned behaviors (i.e., for culture); however, culture in turn favored the selection of genomes that further increased the capacity of modern humans to engage in culture. The paper by Fouts and Jensvold ("Armchair Delusions Versus Empirical Realities: A Neurological Model for the Continuity of Ape and Human Languaging") calls into question the view that the capacity for language arose *de novo* in a relatively recent, common ancestor of all modern humans. In challenging the accepted view that there were no earlier evolutionary predecessors to human language, Fouts and Jensvold offer evidence from sign language studies of chimpanzees and from similarities between humans and chimpanzees in the neocortical structures concerned with language. They argue that the common ancestor of humans and chimpanzees may well have had the capacity for rudimentary human language.

Our goal in publishing these papers is to spur further innovative and, indeed, provocative discussions among natural scientists and humanists on the origin and evolution of the human species. There is a wealth of material to explore.

MORRIS GOODMAN and ANNE SIMON MOFFAT
January 2002

Acknowledgments

Many organizations and individuals contributed in many ways to the scholarship that produced this volume.

In 1998 Michael Teitelbaum of the Alfred P. Sloan Foundation spurred our investigation of humankind's origins with funding of a public conference on "Humankind's Evolutionary Roots: Our Place in Nature," held at the Field Museum in Chicago, Illinois, on October 9–11, 1998. The Foundation's suggestion that the conference open selected sessions to the public was visionary. About 400 junior and senior scholars attended the conference, including 80 from local high schools. We are grateful to school officials for providing transportation to the conference. Also, we are indebted to the Field Museum, and particularly to John Flynn of its Geology Department, for putting the museum's extensive facilities at our disposal.

We are grateful to the Presidents and Officers of the American Academy of Arts and Sciences, including Daniel C. Tosteson, James O. Freedman, and Patricia Meyer Spacks, and also to Leslie Berlowitz and Corinne Schelling, with whom we worked on this study.

Robert McCormick Adams and his colleagues on the Academy's Committee on Studies must be thanked for their ongoing support of the project as an official Academy endeavor, and especially for financial support for a planning meeting held in Cambridge on July 7–8, 1999.

That meeting led to a successful proposal to the National Science Foundation (NSF), which resulted in an award from the Biocomplexity Special Competition entitled "Biocomplexity Incubation Activity: Development of the Human Species and its Adaptation to the Environment" (award number BCS-0083721). The funds supported a multidisciplinary conference attended by 35 at the House of the Academy on July 6–8, 2001, and a subsequent planning meeting at the University of Michigan, Ann Arbor, on November 2–3, 2001. We are indebted to the NSF, to Academy staff, to the President's office at the University of Michigan, and to the invited speakers for their generous support.

We are also grateful for the sustained involvement of the Midwest Council of the American Academy of Arts and Sciences, which offered valuable guidance since the project's inception almost five years ago. Special thanks go to the three Midwest Council chairs, Robert Haselkorn (University of Chicago), Roger Myerson (University of Chicago), and Martin Dworkin (University of

Minnesota), who advised and encouraged us.

Dozens of scholars have contributed to these various conference and planning meetings, but the sustained support of a select few has been particularly valuable. These include Academy Fellows Jeanne Altmann (Princeton University), Francisco Ayala (University of California, Irvine), and Walter Fitch (University of California, Irvine), as well as John Flynn (Field Museum), Deborah Gumucio (University of Michigan), Edwin McConkey (University of Colorado), Richard Potts (Smithsonian Institution), Peter Richerson (University of California, Davis), and Carl Simon (University of Michigan).

This slim volume offers only a small sample of the diverse ideas on humankind's origins that have been generated during the course of this project. It includes a few of the papers presented and discussed at the July 2001 meeting. We hope, however, that this book will encourage ongoing discussions among natural scientists, social scientists, and humanists on just what makes us human.

CHAPTER 1

Functional DNA in Humans and Chimpanzees Shows They Are More Similar to Each Other Than Either is to Other Apes

DEREK E. WILDMAN, LAWRENCE I. GROSSMAN, AND MORRIS GOODMAN

Any discussion of the genomic origins of humankind must necessarily include a comparison of our closest living relatives, the chimpanzees. This paper discusses our current state of knowledge about the DNA gene sequences currently available for humans and chimpanzees. We have found that humans and chimpanzees share more than 99% of their genetic material. Despite their genetic similarity, however, there are obvious phenotypic differences between humans and chimpanzees. These phenotypic differences include the size of the brain's neocortex, the mode of locomotion, and the ability to produce complex vocalizations. Our long-term goal is to discover the genetic underpinnings of this phenotypic diversity.

King and Wilson (1975) suggested that most of the genetic causes of phenotypic differences between humans and the great apes are the regulatory sequences that control the timing and pattern of genic activity. However, differences may also exist in the structures of the proteins encoded by genes, which undoubtedly account for some of the observed differences in phenotypes. Structural differences cause proteins to function differently, especially in the ways that multiple proteins interact with each other. This paper examines a class of structural changes, called nonsynonymous substitutions at the DNA level, that are known to vary within the group of primates that

includes humans, the two species of chimpanzees, gorillas, and orangutans. We show that at this functional genetic level, humans and chimpanzees are more similar to each other than either is to any of the other apes. We also identify which genes studied to date are structurally different in humans and chimpanzees. These genes warrant further study because they point to possible pathways that are unique to humankind.

The obvious physical resemblance between humans and the great apes has been a common observation among Western scientists for centuries. In 1758 Linnaeus gave the name *Homo troglodytes* to a creature that may have been a chimpanzee. The common chimpanzee was not formally described until the early nineteenth century, when Oken (1816) described the genus *Pan*. We now know that there are two distinct chimpanzee species: the common chimpanzee, usually called *Pan troglodytes*, and the bonobo, or *Pan paniscus*. While most workers continue to recognize the chimpanzees to be a separate genus, a movement is emerging in the scientific community to recognize the close evolutionary relationship between humans and chimpanzees by placing them in the same genus, which by the rules of zoological nomenclature must be *Homo* (Diamond, 1992; Avise and Johns, 1999; Goodman, 1999; Castresana, 2001; Wildman et al., submitted). Chimpanzees and humans are believed to have separated evolutionarily between 5 and 7 million years ago. Our next-closest relative, the gorilla, branched off of our lineage about 8 million years ago, while the orangutan has been on a separate evolutionary trajectory for the past 12 to 16 million years.

There have been many studies of both human and chimpanzee infraspecific (i.e., within the species) variation. Notable among these is a study by Cann et al. (1987), which proposed that modern humans have a shallow genetic history going back only 200,000 years. That study, from which the name Mitochondrial Eve was taken, has since been backed up by numerous other studies, most recently by that of Kaessmann et al. (2001). Interestingly, chimpanzees show a severalfold increase (infraspecifically) in their genetic makeup when compared with humans (Kaessmann et al., 2001). Thus, chimpanzees are more genetically diverse than humans. It remains to be seen whether the human or chimpanzee pattern is the norm in other ape and primate species.

BIOINFORMATICS AND PHYLOGENETICS

Internet based, bioinformatic techniques were used to access the National Institutes of Health Genbank database and to compare those gene sequences available for humans and other apes, in order to determine what differences exist that cause proteins to change their shapes and/or their interactions with one another. The genetic

code works in a triplet manner, in which specific combinations of three DNA nucleotide bases code for each of the twenty or so amino acids, which are strung together to form the proteins that are the building blocks of all life. We examined in detail those observed nucleotide substitutions that cause amino acid replacements in the species under study.

Genbank searches were conducted for those genes that have been sequenced in humans and chimpanzees. When possible, the sequences from gorillas, orangutans, and Old World monkeys (the closest relatives to humans and the apes) were also analyzed. The other major group of apes, the gibbons and siamangs, were not looked at in our study because few data on their genes exist. We were able to compare the protein-coding sequences from 70 genes, totaling 73,104 nucleotide bases. Fewer nucleotides were compared in other taxon pairs (Table 1). Possible pseudogenes and paralogs were not included in the study. This study is the largest comparison of human and chimpanzee gene sequences to date, but it should be remembered that 70 genes is well less than 1% of the 40,000 or so genes that exist in the human genome. Whether our sample of genes is representative of the pattern of similarity and difference remains to be determined, and will remain unclear until much more of the entire chimpanzee genome is sequenced.

TABLE 1: CODING SEQUENCE DIFFERENCES IN CATARRHINES

TAXON PAIR	NO. OF BASE PAIRS	TOTAL DIFF.	KA	KS	KA/KS
HUMAN VS. CHIMPANZEE	71,307	0.0099	0.0070	0.0179	0.3921
HUMAN VS. GORILLA	42,849	0.0110	0.0090	0.0171	0.5240
HUMAN VS. ORANGUTAN	35,373	0.0198	0.0141	0.0343	0.4108
HUMAN VS. OLD WORLD MONKEY	18,507	0.0334	0.0232	0.0590	0.3930
CHIMPANZEE VS. GORILLA	42,849	0.0106	0.0075	0.0188	0.4003
CHIMPANZEE VS. ORANGUTAN	35,373	0.0194	0.0133	0.0342	0.3885
CHIMPANZEE VS. OLD WORLD MONKEY	18,507	0.0308	0.0232	0.0563	0.4117
GORILLA VS. ORANGUTAN	32,826	0.0178	0.0126	0.0304	0.4142
GORILLA VS. OLD WORLD MONKEY	13,248	0.0309	0.0227	0.0522	0.4352
ORANGUTAN VS. OLD WORLD MONKEY	14,718	0.0352	0.0269	0.0599	0.4497
AVERAGE	32,556	0.0219	0.0160	0.0380	0.4220

In order to analyze which amino acid replacements have occurred during the evolution of humans and apes, the evolutionary relation-

ships among the species being studied must be inferred. The evolutionary relationships, or phylogeny, of humans and apes is fairly well understood, and we have contributed to that understanding by analyzing the current data set in a phylogenetic context. This was accomplished by use of the phylogenetic methods of maximum parsimony, maximum likelihood, and neighbor joining available in the computer software program PAUP* (Swofford, 2001). Phylogenies were inferred by using all of the available data and by partitioning the data into first, second, and third codon positions. Support for the strength of the phylogenetic relationships among the organisms was measured by a nonparametric statistical method called bootstrap analysis. Bootstrap percentage values range from 0 to 100, and values greater than 70 are generally considered to indicate a phylogenetic relationship that is supported by the data being analyzed.

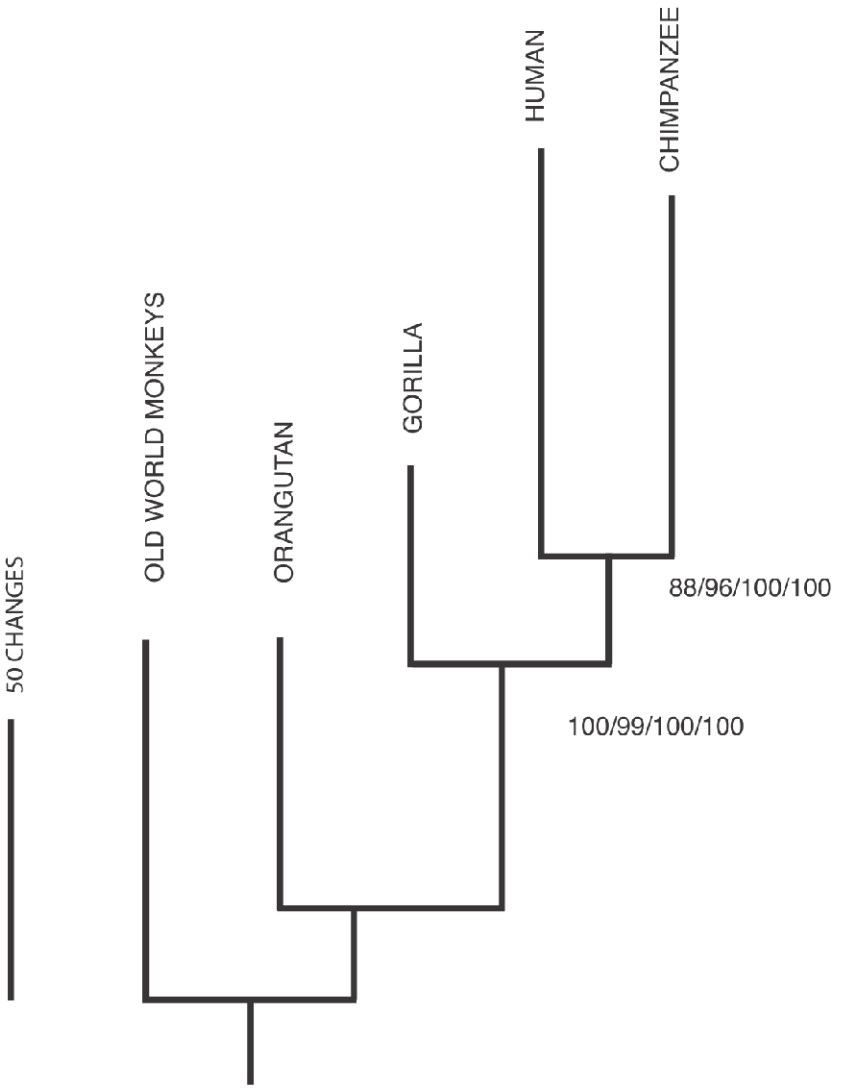
The ratio of nucleotide substitutions that change amino acids (nonsynonymous substitutions, or Ka) to those substitutions that do not change amino acids (synonymous substitutions, or Ks) was calculated. These different types of substitutions result from the redundancy inherent in the genetic code. This calculation is called the Ka/Ks ratio when calculated on a substitutions per site basis. To calculate Ka/Ks, we used the method of Li (1993), implemented in the computer program FENS (de Koning et al., 1998). Generally, a Ka/Ks ratio greater than 1 indicates that a gene is undergoing the effects of positive Darwinian selection (selection for a new gene sequence), whereas a ratio of less than 1 indicates that the gene is experiencing the effects of purifying selection (selection for the current gene sequence) among the organisms being analyzed. Given the short amount of evolutionary time involved among the species examined and the effects of selection, it is necessary to examine the data in a more statistically rigorous manner. Toward this end, standard deviations of the average Ka/Ks values for all of the genes were calculated, and those genes that are more than two standard deviations away from the mean value were considered most clearly to be undergoing elevated selective pressure.

HUMANS AND CHIMPANZEES ARE SISTER TAXA

Figure 1 (on page 5) shows the phylogenetic relationships inferred among the study taxa. The tree shown retains its topology (branching order) regardless of the phylogenetic method or data partition of nucleotide bases. Data were partitioned into first, second, and third codon positions, and all of the data were also combined into a single data set. The data clearly support the hypothesis that humans and chimpanzees are sister taxa, to the exclusion of gorillas and orangutans. Gorillas are the closest relatives of humans and chimpanzees, given the current data, which concur with fossil and other evidence suggesting that these three taxa evolved in Africa. Orangutans live in Asia and are the least closely related great apes.

The tree shown in Figure 1 was inferred from second codon positions only. These positions are exclusively nonsynonymous, causing changes in the proteins they encode, and it is interesting to note that these potentially structural changes also support the grouping of humans and chimpanzees (see below). The maximum parsimony bootstrap analysis of 1,000 branch and bound replicates supports the human-chimpanzee relationship with a value of 96, and the grouping

Figure 1. Hominid phylogeny based on coding DNA sequences. Phylogenetic tree inferred using parsimony. Bootstrap values are shown as follows: first codon position/second codon position/third codon position/all bases included. Branch lengths reflect an estimate of the absolute number of amino acid (second position changes) replacements on each lineage.



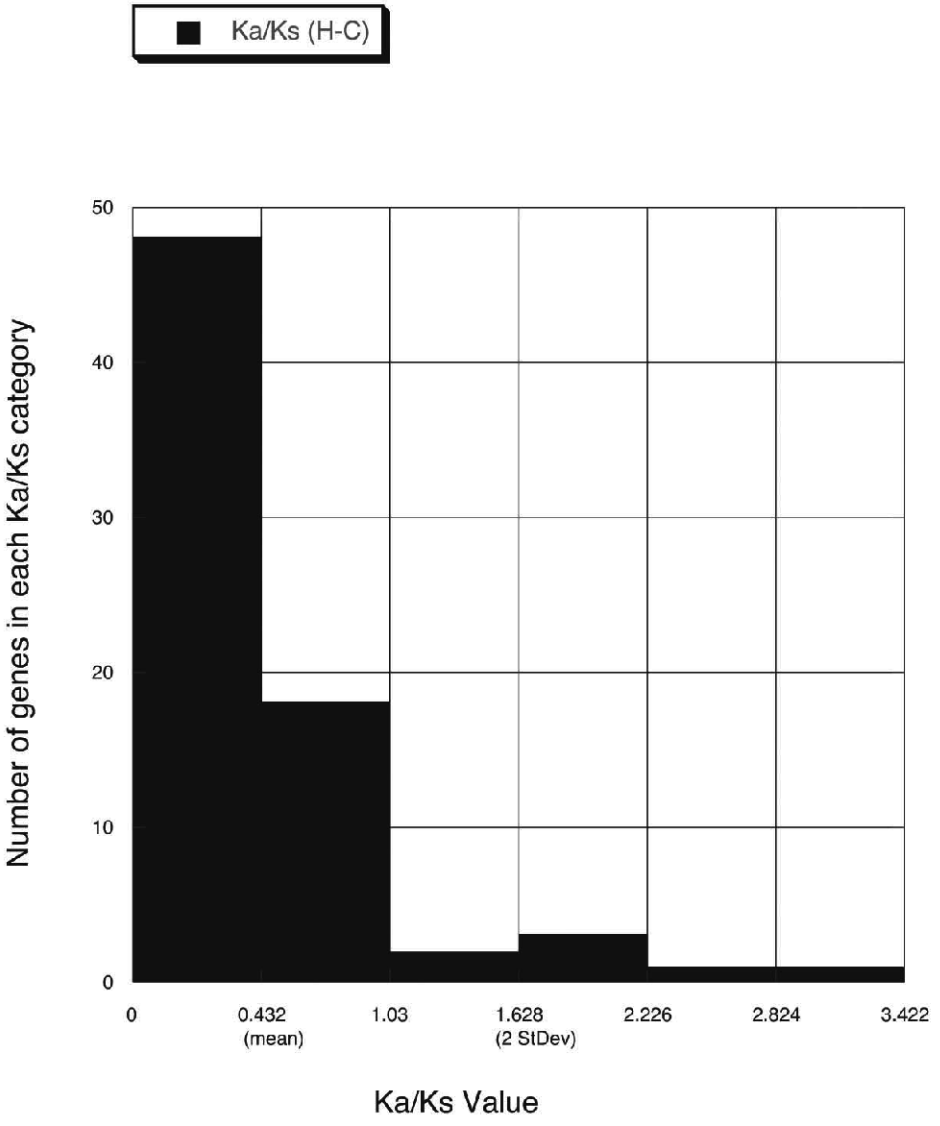
of humans, chimpanzees, and gorillas with a bootstrap value of 99. The maximum likelihood score for this tree is –ln L = 33971.58.

The summations of the Ka/Ks values are shown in Table 1. A complete list of the genes analyzed and their values are available from the authors on request. The average Ka/Ks value between humans and chimpanzees is 0.42, suggesting that most genes are experiencing purifying selection. This value is similar to those obtained in the comparisons between humans and gorillas (Ka/Ks = 0.52), humans and orangutans (Ka/Ks = 0.41), and humans and Old World monkeys (Ka/Ks = 0.39). The Ka/Ks ratio between chimpanzees and gorillas is 0.40. Thus, the notion that most genes are evolving neutrally is not supported by the current data.

With the average Ka/Ks values known, it is now possible to determine which genes have Ka/Ks values that deviate statistically from the mean values. Many genes are undergoing purifying selection, as indicated by a Ka value of 0. These genes include histamine H2 receptor, homeobox protein OTX1, homeobox protein OTX2, HTR1B gene for 5-hydroxytryptamine (serotonin) receptor 1B, and HTR1D gene for 5-hydroxytryptamine (serotonin) receptor 1D, among others. These genes that are undergoing purifying selection must have essential functions in humans and chimpanzees. The distributions of Ka/Ks values are shown in Figure 2 (on page 7).

The standard deviation for the human chimp values is 0.59; therefore, only those genes that have a Ka/Ks value of approximately 1.6 are considered to be unambiguously undergoing positive selection by our criterion. This is because these genes are two standard deviations away from the mean value. However, a value greater than one is generally taken to indicate positive selection, and therefore those genes are also discussed. The gene with the highest Ka/Ks value when humans and chimpanzees are compared is the sex-determining region Y (SRY) gene, which has a Ka/Ks value of 3. This gene has unambiguously undergone positive selection since humans and chimpanzees diverged from each other. Six other genes—BRCA1, Alpha2-HS glycoprotein, interleukin 8 (Il8RA), OR1G1 olfactory receptor gene, protamine p2, and Rh50 glycoprotein (RHAG)—have Ka/Ks values greater than one (Table 2 on page 8). Thus, of the 70 genes analyzed, five and possibly as many as seven are under the effects of positive selection. If these values are extrapolated to the 40,000 total genes in the genome, we can expect that at least 2,850 and possibly as many as 4,000 genes have undergone positive selection since the time of the most recent common ancestor.

Figure 2. Histogram of Ka/Ks values for individual genes between humans and chimpanzees. All 70 genes were analyzed. Incremental values represent standard deviations from the mean. The majority of genes have values <0.42, suggesting that most genes are under the effects of purifying selection. Seven genes have values >1, suggesting positive selection.



HUMANS AND CHIMPANZEES SHARE 99 PERCENT OF THEIR CODING DNA

The analysis presented in this study unambiguously shows that chimpanzees are our closest relatives, to the exclusion of other primates.

Furthermore, the functional genetic differences that are represented by nonsynonymous sites also show this relationship. The notion that the great apes form a functional and evolutionary grade is not supported by our analysis. Rather, humans and chimpanzees are a functional evolutionary clade. We anticipate that future analyses of the gene promoters and other regulatory regions will also show this relationship.

When humans and chimpanzees are compared, of the genes apparently undergoing positive selection, the sex-determining region of the Y chromosome (SRY) has the highest value (3.0). This has been noted in other studies (Pamilo and O'Neill, 1997; Patel et al., 2001), and it is interesting to contemplate because reproductive patterns, although similar between the two taxa, are different in that humans have longer gestations, interbirth intervals, and generation times. Another locus, BRCA 1, also shows an elevated Ka/Ks value, and is related to breast cancer etiology, specifically in terms of DNA repair mechanisms; differences between the two species may reflect variation between human and chimpanzee reproductive biology (Huttley et al., 2000). The other genes that show elevated ratios are listed in Table 2.

TABLE 2: GENES THAT SHOW KA/KS VALUES >1 BETWEEN HUMANS AND CHIMPANZEES

GENE NAME	NO. OF BASE PAIRS	TOTAL DIFF.	KA	KS	KA/KS
SEX-DETERMINING REGION Y (SRY)	699	0.0130	0.0150	0.0050	3
BRCA 1	5592	0.0070	0.0080	0.0035	2.29
I18RA	1050	0.0050	0.0060	0.0030	2
ALPHA2-HS GLYCOPROTEIN	1101	0.0080	0.0090	0.0050	1.8
RH50 GLYCOPROTEIN (RHAG)	1284	0.0080	0.0090	0.0050	1.8
PROTAMINE P2	309	0.0320	0.0370	0.0240	1.54
OR1G1 OLFACTORY RECEPTOR GENE	939	0.0170	0.0190	0.0130	1.46

The coding sequences analyzed in this study show that in the 73,104 bases studied, humans and chimpanzees are 99.01% similar. This value is approximately 0.5% higher than the estimated total genomic differences shown by previous DNA-DNA hybridization studies (Sibley et al., 1990). Most of the genome is noncoding DNA, and therefore the total genome value shows more difference than the coding comparisons. This is because most coding DNA is experiencing the effects of purifying selection and is less likely to change, as most mutations in coding DNA are deleterious. It has been estimated that our genome contains approximately three billion base pairs, of which only 1.5% represent gene coding nucleotide positions. Thus, we suspect that humans and chimpanzees differ at approximately

445,000 coding positions. Of these differences, the key ones at the nonsynonymous sites are predicted to be found on between 2,850 and 4,000 genes. Finding these differences, along with their promoter differences, is essential to completing the study of the genomic changes that are unique to humankind.

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CHAPTER 2

Primate Genomics: A Rich Resource for Functional Genomics

DEBORAH L. GUMUCIO, DAVID M. THOMAS, PHILIP SCHANER, NEIL RICHARDS, WESLEY MARTUS, ANISH WADHWA, AND MORRIS GOODMAN

REGULATORY CHANGES IN THE PRIMATE γ GLOBIN GENE THAT LED TO EVOLUTION OF A NOVEL DEVELOPMENTAL EXPRESSION PATTERN

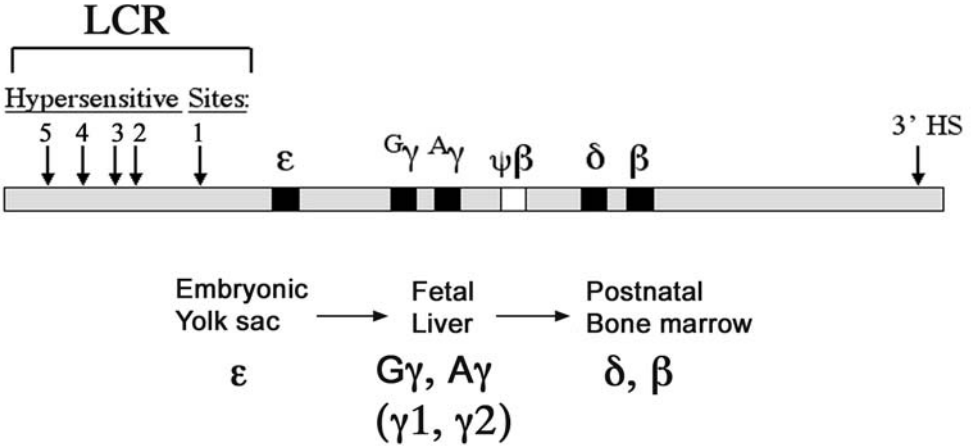
The primate γ globin gene locus provides an excellent example of the role of genetic regulatory changes in shaping new gene expression patterns (reviewed in Goodman et al., 1996). The expression of γ globin in the fetal stage is a pattern unique to anthropoid primates (New World monkeys, Old World monkeys, apes, and humans). In most other non-primate mammals, and in non-anthropoid primates (e.g., the galago and the lemur), γ is expressed exclusively in the embryonic time period and silenced in the fetus (Tagle et al., 1988). Thus, the evolutionary history of this trait (fetal expression of γ) indicates that it first appeared after the separation of the non-anthropoid primates from the anthropoids 58 million years ago (mya) and before the separation of the anthropoid primates into two major groups 40 mya—the platyrrhines (New World monkeys) and the catarrhines (Old World monkeys, apes, and humans). All platyrrhines and catarrhines thus far examined express γ in fetal life, though the timing of γ silencing in these two major groups is different (Goodman et al., 1996).

Interestingly, the expression of γ in a different developmental stage also involves a movement in the spatial aspect of γ expression within the developing organism. While embryonic or primitive erythropoiesis occurs in the yolk sac, fetal (definitive) erythropoiesis takes place initially in the fetal liver. Thus, the "fetal recruitment" of γ requires the expression of γ in a new environment, the fetal liver.

Expression of this new protein (γ) in an environment in which it was previously not expressed has the potential to place additional selective pressures on the protein itself. Thus, as a result of the regulatory change, a protein coding change can occur. This is in fact believed to be the case for γ, as discussed further below.

To understand the full impact of a change in the regulatory pattern of the γ gene, and to eventually decipher the causes of such a change, it is necessary to examine the chromosomal locus in which this gene resides. The β–like globin gene cluster encompasses more than 100 kb of sequence (Figure 1). Five active genes, each with a distinct developmental expression pattern, compose the β–like globin cluster in humans and other anthropoid primates. The ε gene is exclusively embryonic and is silenced in fetal life. The two γ genes (both of which were derived by gene duplication 58 to 40 mya; Fitch et al., 1991) are also expressed at low levels embryonically, but at the beginning of fetal life they are up-regulated, and they provide the majority of the β–like globin chains throughout fetal life. The "switch" from embryonic ε to fetal γ expression at the end of embryonic life is followed by a second switch, from fetal to postnatal expression, that occurs at birth. This latter switch involves the down-regulation of the two γ genes and the up-regulation of the "adult" δ and β genes. The δ gene is weakly expressed in most individuals, and the adult β gene is the major contributor of β–like protein to the adult hemoglobin molecule.

Figure 1. Schematic representation of the human β-like globin locus. The locus encompasses over 100 kb of DNA and contains five active genes (black boxes) and one pseudogene (white box). Upstream and downstream from the genes are regions of altered chromatin structure detected as hypersensitive sites (HS). The locus control region (LCR) comprises the cluster of hypersensitive sites upstream of the locus. Two transcriptional switches in gene activity characterize this locus: the ε to γ switch that occurs at the end of embryonic life, and the γ to β switch that occurs at birth.



Despite decades of study, the molecular mechanisms that control these two switches in gene activity within the β–like cluster are unknown. In addition to the genes themselves, the β cluster contains important upstream regulatory elements within a region called the locus control region, or LCR (reviewed in Li et al., 1999). Deletion of the LCR results in down-regulation of the expression of all of the genes in the β–like cluster (Reik et al., 1998). The LCR itself is detectable in chromatin as a series of hypersensitive sites, indicating altered or open chromatin structure. Within these regions lie powerful enhancers, chromatin-modifying elements, boundary or insulator elements, and matrix attachment sites.

Two current models, called the looping model and the linking model, have been put forward to explain hemoglobin switching; both can explain current experimental data, and it is not clear which is more correct. According to the looping model, the five globin genes compete for interaction with the LCR (Wijgerde et al., 1995). The LCR acts as a holocomplex that is capable of interacting with each of the genes, and the LCR itself is limiting in these interactions. Inherent in this model is the possibility that the LCR can actually flip-flop between different genes at any developmental time. The length of time that the LCR remains engaged by a given gene in a given developmental time period depends on the stability (or half-life) of the interaction in that time period. For instance, the half-life of the LCR:γ interaction in the fetal life is long, while that of the LCR:β interaction is short. Near birth, there is a shift that strengthens the LCR:β interaction and prolongs its half-life relative to LCR:γ; thus, the γ to β switch occurs. Though there is no physical evidence for this model, it is widely accepted and consistent with present data.

The second model, called the linking model, was outlined recently by Bulger and Groudine (1999). Initially proposed to account for long-range promoter:enhancer interactions in *Drosophila* (Dorsett, 1999), this model posits that DNA-binding proteins plus non-DNA-binding "facilitators" form a continuous protein chain that serves to link the LCR to a gene promoter. Such facilitators (e.g., chip, nipped-B) have been identified in genetic screens for factors that promote long-range enhancer:promoter interactions (Morcillo et al., 1997; Agulnick et al., 1996). The idea behind this model is that the promoter by itself is unable to recruit some limiting activity, but linking the promoter to the enhancer by the protein chain provides that limiting factor. Local perturbations of the chain, perhaps provided by insulator-like sequences located in promoter regions (Dorsett, 1999; Bulger and Groudine, 1999), could provide a means for expression of γ and not the distal β gene in the fetal stage. Once γ is transcriptionally silenced after fetal life, the chain could continue to β. Importantly, the linking model also proposes that the protein chain links are flexi-

ble or transitory, so that the LCR could be linked to γ at one moment and to β at the next (Dorsett, 1999).

With these models in mind, we can speculate as to what kind of evolutionary change might result in alteration of the expression pattern of γ. At least two possibilities exist: (1) The *trans* model predicts that the ancestral fetal liver environment could not support an interaction between the γ gene and the LCR. Fetal recruitment, therefore, consisted of alterations in the *trans* environment such that this interaction was permitted. (2) The *cis* model predicts that *cis* changes in the DNA occurred. Such changes either established a permissive interaction between the γ gene and the LCR in the fetal stage or removed an element that previously inhibited LCR:promoter interaction in that stage.

We have tested these two possibilities using the transgenic mouse model (TomHon et al., 1997; Yu et al., 2002). We designed the four transgenic constructs shown in Figure 2 (on page 15) and generated multiple lines of mice carrying each construct. Each construct contains a human ε gene, which not only serves as a control for embryonic expression but also separates the γ gene from the LCR by 5 kb, ensuring that long-range promoter:enhancer interactions are necessary to activate γ. Enhancers from different regions of the LCR (HS2 and HS3) were used to test whether the LCR itself plays a role in the expression choice made by the human (fetal) or galago (embryonic) gene.

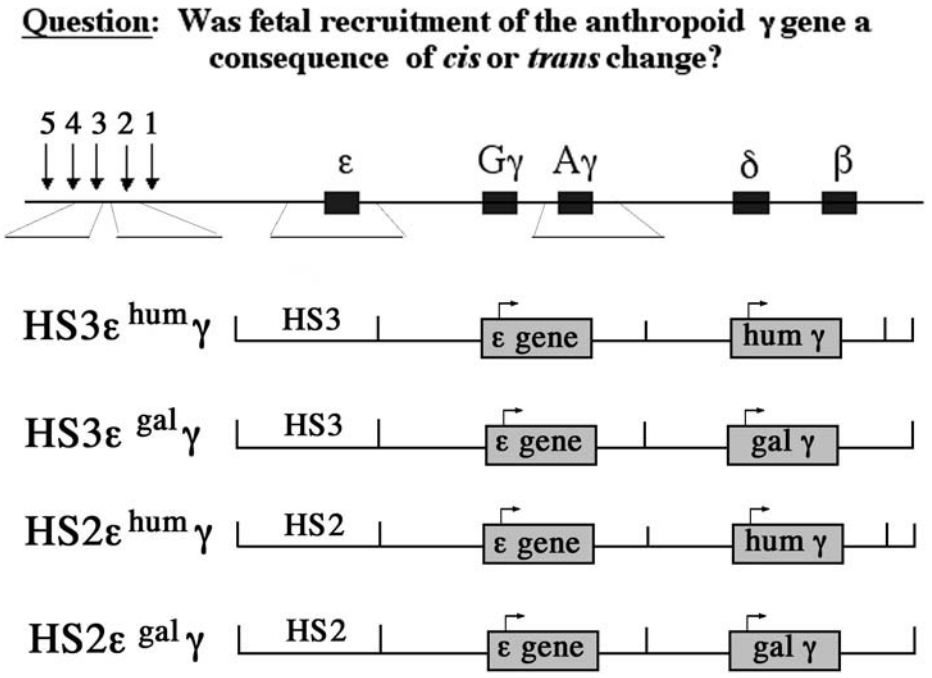
If evolutionary changes in the *trans* environment account for the fetal expression of the anthropoid γ gene, then we would expect that the galago and human γ genes should be expressed similarly to one another when placed in the same environment (the mouse fetal liver). In fact, we would expect that both genes would be expressed embryonically, since this is the expression pattern exhibited by the endogenous mouse γ homolog, βh1. On the other hand, if evolution of DNA sequences in the globin cluster itself are responsible for fetal recruitment, then we would expect the human γ to be expressed in fetal life and the galago γ gene to be embryonic (assuming that the constructs have included the key *cis* sequences involved).

Figure 3 (on page 16) shows the cumulative results for all four constructs tested (TomHon et al., 1997; Yu et al., 2002). The experiments encompassed a total of 15 independent transgenic lines, seven of which carried galago γ transgenes and eight of which carried human γ transgenes. The results are strikingly consistent. The galago γ genes are regulated similarly to the human ε genes: expressed at high levels in embryonic life and silenced in the fetal liver. The human γ genes are expressed variably in embryonic life but are further activated in the fetal liver environment. These data indicate that *cis* sequences are responsible for these distinct expression patterns. Moreover, both regions of the LCR (HS2 and HS3) support these distinct γ expression patterns, indicating that the critical *cis* differ-

ences are located in the 4 kb γ gene fragments themselves.

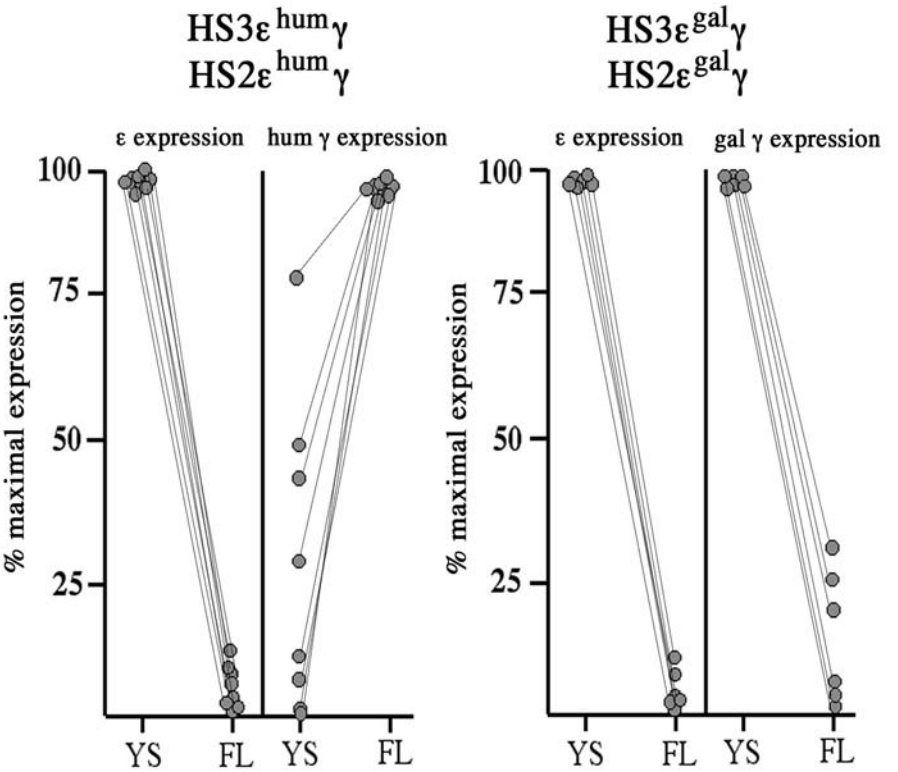
These findings permit the synthesis of a more detailed evolutionary model for the acquisition of a fetal expression pattern by the γ gene. Figure 4 (on page 17) shows that the γ gene was duplicated in the same evolutionary time window in which fetal recruitment occurred (58 to 40 mya). This duplication was brought about by an unequal crossover involving repetitive line elements situated on either side of the γ gene (Fitch et al., 1991). It is not clear from the present data whether fetal recruitment preceded or postdated the γ duplication event, but the following working model is a parsimonious reconstruction that is consistent with all available data. This model proposes that the γ duplication was the initial event and pro-

Figure 2. Transgenic constructs used to examine the mechanism of fetal recruitment of the anthropoid γ gene. The human β-like globin cluster is shown at top. The HS3ε^{hum}γ construct includes a 1.9 kb Hind III fragment containing the core of HS3; a 3.7 kb EcoRI fragment containing the human ε gene; and a 4.0 kb fragment encompassing two adjacent HindIII fragments that contain the human γ gene (TomHon et al., 1997). The HS3ε^{gal}γ construct contains the same HS3 and ε fragment, but a 4.4 kb galago γ gene fragment is substituted for the human γ gene fragment. The HS2ε^{hum}γ and HS2ε^{gal}γ constructs are identical to the HS3-containing constructs, except for the substitution of a 1.9 kb fragment encompassing the human HS2 core region for the HS3 region in both the constructs (Yu et al., 2002). All four of these constructs were tested in transgenic mice (TomHon et al., 1997; Yu et al., 2002).



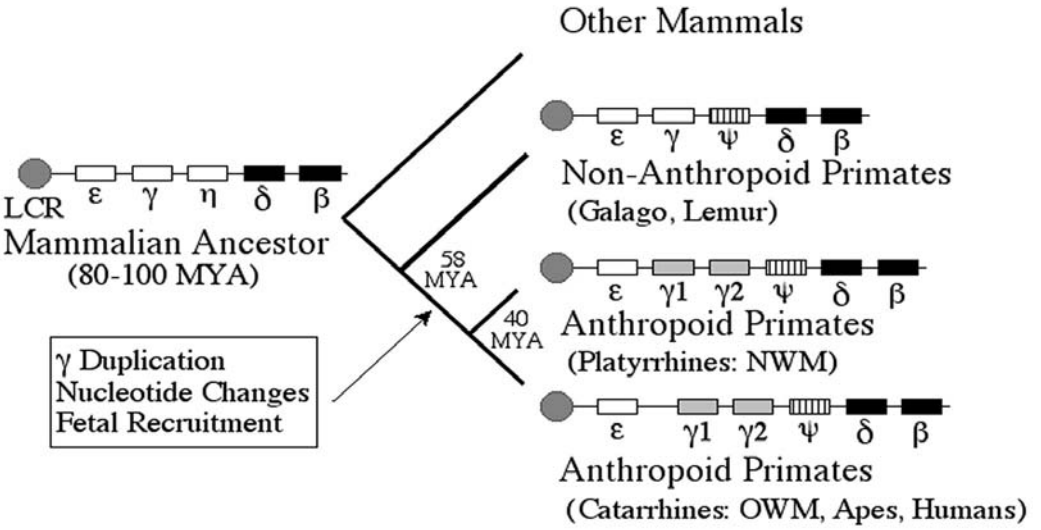
vided a substrate (the recently duplicated γ gene) that was redundant and therefore free to collect changes that could alter its expression pattern. Initially, this γ2 gene was probably embryonically expressed (as was its ancestor) but was likely to be very poorly expressed because of its increased distance from the LCR and because of competition from the two other embryonic genes (ε and γ1) interposed between it and the LCR (Chiu et al., 1999). The relaxed functional constraints on this relatively silent and redundant gene could permit it to gain nucleotide substitutions. If those changes altered the expression pattern, and if this altered pattern was then selected for,

Figure 3. Expression of the HS3ε^{hum}γ, HS3ε^{gal}γ, HS2ε^{hum}γ, and HS2ε^{gal}γ constructs in transgenic mice. The left panel shows results when the human γ gene is contained in the construct; results for both HS3- and HS2-driven constructs are combined. In both cases, the human ε gene is embryonic and silenced in the 14 day fetal liver, while the human γ gene is expressed at variable levels in embryonic life, but consistently at high levels in the 14 day fetal liver. The right panel shows results when the galago γ gene is contained in the construct; results for both HS3- and HS2-driven constructs are combined. In both cases, the human ε gene is embryonic and silenced in the 14 day fetal liver; the galago γ gene is also expressed embryonically, in a pattern very similar to that of the human ε gene.



we would expect evolutionary reconstructions to reveal bursts of nucleotide change that can be traced to this evolutionary window (58 to 40 mya) and that have been conserved since then by purifying selection. This is exactly what is observed (Fitch et al., 1991; Goodman et al., 1996). Importantly, then, if these changes altered the γ expression pattern and the gene was expressed in the new (fetal) environment, then the protein itself would be subject to new selective pressures. One of these pressures during the evolution of the anthropoid primates was surely the pressure to prolong the gesta-

Figure 4. Evolutionary history of the primate globin genes. The LCR is depicted as a gray circle at the 5' end of each cluster; clusters are not drawn to scale. White boxes = embryonically expressed genes; black boxes = post-embryonically expressed genes; gray boxes = fetally expressed genes; striped boxes = pseudogenes. Though not diagrammed here, it is clear that prior to the separation of marsupial and placental mammals 135 million years ago (mya), two globin genes existed (Goodman et al., 1996): one (ε) was embryonically expressed, while the other (β) was expressed in post-embryonic life. This two-gene cluster, 5'-ε-β-3', is still seen in marsupials. But in early placentals, ε duplications produced three embryonic genes (ε, γ, and η), and a β duplication produced two post-embryonic genes (δ and β). A globin cluster related to this ancestral five-gene cluster (shown at left) has persisted in all extant eutherian mammals. Three events characterize the evolutionary window between 58 and 40 mya: duplication of the γ gene; nucleotide changes in regulatory and coding regions; and the alteration in the expression pattern of γ, causing this previously embryonic gene to be recruited for fetal expression. All anthropoid primates have two γ genes that are both fetally expressed. See text for additional details.



tional period to allow increased development of a more complex brain. During this prolonged gestation, oxygen supplies would be critical, and the evolution of a γ globin that has alterations in its diphosphoglycerate (DPG) binding site that serve to increase oxygen affinity for the fetal blood would be imagined to be highly beneficial. Such coding changes are indeed seen in the present-day anthropoid γ gene, and these changes do have an effect on oxygen affinity in fetal hemoglobin. Finally, to account for the finding that in all anthropoid primates, both of the γ genes are fetal and carry the DPG changes that affect oxygen affinity, it is necessary to evoke a gene conversion in which regulatory and coding changes accumulated in the γ2 gene could be copied onto the γ1 gene. Indeed, we have found evidence for an ancient gene conversion with this polarity (Chiu et al., 1997; Hayasaka et al., 1993).

A related evolutionary aspect to contemplate is the fact that the timing of the γ to β switch is different in the major primate branches. In galagos and other non-anthropoids, this switch occurs at the end of embryonic life; in anthropoids, it occurs in mid-fetal (platyrrhines) or late fetal (catarrhines) life (Tagle et al., 1988; Johnson et al., 1996). The fact that in the transgenic mouse model, the galago γ gene is consistently silenced at the end of embryonic life suggests that alterations in the timing of γ silencing (rather than in the timing of β activation) may be the major factor driving these differences in switch timing. This possibility could be directly tested by substituting a galago β gene for a human β gene in the context of an otherwise complete human globin gene locus.

Given that *cis* differences are responsible for the distinct developmental expression patterns of the human and galago γ genes in the murine background, how can the key *cis* differences be pinpointed? To approach this, we developed a strategy called differential phylogenetic footprinting (Gumucio et al., 1994). This strategy involves the following steps: (1) Align all available non-anthropoid primate (and non-primate) γ gene sequences (i.e., those that are embryonically expressed) and derive a consensus sequence. (2) Similarly align all available anthropoid γ gene sequences (those that are fetally expressed) and derive a consensus sequence. (3) Compare the two consensus sequences, looking for unambiguous differences between the two. (4) Where such differences are found, determine whether the sequence differences cause changes in the pattern of nuclear factors that bind to the region. (5) Where such binding differences are found, determine if these result in alterations in expression profile in cell-based or transgenic mouse systems.

Applying differential phylogenetic footprinting to the γ globin gene, we have identified several regions in the proximal and distal γ promoter at which sequence differences cause differential binding of nuclear factors to orthologous human and galago *cis* elements. These

include the –1086 region, where a YY1 binding site is located on the opposite strand in the human and the galago (Zhu et al., 1999); the –175 region, where the galago γ gene fails to bind Oct-1 (Gumucio et al., 1990); the –140 CACCC region, where the galago γ sequences fail to bind CP-1/NF-Y (Gumucio et al., 1994); the –88 CCAAT box region, where a number of cell-type-specific proteins bind to the galago sequences but not to the human sequences (Gumucio et al., 1994); the –50 region, where SSP (stage-specific protein) binds to human but not to galago sequences (Jane et al., 1992). Of these, only the human YY1 site at –1086 has been tested in transgenic mice (Zhu et al., 1999); ablation of this site results in failure of activation of the γ gene in fetal life. In cell-based assays, functional differences between human and galago sequences have been detected for the –175 (Gumucio et al., 1990), –88 (Gumucio et al., 1994), and –50 (Jane et al., 1992) regions; these need now to be tested in the mouse model. For the –140 (CACCC) region, binding alterations for NF-Y are detectable, but human and galago sequences are phenotypically indistinguishable in transient cell-based assays (Gumucio et al., 1994). Given the critical importance of this element to expression of all the β–like globin genes, these changes need to be more carefully tested in a transgenic mouse model. Indeed, recent work indicates that recruitment of NF-Y to the duplicated CCAAT box region plays a role in the chromatin opening of the gamma gene promoter, as well as in the communication between the gamma gene promoter and the LCR (Duan et al., 2001).

It is clear that a significant amount of work must be done in order to prove that specific *cis* elements identified by differential phylogenetic footprinting represent key elements responsible for driving distinct expression patterns. The problem is particularly difficult in a locus such as the β–like locus, where gene competition and long-range promoter:LCR interactions make regulatory networks difficult to unravel. In addition, it is important to recall that since the burst of nucleotide changes that altered γ gene expression occurred during a time when the gene was probably weakly expressed (or not expressed), it is possible that more than one *cis* change occurred. This could be tested by the creation and testing of chimeric human/galago γ genes (currently under way). The eventual success of these functional studies has the potential to reveal regulatory elements that are important for globin switching and for gene:LCR interaction. Identifying these *cis* elements is the first step in understanding the basic mechanisms underlying these processes.

EVOLUTION OF CODING SEQUENCES: LESSONS FROM THE FAMILIAL MEDITERRANEAN FEVER (MEFV) LOCUS

In contrast to the β–like globin genes, which have been under functional and evolutionary scrutiny for decades, the MEFV locus is a

recently identified, novel gene (French FMF Consortium, 1997; International FMF Consortium, 1997). Mutations in pyrin, the protein product of the MEFV gene, cause familial Mediterranean fever (FMF), an acute inflammatory disorder. Patients with FMF suffer inflammatory attacks that last one to three days and are accompanied by fever and severe pain localized to the chest, abdomen, or joints (usually monoarticular). The attacks are characterized by neutrophil infiltration of the pleura, peritoneum, synovium, or, less commonly, skin or muscle (reviewed in Kastner, 1998). Some FMF patients also develop amyloidosis leading to chronic renal failure.

The pyrin protein enjoys a restricted expression pattern (French FMF Consortium, 1997; International FMF Consortium, 1997; Centola et al., 2000; Matzner et al., 2000) and is detectable only in certain white blood cells (neutrophils, monocytes, and eosinophils) and in certain fibroblasts (skin, peritoneal, and synovial). Based on the inflammatory nature of FMF, it has been proposed that the pyrin protein acts as a rheostat to control the inflammatory response of the neutrophil, though the manner in which this thresholding action is exerted is not known.

Thus far, 22 different pyrin mutations have been identified (French FMF Consortium, 1997; International FMF Consortium, 1997; Bernot et al., 1998; Booth et al., 1998; Samuels et al., 1998; Aksentijevich et al., 1999; Cazeneuve et al., 1999; Dode et al., 2000; Domingo et al., 2000; Notarnicola et al., 2001; Schaner et al., 2001). All but one are either single codon (trinucleotide) deletions or missense mutations, and most of the missense changes are very conservative in nature. A single truncation (stop codon) mutation has been observed that results in the production of a pyrin molecule that lacks the last half of the C-terminal exon, exon 10 (Notarnicola et al., 2001). The idea that exon 10 is a functionally important domain is reinforced by the fact that the majority (67%) of FMF mutations are found in this exon, which encodes an rfp or B30.2 domain. This domain is found in a wide variety of proteins and has been predicted to participate in ligand binding or cell signaling (Schultz et al., 1998), but no definitive function has yet been assigned.

Interestingly, studies in human populations indicate that several of the known pyrin mutations appear to be under positive selective pressure (Aksentijevich et al., 1999; Booth et al., 2000; Stoffman et al., 2000). The frequency of these mutant alleles in several different human populations is strikingly high, approaching 1:5 for Arabs and 1:7 for Armenians. Though the selective pressure is not known, it has been suggested that carriers of one mutant allele may have a heightened ability to clear a devastating pathogen (Aksentijevich et al., 1999; Booth et al., 2000; Stoffman et al., 2000).

We recently tested whether the rfp domain of pyrin—the domain that carries most of the human mutations—has been under positive

selection during primate evolution (Schaner et al., 2001). Using ClustalW, we sequenced the rfp domain from 20 primates and 2 non-primate mammals and aligned protein and translated amino acid sequences. Alignments were then used to generate phylogenies. The strict consensus nucleotide-based tree shown in Figure 5 is topographically highly consistent with cladistic relationships based on extensive fossil and molecular evidence (Goodman et al., 1998). Bootstrap values for the nucleotide phylogeny (numbers along branches in Figure 5 on page 22) indicate that this topology is robust. The resolution within the primates is surprisingly detailed given this short (480 bp) nucleotide sequence and the recent divergence of some of these species.

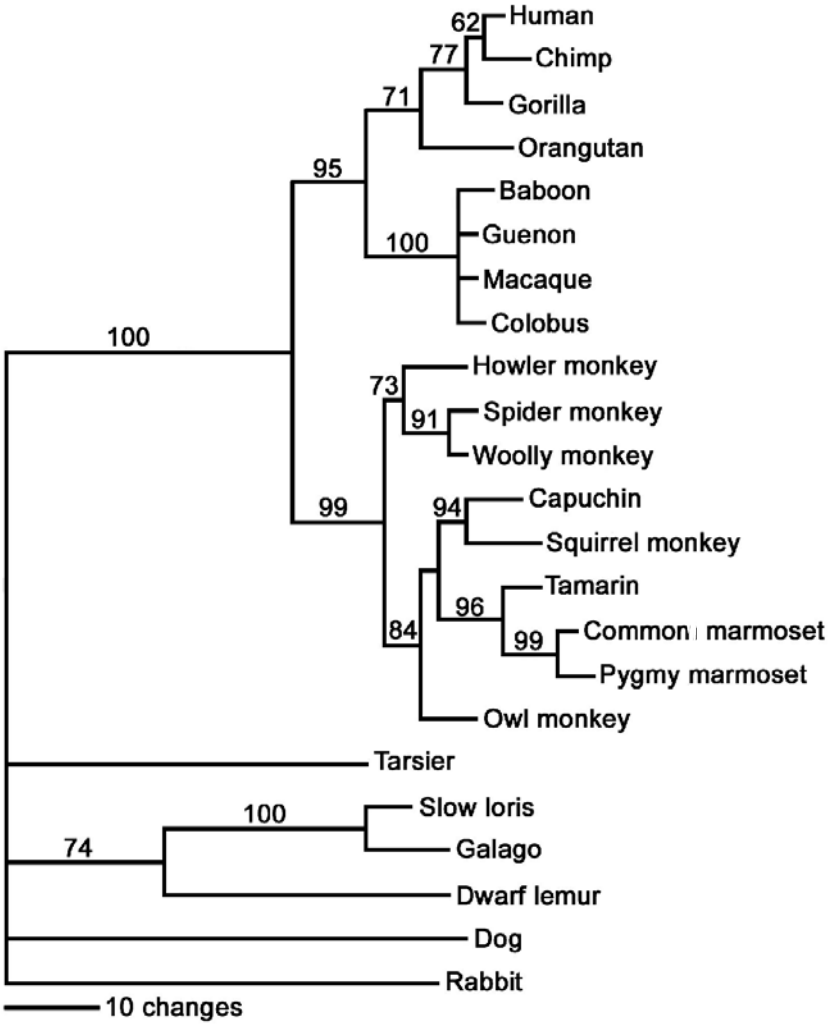
The high resolution observed in our phylogeny suggested a mechanism for this functional change—namely, rapid evolution. In order to investigate this question further, percent divergence values were calculated for this region of pyrin (Murphy, 1993). Between dog and human, 25% of amino acids have changed identity. Analysis of over 600 proteins has revealed that the average rate of divergence between human and rodent for most intracellular proteins ranges from 2 to 12% (Murphy, 1993). Extracellular proteins, such as plasma and exocrine proteins, diverge an average of 28%, while host defense ligands and receptors diverge an average of 35%. The rate of divergence of pyrin is twice that of intracellular proteins and more similar to rates seen in extracellular proteins. Interestingly, although pyrin lacks a consensus secretory signal sequence, other members of the rfp domain family of proteins (stonustoxins A and B) are secreted without one (Ghadessy et al., 1996), as are some pro-inflammatory molecules (e.g., IL-1β) (Rubartelli et al., 1990). The possibility that pyrin is secreted needs to be experimentally tested.

Pyrin's rapid evolution could be due to positive Darwinian selection or to relaxed functional constraint. Positive selection can be directly assessed using the ratio of non-synonymous substitutions per non-synonymous site (d_N) to synonymous substitutions per synonymous site (d_S). We therefore used maximum likelihood to examine the d_N/d_S ratio (Goldman and Yang, 1994; Yang, 1998). When the d_N/d_S ratio is greater than 1.0, this is strong evidence of positive selection; the d_N/d_S ratio for most proteins ranges from 0.3 to 0.03 (Li et al., 1987). We found evidence for high d_N/d_S ratios on several of the evolutionary branch points that mark major cladistic separations during primate history. For example, the separation of platyrrhines (New World monkeys) from catarrhines (Old World monkeys, apes, and human) is marked by a d_N/d_S ratio of 1.5 on the branch leading to the New World monkeys.

When the specific amino acids that have been mutated in FMF were examined, an even more striking picture emerged. At 7 of the 10 mutant positions studied, amino acid residues that are considered

mutant in humans were found as wild-type in primates (Schaner et al., 2001). As it is unlikely that the primates that carry these "mutant" amino acids all suffer from FMF, we interpret this result to indicate that amino acid changes at these positions cause changes in the function of pyrin. This situation (in which a mutation recapitulates

Figure 5. Consensus phylogeny for pyrin nucleotide sequences. This phylogram was constructed with PAUP (phylogenetic analysis using parsimony) 4.0b8 (from the 480 bp nucleotide sequence). Maximum parsimony was used as the optimality criterion, and trees were obtained via a heuristic search (with random addition of branches and 100 replicates) (Swofford, 1998). Three equally parsimonious trees were obtained, varying only in the arrangement of the Old World monkeys (collapsed in the strict consensus). The tree was rooted using dog and rabbit as outgroups. Bootstrap values, when greater than 50, are shown along lineages (average of 100 resamplings).



sequences of homologues in other species, as in convergent evolution) has also been noted for the α–synuclein gene identified in individuals with Parkinson's disease (Polymeropoulos et al., 1997). In this case, a single alanine to threonine mutation at position 53 is associated with disease. The rat and the zebra finch also carry threonine at this position.

In the case of pyrin, a common pattern is that the human mutations actually recapitulate ancient sequence states seen during primate evolution (Figure 6 on page 24). For example, at position 761, the wild-type amino acid is arginine. The mutant amino acid is histidine. Examination of the primate sequences show that only humans and apes carry arginine at this position. All non-primate mammals, all prosimian primates (except the fat-tailed dwarf lemur), all New World monkeys, and all Old World monkeys carry histidine instead. Thus, the change from arginine to histidine was a relatively recent event that occurred after the separation of Old World monkeys from the apes. Yet a change back to arginine causes FMF in humans. Together, these data suggest that pyrin has been evolving through positive selective pressure. Interestingly, changes in pyrin sequence often correlate with major cladistic branch points, suggesting that as species encounter new environments, new selective pressures may force a change in pyrin sequence.

Though the selective agent itself is not known, these data fit well with the possibility that pyrin interacts directly with pathogens, or with other molecules of the innate immune pathway, which also evolve rapidly. Furthermore, the evolutionary data suggest that the mutations may be imparting a novel function to pyrin (as opposed to a reduced function). Indeed, recent studies indicate that heterozygotes carrying one pyrin allele do have a phenotype, albeit a laboratory phenotype (i.e., elevation of CRP and SAA; Tunca et al., 1999). In addition, one group has found evidence of disease exacerbation in heterozygotes (Booth et al., 2000). Thus, rather than being a recessive disease, as first imagined, FMF may actually be a dominant disease of reduced penetrance. The mutations in the rfp domain, at least, might be gain-of-function mutations.

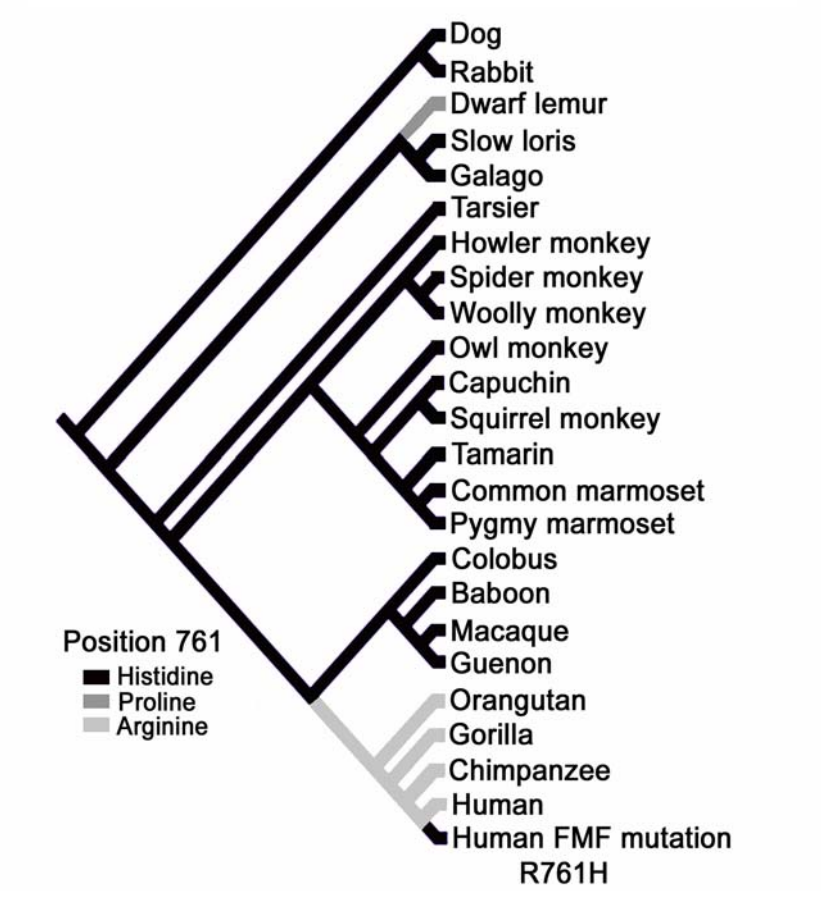
USING EVOLUTIONARY STRATEGIES TO IDENTIFY GENES ASSOCIATED WITH HUMAN DISEASE

The genetic changes that cumulatively endowed human characteristics were probably selected for during millions of years of primate evolution. These changes were selected because they endowed new or better function for the species in a given environment. However, when environmental change outpaces genetic change (as it often does), traits selected in one environment can be unsuitable or detrimental in another—an effect that can account for the apparently paradoxical fact that a number of devastating human diseases seem to

have been selected for during human evolution, including sickle cell anemia, attention deficit disorders, cystic fibrosis, schizophrenia, manic depression, and susceptibility to AIDS (reviewed in Nesse and Williams, 1996).

It follows from the above that large-scale strategies aimed at identification of genes that were subject to positive selection during primate evolution will pinpoint some of the genes that shaped our phenotype. Moreover, many of these same genes are likely to have major health implications. To survey the primate genomes for positively selected coding changes will require that the complete genomic sequences of a number of primates be obtained. Most beneficial would be those of the chimpanzee, orangutan, baboon, capuchin monkey, spider monkey, and galago. These genomes together would

Figure 6. History of amino acid change at amino acid position 761. The history of position 761 is superimposed onto a total evidence phylogeny (Goodman et al., 1998), using MacClade (Maddison and Maddison, 1992). The different shades of gray show the most parsimonious inferred evolutionary history of amino acid change. Shadings correspond to different amino acids, as detailed in the figure. The results emphasize the fact that the 761 mutant character state is identical to the ancestral character state.



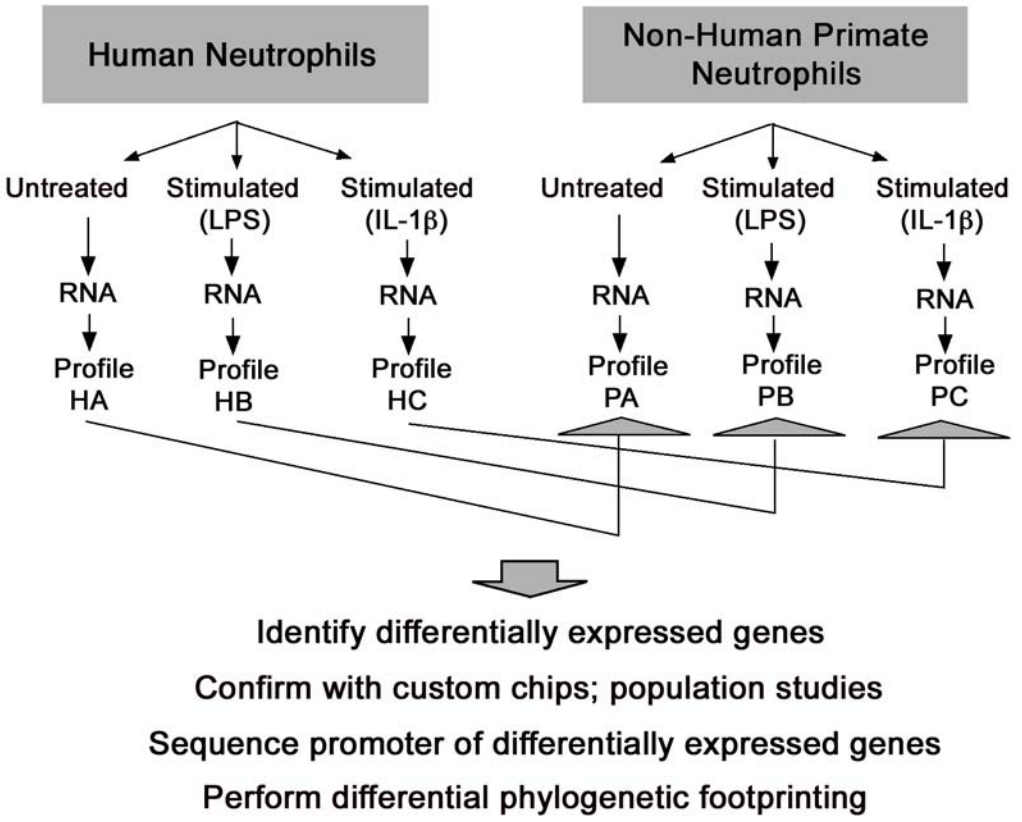
provide an excellent and deep evolutionary database that would be useful for phylogenetic analysis. However, it will be some time before all of these sequences are available, making an immediate survey of positively selected coding regions a difficult task.

However, other strategies can be used to examine evolving regulatory elements. On the basis of our experiences in the globin cluster, we propose that differential phylogenetic footprinting can be utilized on a larger scale to identify such species-shaping regulatory elements, and we outline a possible strategy below.

To begin an analysis of selected regulatory changes that shaped the human species, we would select a tissue that is known to interact with the environment. For example, a likely target is the neutrophil, the white blood cell critical for innate immune function, the mediator of the immediate immune reaction against bacteria, viruses, parasites, and other pathogens (Hoffman et al., 1999). Other equally suitable tissues that interact with the environment include skin, gut, and brain frontal cortex. However, the neutrophil has another advantage in that it is easy to obtain via minimally invasive techniques. Figure 7 (on page 26) outlines a possible scheme for the identification of genes that are differentially expressed in human and non-human primates (e.g., chimpanzee or baboon) neutrophils. These genes could be directly involved in the species-specific reaction to pathogens by the innate immune system or the species-specific reaction to inflammatory triggers. Human and non-human primate neutrophil mRNA would be isolated, and messages that are differentially expressed would be assessed either by message profiling (serial analysis of gene expression, or SAGE; Velculescu et al., 1995) or by microarray strategies. To also assess differences in neutrophil activation, it would be prudent to survey unstimulated neutrophils, as well as neutrophils stimulated with lipopolysaccharide (to simulate bacterial activation) and with IL-1β (to simulate inflammatory activation). Custom microarray chips designed to represent the mRNAs found to be differentially expressed can be used to confirm initial findings and to establish levels of variation inherent in human and non-human primate populations. An interesting side note: It is possible that a survey of humans with different acute and chronic inflammatory conditions will show a distinct signature on such a chip. Once specific mRNAs are confirmed as differentially expressed in humans and chimps, these candidates can be subjected to differential phylogenetic footprinting, as described above, to identify the genetic changes involved in these expression differences.

Of course, this strategy assumes that *cis* changes dictate the expression differences and that transcriptional control is involved; where these assumptions do not hold, this strategy cannot be applied. However, recent work in a variety of biological model systems has shown that *cis* changes in gene regulatory regions, rather

Figure 7. Scheme for the detection of neutrophil genes that are differentially expressed between human and non-human primates. Human and non-human primate (e.g., baboon, chimpanzee) neutrophils are isolated. One aliquot is left untreated (A); a second aliquot is treated with LPS (B); and a third aliquot is treated with IL-1β (C). mRNA is prepared from all human (H) and non-human primate (P) samples and used to generate SAGE (Velculescu et al., 1995) libraries for mRNA profiling, or used to perform microarray analyses using commercially available chips. All identified differentially expressed messages are used to create a custom chip that can be retested to establish the normal degree of variation in these messages among the human and primate populations. In addition, the results would confirm specific candidate mRNAs as differentially expressed in humans and primates. For those differentially expressed candidates, phylogenetic footprinting would be applied. Thus, the first 1 to 2 kb of presumed promoter sequence would be identified in the human genomic database. On the basis of this sequence, primers would be generated to amplify the corresponding region from chimpanzee, orangutan, two Old World monkey species and two New World monkey species. The galago sequence will also be determined where practical, and the mouse genomic sequence may be available soon. Alignments of these sequences would be generated and used to identify sequence differences that are human-specific, catarrhine-specific, or anthropoid-specific. Those that fit these criteria would be subjected to nuclear factor binding studies and, finally, to functional analysis in appropriate cell lines or in transgenic mice.



than alterations in protein coding sequences, provide the major driving force for evolution of morphological diversity (Carroll, 1995). The value of the differential phylogenetic footprinting approach is that it uses the predictive power of evolution to rapidly target functional experiments to identify evolving regulatory elements that could account for phenotypic change and biological diversity. Though it is clear that the rate limiting step in the final identification of the specific *cis* sequences that account for expression changes is the functional proof of the regulatory role of those elements, it should also be immediately obvious that it is the differentially expressed gene itself that holds the promise of understanding the basis for the phenotypic differences between humans and non-human primates, and the basis for human-specific disease susceptibility.

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CHAPTER 3

Complexity and Adaptability in Human Evolution

RICHARD POTTS

The principal idea of this paper is that a connection exists between the evolution of biological complexity and adaptability in organisms. This hypothetical connection is underexplored in evolutionary biology, perhaps because adaptable responses to changing environments are more difficult to characterize—especially in natural settings—than the match between an adaptation and any single habitat in which it is currently found. Regarding hominin evolution, however, the question of adaptability and how it evolves is central to understanding the ecological and adaptive history of humans.

Let me introduce the idea of adaptability with an example drawn not from paleoanthropology but rather from the study of slime mold. While recently reading the book *Signs of Life* by Ricard Solé and Brian Goodwin (2000), I was reminded of the peculiar life cycle of the cellular slime mold *Dictyostelium discoideum*. Individual amoebas of this species typically inhabit moist environments, where they grow, divide, and eat bacteria in rotting vegetation. When dry conditions set in, bacteria are in low supply, and the amoebas emit a chemical distress signal, cyclic adenosine monophosphate. The release of this molecule leads to a process of aggregation, in which individual amoebas gravitate toward the source of the signal and also begin to emit their own pulses of the molecule. Eventually, each aggregate, consisting of thousands of cells, is transformed into a fruiting body—essentially, a multicellular organism made up of a stalk and a cap of spores. In this multicellular state, other food sources are metabolized, thus preventing starvation. When moist conditions return, the spores germinate and release single-celled amoebas, which go about foraging for bacteria.

Although the multicellular stalk phase might be deemed an adaptation that prevents starvation during dry periods, the entire life cycle of the slime mold represents an ability to adapt to both wet and arid times, to the disparity between bacterial plenty and dearth. It is the

variability of conditions that helps explain the intricacies of the life cycle of *D. discoideum*.

Biologists and nonscientists alike are familiar with the idea that environments have defining characteristics and that organisms are well matched to the constant or consistent features of specific habitats. Penguins have anatomical features, physiological properties, and dietary specializations that are well tuned to cold Antarctic settings. Aardvarks, by contrast, possess structures and behaviors oriented specifically toward digging, finding, and eating ants in African environments.

The environments in which organisms evolve can, however, be portrayed in at least two ways: first, in terms of their stable or consistent features (i.e., the unvarying qualities that make a habitat recognizable), and second, in terms of their dynamic qualities (i.e., the variable properties of an environment, to which an individual organism and its genetic lineage must accommodate if either is to persist).

Adaptability can be defined in terms of three qualities: the ability of an organism to persist through environmental shifts, to spread to new habitats, or to respond in novel ways to its surroundings. These aspects of adaptability, all of which are evident in human evolutionary history, can be difficult if not impossible to predict from a reconstruction of any single habitat in which that particular organism has lived. Rather, my thesis here is that environmental dynamics and inconsistencies that result from changing adaptive or fitness conditions must be taken into account.

This idea is strongly suggested by events in human evolutionary history, particularly dispersals into climatic regimes unprecedented in the ancestral habitats of earlier species. Biased by present-day hindsight, the apparent theme of human evolution is a change of small, tropical, apelike populations restricted to equatorial Africa into a descendant (the only remaining descendant, *Homo sapiens*) that is distributed worldwide in virtually all terrestrial habitats and capable of altering them in unprecedented ways.

Human evolution has, in fact, been characterized by two different ecological themes: habitat-specific adaptation and increased adaptability. Most hominin lineages were ecologically and geographically constrained. Between 2.3 and 1.4 million years ago (mya), for example, the biped *Paranthropus boisei* (sometimes referred to as *Australopithecus boisei*) appears to have been confined to certain savanna areas of East Africa. Later on, between 200,000 and 30,000 years ago, Neanderthal populations (widely thought to comprise a distinct species, *Homo neanderthalensis*) were limited to seasonally or perennially cold regions of western Eurasia. By contrast, other species, such as *Homo ergaster* and *Homo erectus*, extended the ecological boundary conditions typical of earlier species. They spread over wider geo-

graphic ranges and were evidently capable of living in rather different settings from those familiar to prior hominins. This expansion in adaptive possibilities became exaggerated in *Homo sapiens*. Apparently, some process enabled certain lineages to become decoupled from any single ancestral environment.

The question posed here is, How do significant increases in adaptability come about? All individual organisms live in particular habitats. The survival and reproduction of organisms take place in specific environments. How, then, can an organism's lineage accrue genetic variations that enable it to significantly expand the range of adaptive environments in which it can thrive? The thesis developed in this paper is that the evolution of adaptability may be an unanticipated consequence of individuals living, dying, and reproducing under specific environmental conditions. That is, the evolution of adaptable phenotypes cannot be predicted from habitat-specific selection at any one time or place. Rather, it emerges from the pattern of natural selection in relation to environmental dynamics at various temporal scales.

Evolutionary biology has tended to overlook the issue of how organisms adapt to the inconsistencies in their surroundings and have favored a more static view of environments and adaptive problems. By finding new ways to measure the variability and inconsistency of adaptive settings, it may be possible to resolve how the adaptable properties of organisms have evolved. With its focus on human evolution, this paper examines environmental variability over the past several million years, summarizes recent studies of early human environmental dynamics, and addresses the processes involved in evolving adaptability.

THE SPECTRUM OF ENVIRONMENTAL DYNAMICS

Environmental variability occurs at all time scales. Wet-dry and warm-cold seasonal oscillations are important in the lives of relatively long-lived organisms. Seasonality has a specific rhythm that is predictable to organisms. This is evident, for example, in the Serengeti, in Tanzania, where a multidecade rainfall record shows that each year exhibits a wet and a dry season, and the large, migratory herbivores respond to this pattern in a predictable manner. The annual rhythm of precipitation is embedded, however, in a longer, less predictable pattern of interannual and interdecadal variation. Long-term deviations from the seasonal pattern have a far-reaching impact on organisms and their surroundings, driving changes in metric tons of grass, herbivore population densities, the number of lions, interspecific competition for prey, and so on. While seasonality is important in the lives of organisms, inconsistency in the pattern over the longer term holds a particularly powerful influence on population size, sur-

vival, and reproductive success.

Table 1 presents a conceptual model of environmental variability at different time scales, applicable to regions where wet-dry seasonality prevails. In this model the annual pattern of seasonality gives way to departures from the pattern over a scale of decades and centuries. Over greater time spans, these departures appear to become organized in ways that result in dramatic habitat variability. According to this model, a spectrum of environmental variability exists from the short-term to longer time scales, at which truly significant revisions in the landscape and resources take place, including the reorganization of water bodies, vegetation remodeling, and glacial-interglacial fluctuation. Over time scales greater than 10^4 years, interactions among orbital cycles of insolation create nonlinear shifts in climate at both shorter and longer periodicities.

TABLE 1: A CONCEPTUAL MODEL OF ENVIRONMENTAL VARIABILITY AT DIFFERENT TEMPORAL SCALES, BASED ON LOW-LATITUDE SETTINGS WITH WET-DRY SEASONALITY

TEMPORAL SCALE	ENVIRONMENTAL VARIABILITY
ANNUAL	RAINY SEASON ↔ DRY SEASON
DECADE (10^1 YRS)	MODAL RAINY SEASON → INTENSE RAINY SEASON → RAINY SEASON FAILURE
10^2 TO 10^3 YRS	STRONG MONSOON RUNOFF, LAKE LEVEL RISE, INCREASED C_3 PLANT COVER ↔ DROUGHT, LAKE LEVEL FALL, INCREASED C_4 PLANT COVER, INITIAL DESERTIFICATION
10^4 YRS	PROLONGED STRONG MONSOONS ↔ PROLONGED INTERVALS OF DROUGHT EPISODIC TECTONIC & VOLCANIC IMPACT REORGANIZATION OF HYDROLOGICAL SYSTEMS & VEGETATION COVER, LOCALLY & REGIONALLY
10^5 TO 10^6 YRS.	PROLONGED TEMPERATURE RISE, REDUCED ICE VOLUME, ELEVATED SEA LEVEL ↔ PROLONGED TEMPERATURE DROP, ICE VOLUME RISE, LOW SEA LEVEL; DESERTS WIDESPREAD AMPLIFIED REMODELING OF ENVIRONMENTS ON CONTINENTAL & GLOBAL SCALES

It is crucial to appreciate that long-term climate changes (e.g., ones that correlate with precessional and eccentricity oscillation) are consequences of altered temporal patterns and intensity of seasonali-

ty. Thus, there is a necessary and important connection between short and long time scales of variability. As posited in Table 1, however, a break or discontinuity in the temporal spectrum of environmental change typically takes place between 10^3 and 10^4 years. Environmental remodeling produced by long-term dry-wet or glacial-interglacial oscillation entails wholesale reorganization of the landscape. Thus, it is not a linear result of any given seasonal pattern of rainy-dry or winter-summer oscillation.

The existence of large, long-term environmental shifts is borne out by virtually every paleoclimate record available. Many of these records are derived from deep-sea cores and have proved useful in exploring the environmental conditions of human evolution (e.g., see Vrba et al., 1995). In ocean water, for example, the oxygen isotope ratio ($\delta^{18}O$) varies as a result of fluctuation in temperature, water evaporation, and global ice volume (i.e., the sequestering of ocean water in continental glaciers). The dominant trend in $\delta^{18}O$ (measured in benthic foraminifera) over the past 6 million years has been the enrichment in ^{18}O relative to ^{16}O (Shackleton, 1995), which implies considerable global cooling and glaciation, with associated increases in ocean evaporation, lowered sea level, and continental drying. However, this trend comprises innumerable deviations (i.e., periodic ebbs in ice volume and rises in temperature and sea level). A case can be made that the heightened amplitude of $\delta^{18}O$ oscillation, rather than being mere noise in a signal of ^{18}O enrichment, also represents an important climatic signal of the past 2.5 million years (Potts, 1996b). A compilation of $\delta^{18}O$ records for the late Cenozoic indicates, in fact, that a dramatic rise in the amplitude of $\delta^{18}O$ oscillation has occurred over the past 6 million years (Figure 1 on page 38), corresponding to the period of human origins and the evolution of the modern biota.

The importance of the oscillatory signal is confirmed by terrestrial dust flux, another climate proxy derived from deep-sea cores. Dust flux reflects the input of continental detritus to ocean settings. It varies as a result of change in land vegetation cover and wind strength. Over the past several million years, the general trend has been an increase in dust input, reflecting greater aridity, reduction in tree cover, and periodic shifts in the monsoonal wind pattern (deMenocal, 1995). Spectral analysis of the dust data, however, reveals large episodic shifts in the oscillatory tempo and amplitude. This result indicates that deep-sea dust flux is a remarkable record of environmental variability, not simply marking an aridity trend (deMenocal and Bloemendal, 1995; Potts, 1998b).

It is thus apparent that Pliocene and Pleistocene organisms have faced a great deal of environmental complexity—that is, nested or embedded patterns of variation resulting from the interaction of climate cycles at diverse temporal and spatial scales. Unlike seasonality, the longer-term patterns appear to be largely unpredictable to the

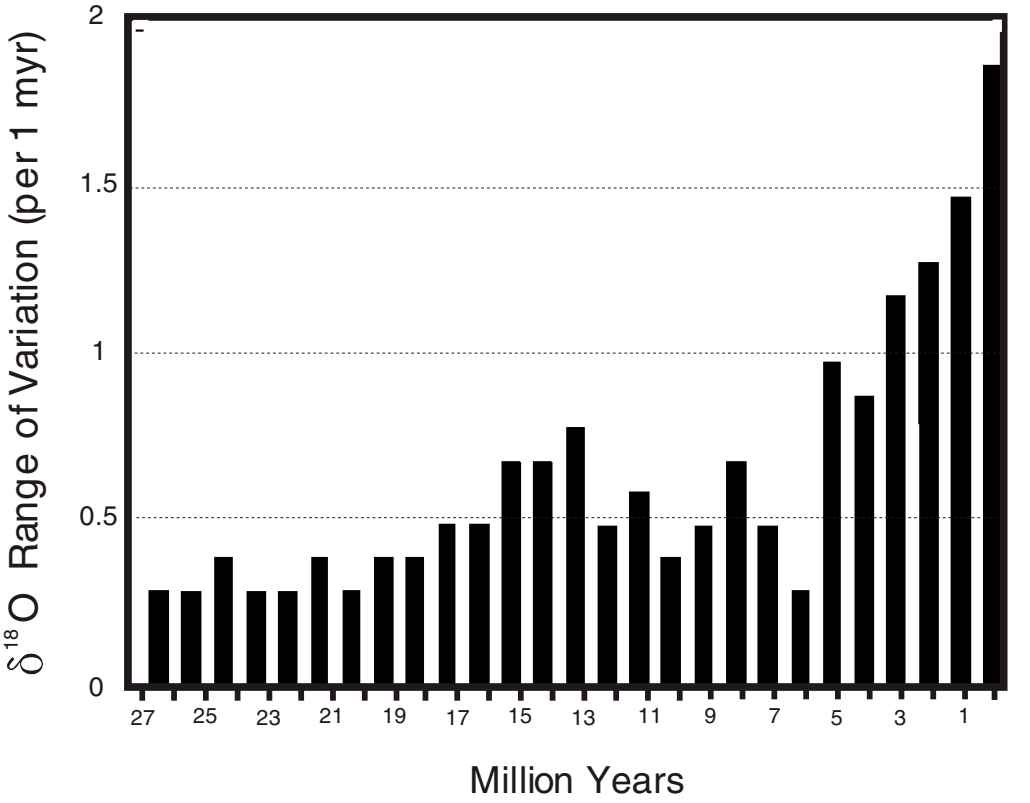
organism. One of the responses to this complexity in the environmental domain is the evolution of adaptability. In simple terms, there are three possible responses by organisms:

Mobility: Organisms track their favored environment or critical resource as it becomes displaced geographically during climate change; this response relies on the organism's ability to move or disperse.

Adaptability: Organisms accommodate to new environmental conditions; this response broadens the range of environments that an organism's lineage can inhabit.

Extinction: The population or lineage becomes extinct because of an inability to accommodate to new environments or to track previously favored ones.

Figure 1. Range of variation in the oxygen isotope ratio ($\delta^{18}O$) in intervals of one million years, from 27 million years ago to the present, based on composite records from deep-sea cores. These data indicate a substantial rise in $\delta^{18}O$ variation, starting in the interval between 6 and 5 million years ago and continuing to the present. (From Potts, 1996a.)



The first two responses enable an organism's lineage to persist through intervals of environmental change. Mobility and resource tracking, on the one hand, serve to stabilize the conditions of natural selection. For an organism that depends on a single type of food or a narrow climatic regime, it may well be possible to consistently follow one particular type of habitat over time. Increased adaptability, on the other hand, suggests that an organism's lineage has faced inconsistencies in its adaptive setting. For an organism whose ecological ties are already diverse, as is the case for many large mammals, it may prove impossible to move in such a way as to replicate prior combinations of resources, competitors, predators, and parasites during a period of climatic change (Williams, 1992). For such an organism, the environment of natural selection is altered along a spectrum from slight to drastic over time. Periodic revamping of selective conditions may be the rule rather than the exception.

To refer back to our introductory example, just as cellular slime mold has become equipped to respond developmentally to environmental variations over short time frames, an organism's lineage (its gene pool through time) may become equipped to respond to novel settings, enabling it to persist across environmental boundaries. The way this occurs is not by adaptation to the environment in the usual way that habitats are defined, as a static set of defining characteristics and adaptive problems posed to particular organisms. Instead, the dynamics of environments from short to long time scales are critical to the evolution, or evolvability, of adaptability.

ENVIRONMENTAL CONDITIONS OF HUMAN EVOLUTION

Prior Hypotheses

In prior attempts at explaining human evolutionary history, habitat-specific scenarios have dominated. *Habitat-specific hypotheses* associate the origin of evolutionary novelties with the onset or spread of a particular habitat type—for example, replacement of forests by open savanna vegetation. The best-known example in paleoanthropology is the savanna hypothesis, which states that habitual bipedal locomotion evolved as proto-hominins moved from the trees to the ground during the spread of grassy savanna (or savanna-mosaic) vegetation. As open habitat continued to spread, a cascade of other changes were set in motion, including tool use and manufacture, a dietary shift, more complex sociality, and enlarged brains (Washburn, 1960; Wolpoff, 1980; Klein, 1989). This idea has recently been rephrased by several paleontologists and paleoclimatologists, who have emphasized the onset of cooler, drier conditions during the late Miocene and/or late Pliocene. The emergence of early humans, especially the genus *Homo*, is considered part of the evolution of the arid African biota (Vrba, 1988, 1995; Vrba et al., 1989; Prentice and Denton, 1988;

Stanley, 1992; deMenocal, 1995). Whether savanna aridity or glacial cold may have offered the driving force, habitat-specific accounts of human evolution are compatible with the idea that novel, complex adaptive behaviors require long-term directional selection, consistent over numerous generations, in response to a particular setting or environmental trend.

By contrast, *variability hypotheses* of human evolution focus on environmental dynamics and the inconsistencies in adaptive regime that an organism may face over time and space (Potts, 1998b). These hypotheses attempt to explain the origin of evolutionary novelties in terms of how they enhanced adaptive versatility. Some recent efforts have begun to test habitat-specific versus variability hypotheses by studying early human sites. In this section my aim is to provide examples from East Africa, where our research teams have been engaged in measuring environmental dynamics at various temporal and geographic scales in regions where early human populations lived.

Examples summarized here derive from two sedimentary basins, the Olorgesailie region (ca. 300 km²) in the rift valley of southern Kenya, and the Turkana region (ca. 136,000 km²) of northern Kenya and southern Ethiopia (Figure 2 on page 41). Together, these two basins encompass most of the time span of human evolutionary history. The Olorgesailie sequence covers the past 1.2 million years, whereas the Turkana stratigraphic record, at least the part examined here, covers the past 4.5 million years.

Methods

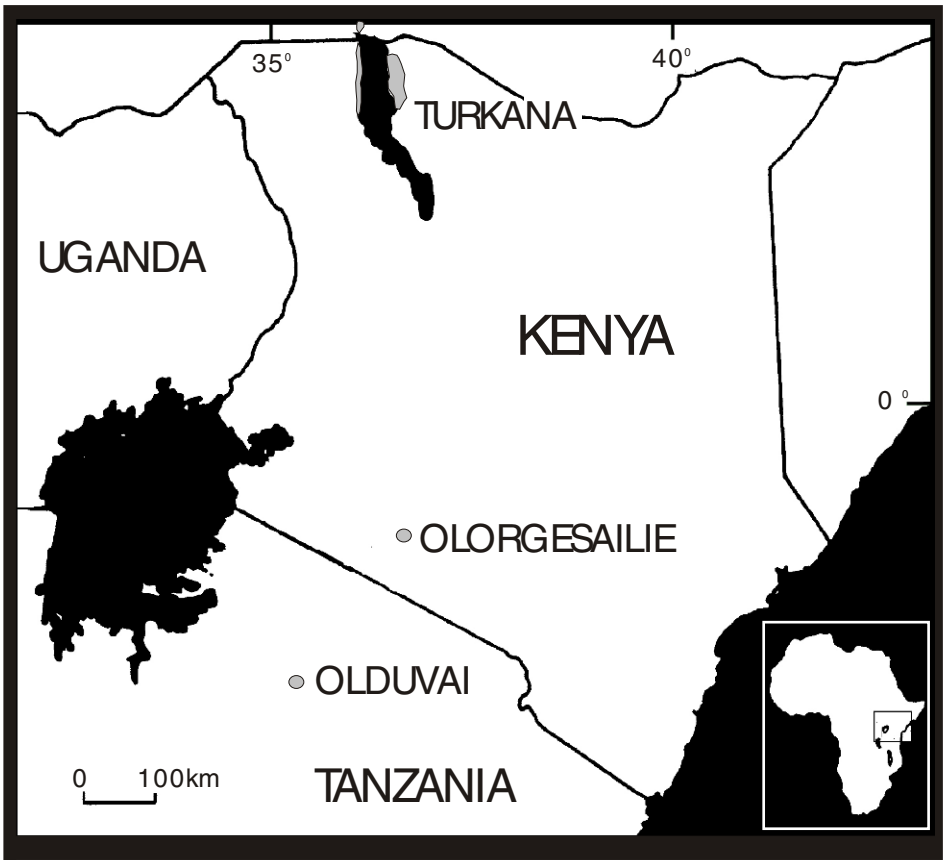
Recorded in the eroded terrain of the Olorgesailie region is a sequence of Pleistocene strata more than 80 m thick. Over the past sixty years, Olorgesailie has drawn considerable attention as one of the most prolific archeological sites of middle Pleistocene human activity, represented by thousands of Acheulean stone handaxes (Isaac, 1977). Besides abundant stone artifacts, the Olorgesailie basin preserves a sequence of faunal and paleobotanical remains, lake and fluvial sediments, paleosols, and volcanic tephras that provide precise chronological control (Deino and Potts, 1990). In parts of this sedimentary sequence, it is possible to discern fine (1 to 10 mm vertical scale) environmental fluctuations that probably represent seasonal or interannual flux of terrestrial silt into diatomite lake deposits. On a larger stratigraphic scale (20 cm to 10 m vertical scale), more dramatic environmental alterations can be observed. These include extensive drought that dried the lake, tectonically controlled episodes of basin-wide flooding, fluctuation between lacustrine/wetland and fluvial environments, and blanketing of the landscape by volcanic ash.

Using more than 250 detailed stratigraphic sections recorded throughout the basin, a thorough study of lateral (spatial) and verti-

cal (temporal) variation in environments and landscape structure has been undertaken, led by A. K. Behrensmeyer, R. Potts, and P. Ditchfield. Taking into account lateral facies variation, at least 26 basinwide revisions of the landscape took place over the past 1.2 million years. These landscape remodelings are called first-order changes.

A *first-order change* is defined as a major reorganization of the landscape and associated basin hydrology, substrate, habitable area, local climate, and habitat patchiness. Categories of first-order change include (1) large volcanic eruptions that rapidly deposited thick tephra (e.g., >1 m of ash or pumice), (2) transitions between lake- and alluvium-dominated environments, (3) transitions between aggradational and erosional periods, and (4) transitions between sediment aggradation and soil formation (landscape stability). First-order changes are approximately basinwide and may reflect regional or continental-scale environmental shifts.

Figure 2. A map of East Africa with the location of early human sites mentioned in the text. Study of landscape change and responses by early humans has focused on the Turkana and Olorgesailie basins, Kenya.



The period between two successive first-order changes is called a first-order environmental state. Within each of these periods is a series of smaller-scale but still significant shifts called second-order changes. A *second-order change* is defined as any large environmental shift that has occurred without altering the overall structure of basin hydrology and its resources. Such perturbations include, for example, unusual fluctuations in lake level, pedogenesis following flooding, or large shifts in fluvial hydrology. Such second-order changes are evident on a spatial scale of minimally several hundred meters of linear outcrop.

To illustrate how the analysis is organized, Figure 3 (on page 43) depicts a composite section of part of the Olorgesailie Formation. The stratigraphic boundaries of first- and second-order shifts are indicated by arrows. The type of first-order change and the duration of each first-order state are also indicated. Well-constrained radiometric ages assist in calculating the duration of each major environmental state. A composite section is used in Figure 3 to summarize the information, but the analysis requires lateral mapping of sediments and logging of stratigraphic sections throughout the basin in order to determine the nature and spatial extent of landscape change. Wholesale basin reorganization (first-order change) can thus be distinguished from second-order fluctuation, which in turn can be distinguished from smaller variations (e.g., seasonal flooding, channel avulsion, localized erosion) that typify all sedimentary systems.

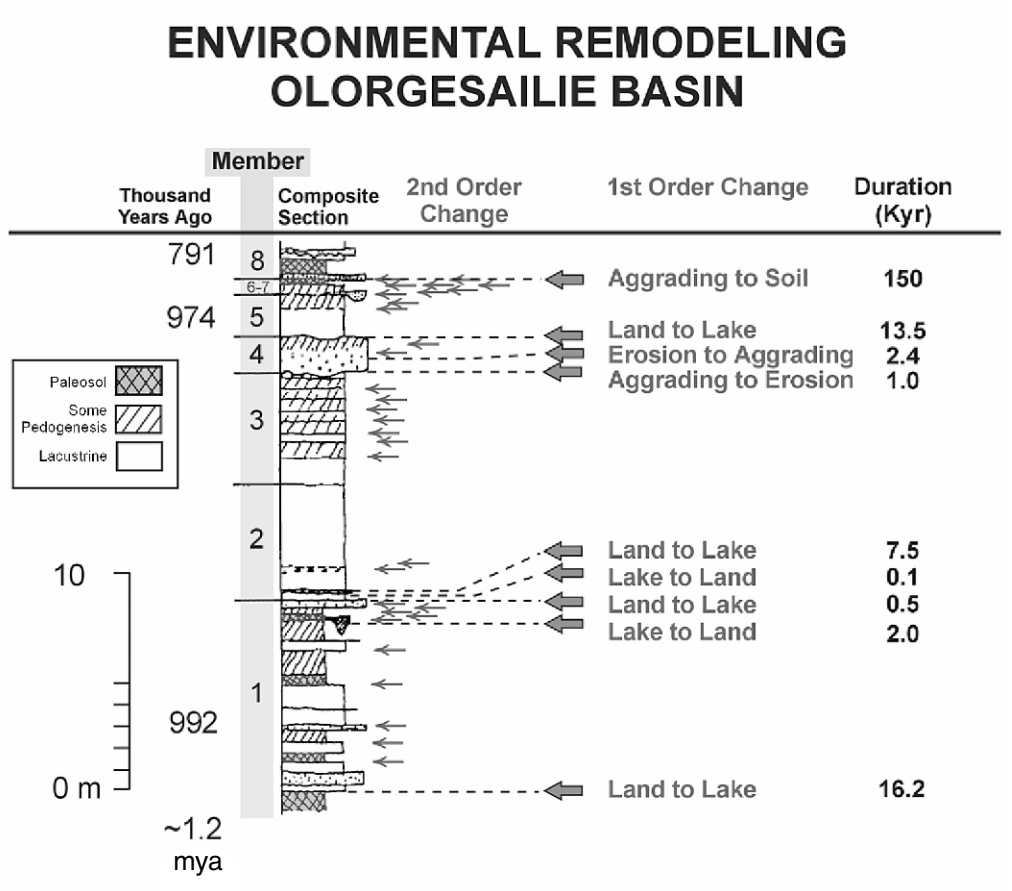
A similar type of data compilation and analysis has been initiated by Feibel for the Turkana basin. Turkana preserves arguably the best existing record of Plio-Pleistocene human fossils and archeological remains in a precisely dated context, particularly the interval between 4.1 and 1.5 million years ago (Brown and Feibel, 1991; Feibel et al., 1989). The Turkana sequence provides less precise information in the time interval best represented at Olorgesailie, the past 1.2 million years.

Reported here is a work-in-progress carried out by the author in collaboration with A. K. Behrensmeyer (Smithsonian Institution), C. Feibel (Rutgers University), and R. Chapman (Smithsonian). The analysis consists of plots of the distribution of first-order change through time and the representation of hominin evidence across major environmental boundaries. In addition, this analysis makes use of an unpublished climate model developed in collaboration with P. deMenocal (Lamont-Doherty Laboratory, Columbia University), based on a record of continental dust derived from ODP Site 659, reflecting West African eolian dust variation (deMenocal, 1995). The model compares dust flux data with predicted orbital eccentricity variations over the past 5 million years. As a result of this comparison, temporally constrained intervals of high and low climate variability have been identified that are applicable to the African conti-

nent. The dust data confirm the existence of these high- and low-variability intervals in the past.

In the analysis that follows, particular attention is paid to four intervals (two each of high and low climate variability) between 1.96 and 1.51 mya, which correspond to the richest part of the fossil record in the Turkana basin, and to two other intervals of high climate variability, from ca. 1.0 to 0.83 mya and from 0.73 to 0.46 mya, which correspond to the greatest abundance of archeological and faunal materials in the Olorgesailie basin. These analyses signify an initial test of how the record of early human fossils and archeological remains map onto major environmental boundaries and climate variability in Africa, where the longest record of human ancestry is known.

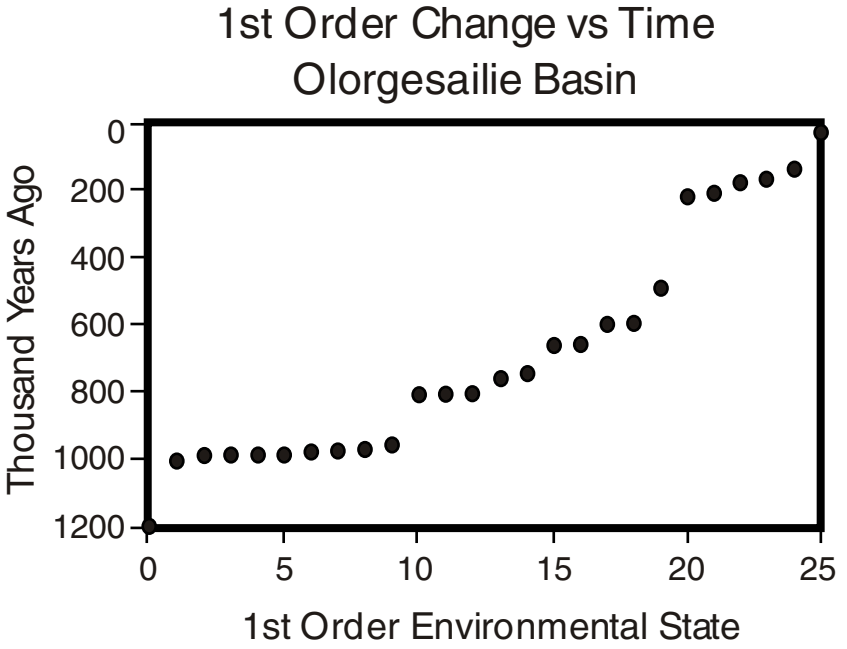
Figure 3. Composite section of Members 1 to 8, Olorgesailie Formation (ca. 1.2 to 0.79 mya), with arrows indicating the stratigraphic boundaries of first-order (basinwide) and second-order landscape changes. The type of first-order change and the estimated duration of each first-order environmental state are given (right side).



Results: The Tempo and Type of Environmental Change

In the Olorgesailie basin, first-order changes are unevenly distributed through time. Figure 4 indicates three intervals of concentrated landscape change, interrupted by periods when no major environmental reorganization took place. The three spurts of first-order change are recorded from 1.0 mya to 960 thousand years ago (kya); from 810 to 493 kya; and from 220 to 140 kya (Figure 4). Within each of these three time spans, the mean frequency of first-order change is once per 4.4 thousand years (kyr), once per 31.7 kyr; and once per 16.0 kyr, respectively. Periods of marked landscape stability include 1.2 to 1.0 mya, during which a volcanic lava landscape with sparse vegetation and soil prevailed, and 960 to 810 kya, represented by a basin-wide mature soil complex. An interval of fairly continuous and widespread erosion may have occurred between 493 and 220 kya, although this period is not yet as well documented as others in the Olorgesailie sequence. These time spans of first-order change and relative stability represent the tempo at which early humans and other inhabitants of the basin experienced large-scale remodeling of their landscape, water, and food resources.

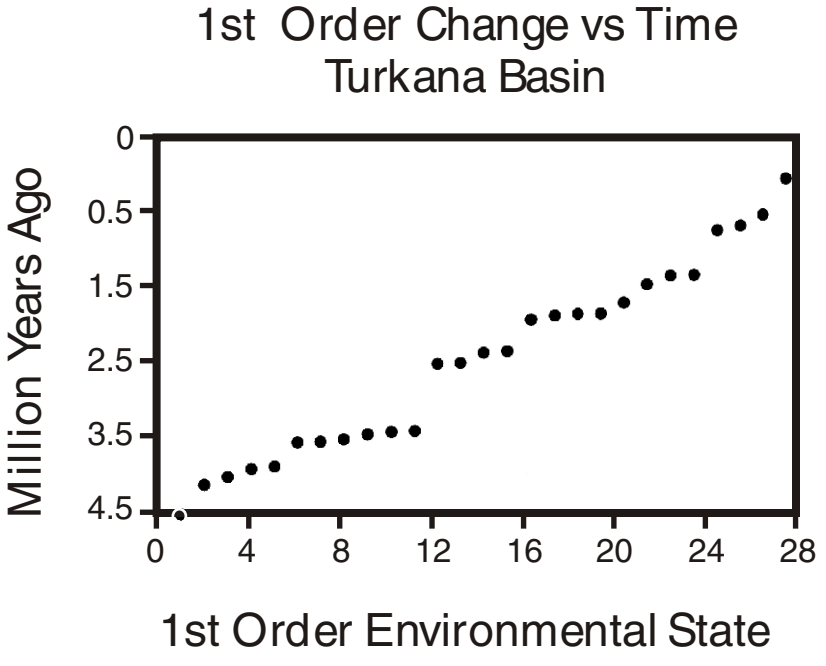
Figure 4. Distribution of first-order environmental changes (horizontal axis) through time (vertical axis) in the Olorgesailie basin, southern Kenya. The horizontal axis indicates the stratigraphic sequence, from 0 (oldest) to 25 (youngest), of first-order environmental states. The vertical distance between successive points denotes the duration of each environmental state.



Each first-order change established an environmental state (e.g., lake-dominated basin, fluvial system, stable landscape, erosional landscape) that persisted for a measurable length of time. The temporal duration of these major states, reflected in Figure 4 by the vertical spacing between data points, indicates no prevailing influence by orbital periodicities, such as precession (ca. 20-kyr) or eccentricity (ca. 100-kyr). First-order environmental states lasted for a wide range of different periods of time. A similar result is obtained when the duration of second-order environmental states is examined (Potts, 2001). The reason is that East African rift basins have been susceptible to the interaction between climatic and tectonic change; faulting and volcanism have had a large impact on the hydrological and climate conditions encountered by hominin populations living in this region (Behrensmeyer et al., in press; Feibel, 1999). Nonetheless, two time intervals of concentrated first-order change coincide with periods of high climatic variability predicted by deMenocal's eccentricity model and confirmed by the African dust record. The high-variability intervals are about 1.0 mya to 830 kya and 730 to 460 kya.

Similar results are evident in the environmental record of the Turkana basin. Shown in Figure 5, twenty-seven first-order changes can be seen over the 4.5 million years of the Turkana sequence. The

Figure 5. Distribution of first-order environmental changes (horizontal axis) through time (vertical axis) in the Turkana basin, northern Kenya. The horizontal axis indicates the stratigraphic sequence, from 1 (oldest) to 27 (youngest), of first-order environmental states. The vertical distance between successive points denotes the duration of each environmental state.



temporal distribution of these events is uneven; in this case, five intervals of concentrated environmental remodeling are evident, with periods of stasis in between. In some cases, the periods of stasis are remarkably long. It is important to note that these stable epochs were not erosional phases for which environmental data are missing. For example, the single environmental state recorded between about 3.4 to 2.5 mya is the Tulu Bor floodplain, which represents a nearly continuous sequence of river floodplain deposits.

The interval between 2.0 and 1.5 mya was one of especially complex environmental change in the Turkana basin. Although this period is indicated in Figure 5 as a single major spurt of first-order change, it was in fact characterized by irregular episodes of regional tectonics, including volcanism (Feibel, 1999). It also encompassed, according to deMenocal's model, two spans of high climatic variability and two of low variability.

For both the Turkana and Olorgesailie basins, the pertinent question is how early hominins and other organisms responded to first-order environmental events, times of landscape stability, and intervals of high and low climatic variability.

Results: Responses by Hominins and Other Animals

At Olorgesailie, important shifts in the faunal and archeological records coincided with the two oldest periods of marked landscape change and high climate variability. During the first interval, a permanent shift in early human behavior occurred. This is manifested as a change in the spatial patterning of stone artifacts across landscapes, from a continuous distribution (e.g., upper Member 1 of the Olorgesailie formation, ca. 990 kya) to a significantly more patchy or focused distribution beginning by around 900 kya (lower Member 7) (Potts et al., 1999). During this interval of landscape change, the toolmakers adopted a more focused spatial response to their habitat, which persisted as a strategy in later periods.

A second interval of significant environmental change, from about 800 to 500 kya, correlates with a key period of faunal turnover related to the formation of the modern African biota. During this time, several large mammal lineages became extinct that had dominated the East African fauna over the previous 1 million years. These lineages include the zebra *Equus oldowayensis*, the elephant *Elephas recki*, the baboon *Theropithecus oswaldi*, the hippo *Hippopotamus gorgops*, and the pig genus *Metridiochoerus*. In the southern Kenya rift valley, the last documented occurrences of these animals range from about 900 to 500 kya. By 400 kya they had disappeared from the fauna and were replaced by closely related living species—*Equus grevyi*, *Loxodonta africana*, *Papio anubis*, *Hippopotamus amphibius*, and the warthog *Phacocheorus aethiopicus* (Potts and Deino, 1995; Potts, 1996a). The extinct forms comprised specialized grazers and residents

of open savanna settings. Their replacements, by contrast, all tend to have diverse habitat preferences, diets, or social grouping behaviors (Potts, 1998a). It is tempting, therefore, to link the adaptability of these modern taxa, and the extinction of their relatives, to the heightened period of environmental variability. What can be said with confidence is that within the Olorgesailie basin, an elevated degree of first-order landscape change was the context in which the specialized grazers began their decline and the modern taxa succeeded them.

In the Turkana basin, a temporally and geographically diverse pattern of faunal turnover occurred between 2.8 and 1.8 mya. In the northern Turkana basin (Omo Shungura Formation), a relatively stable fluvial floodplain characterized this interval, and gradual turnover in fossil bovid lineages took place, particularly between 2.8 and 2.1 mya, coinciding with a global cooling and drying trend (Bobe and Eck, 2001). This period also encompasses two long intervals of high climatic variability in deMenocal's model—2.77 to 2.48 mya and 2.33 to 2.09 mya. In the eastern Turkana basin, faunal turnover was apparently gradual between 2.5 and 2 mya, whereas the following period from 2.0 to 1.8 mya entailed significantly higher mammalian turnover, mainly extinctions (Behrensmeyer et al., 1997). This latter episode correlates with deMenocal's high-variability interval between 1.96 and 1.71 mya and with the series of first-order environmental changes that began in Turkana around 1.95 mya (Figure 5).

Responses by hominins to environmental change are recorded in the stratigraphic distributions of fossils and stone tools. The eastern part of the Turkana region, known as Koobi Fora, has a superb record of hominin fossils, the majority of which occur within the time interval from 2.0 to 1.5 mya (Wood, 1991). A reasonably large sample (N=173) of these fossils can be assigned to the four intervals of high or low African climate variability, and almost all of these fossils can be classified to genus, either *Homo* (*sensu lato*) or *Paranthropus*. (The assignment of fossils to specific time intervals was done with the assistance of C. Feibel of Rutgers University.) *P. boisei* is the only East African lineage during this span assigned to the robust-toothed genus *Paranthropus*. The genus *Homo*, on the other hand, is represented by at least two species, *H. rudolfensis* and *H. ergaster*.

Table 2 (on page 48) shows the results of this analysis. While *P. boisei* is well represented in the Turkana basin back to about 2.3 mya, the first definite appearance of *Homo* in the Koobi Fora sequence occurs during an interval of marked climate variability—1.96 to 1.71 mya—the same period characterized by high turnover of mammalian lineages. Most striking is that *Homo* is represented not by a few specimens but by a distinct predominance of this genus (75%) over *Paranthropus* (23%). During the subsequent period of low climate variability, *Paranthropus* rises in abundance, comprising 57% of the

hominin sample. *Homo* dominates again, however, during the high-variability span that follows, between 1.65 and 1.55 mya, although the fossil sample is relatively small (N = 15). The next low-variability interval, 1.54 to 1.51 mya, is near the last known appearance of *Paranthropus* in the Turkana record; nonetheless, this genus rises slightly in abundance. *Homo* thus decreases but is still dominant.

TABLE 2: RELATIONSHIP BETWEEN EARLY HOMININ GENERA AND CLIMATIC VARIABILITY IN THE KOOBI FORA REGION OF THE TURKANA BASIN, NORTHERN KENYA.

CLIMATE VARIABILITY	N	SPAN (*mya*)	%*HOMO*	%*PARANTHROPUS*
LOW VARIABILITY	28	1.54–1.51	61%	36%
HIGH VARIABILITY	15	1.65–1.55	67%	33%
LOW VARIABILITY	30	1.70–1.66	43%	57%
HIGH VARIABILITY	100	1.96–1.71	75%	23%

In short, fossils of the genus *Homo* are more abundant in intervals of high climate variability, while fossils representing *Paranthropus* increase during low-variability periods, even toward the end of that genus's known existence. At face value, the results of this exercise suggest, but by no means prove, that *Homo* was able to thrive during relatively unstable environmental conditions, with the kind of inconsistency in adaptive conditions in which adaptability is at a premium, whereas *Paranthropus* was apparently favored when climate was more stable.

Turning to the archeological record, stone tools are known in the Turkana basin back to about 2.3 mya. Between 2.3 and 1.9 mya, stone tools are distributed sporadically through the stratigraphic sequence. Toolmakers manifested their presence in relatively brief intervals, and in no instance observed so far do the stone tools occur across (both immediately above and below) the stratigraphic boundary of a first-order environmental change. In other words, stone tools disappear from the Turkana record at first-order boundaries and reappear only some time later. Beginning around 1.7 mya (e.g., Okote Member in East Turkana), artifact sites occur much more abundantly and are recorded nearly continuously through second-order events, a series of small-scale volcanic eruptions. There is still no clear evidence, however, that the toolmakers were able to persist across a first-order change. This aspect of the Turkana basin record is paralleled by archeological finds at Olduvai Gorge, Tanzania. In Bed I and lower Bed II at Olduvai (ca. 1.85 to 1.7 mya), stone tool sites are known in almost every stratigraphic layer (Leakey, 1971; Hay, 1976). This long-term persistence of the toolmakers occurs, however, through a sequence of second-order shifts and intervening periods of stability, not across first-order boundaries.

By contrast, the later archeological record of the Olorgesailie basin is typified by persistence across episodes of major environmen-

tal remodeling. On the basis of investigations to date, stone tools are known to occur in strata representing 16 of the 26 first-order environmental states shown in Figure 4. In at least 10 of these instances, artifacts occur in the sedimentary layer that immediately precedes the stratigraphic boundary of the first-order event. In 12 of those same instances, artifacts are recorded in the stratigraphic layer immediately above the major transition. According to these observations, hominin toolmakers appear to have possessed the means necessary to persist across first-order events, or to recolonize immediately after.

These results sharply contrast with the evidence of older toolmaking populations at Turkana and Olduvai. This suggests that mid- and late Pleistocene toolmakers of Olorgesailie were better able to buffer or accommodate to environmental variability than their earlier counterparts in East Africa. Although there is much left to do to complete the analysis, this type of study may help considerably in assessing the adaptability of hominins to diverse types of environmental change.

Analytical Summary

The results can be summarized as follows:

> In the regions and specific locales where early human populations lived, at least in East Africa, landscapes and resources were susceptible to episodic revision. Genetic lineages of East African hominins encountered dramatic revamping of landscapes at frequencies typically ranging from once per 4,000 years to about once per 70,000 years.
>
> Time intervals characterized by large environmental change were interspersed with periods of stasis. Instances of essentially stable landscape conditions sometimes persisted for periods considerably greater than 100,000 years.
>
> During the late Pliocene of East Africa, the oldest known stone toolmakers appear to have been present sporadically in any one region. Starting about 1.85 to 1.7 mya, the presence of hominin toolmakers is registered more or less continuously in diverse settings, but not across first-order environmental shifts. During the past 1 million years, however, toolmakers (at least those in the southern Kenya rift) had apparently evolved the means to persist across major environmental boundaries.
>
> Different lineages of early hominins responded to environmental variability and stability in different ways. This finding is suggested by the representation of early *Homo* and

Paranthropus in the fossil record of the Turkana basin between 1.96 and 1.51 mya. The results suggest that at least one lineage of *Homo* had furthered its behavioral and ecological means of adaptability beyond those available to prior and contemporaneous hominin species.

DISCUSSION

How Does Adaptability Evolve?

The question arises as to how adaptable characteristics took shape in certain lineages of hominins. In general, how does adaptability evolve in organisms?

One possible solution has been proposed: the variability selection hypothesis (Potts, 1996a, 1996b). The principle of variability selection is as follows: When a lineage of organisms encounters inconsistent conditions of survival and reproductive success, genetic variations that bestow adaptive versatility may be retained. If this lineage faces inconsistency over a prolonged time, a genetic basis may be assembled underlying complex adaptations that promote versatility. These adaptations enable novel responses to the surroundings and augment the options available to the organism. In the term "variability selection," the word *selection* does not merely refer to reproduction and survival at the individual level. It mainly refers to the pattern of natural selection, as also conveyed by such terms as "directional selection," "kin selection," and "sexual selection." In variability selection, it is the pattern of selection over time and space that is important—in particular, a disparity in selective results faced by a single gene pool over time. The variability selection hypothesis is that such disparities, arising out of the spectrum of environmental dynamics, may cause alleles that improve versatility to eventually win out over alternatives that do better only in certain habitats.

The underlying assumption is that adaptive versatility is in fact evolvable. If this assumption is correct, it follows that certain genetic variants (and the developmental programs they engender) may prove better than others at surviving fluctuation in selective conditions. Alternative genetic variants and phenotypic features that assist an organism in only one specific setting are weeded out as the lineage confronts a dynamic sequence of different selective environments.

Natural selection has largely been understood as a process by which organisms adapt to habitats—to the stable features and statistical regularities that make different habitats recognizable. Generation by generation, the consistent aspects of a selective environment lead an organism to become adapted to its surroundings. The variability selection idea places emphasis instead on the dynamic qualities of environments. These variable qualities are ones to which an individ-

ual organism and its genetic lineage must accommodate in order to persist. This hypothesis posits that selection (and the resulting biological organization of an organism) is governed by the temporal spectrum of environmental dynamics, in some cases highly variable sequences. Under these circumstances, it is possible to comprehend how structures and behaviors may evolve that are designed to accommodate to dynamic and even novel settings. If organisms evolved solely in relation to past environmental settings *per se*, it is difficult to see how an organism could be prepared to accommodate to novel surroundings or to make innovative responses to new situations. In variability selection, by contrast, the adaptive qualities of organisms have evolved in relation to past environmental dynamics, which imbues a genetic lineage with the potential for adaptability.

The Complexity Response

If the emphasis accorded here to dynamic environmental properties is correct, it implies that buffering mechanisms should characterize biological systems. Buffering mechanisms should be apparent over a wide time spectrum and at various biological scales of organization—from metabolic buffering inside individual cells (on very short time scales) to ecological buffering by lineages of organisms in response to long-term instability. These means of adjusting to environmental dynamics are likely to represent important aspects of evolutionary biocomplexity (Table 3).

- At the genomal level, unstable selective conditions may yield an increase in genetic polymorphism; complexity arises from building a larger storehouse of alternative genetic variations.
- At the developmental level, inconsistency in environment and selection may lead to a greater degree of phenotypic plasticity; complexity arises, in this case from genotype-environment interactions that expand the developmental reaction norm.
- At the behavioral and ecological level, environmental instability may enlarge a lineage's adaptive versatility; complexity results from decoupling the organism from any single habitat and a freer mapping of its behavior onto environment.

TABLE 3: THE COMPLEXITY RESPONSE: INCREASES IN BIOLOGICAL COMPLEXITY AT THREE LEVELS OF BIOLOGICAL ORGANIZATION IN RESPONSE TO INCREASING ENVIRONMENTAL COMPLEXITY.

GENOMAL:	↑ GENETIC POLYMORPHISM
DEVELOPMENTAL:	↑ PHENOTYPE PLASTICITY
BEHAVIORAL/ECOLOGICAL:	↑ ADAPTIVE VERSATILITY

In the behavioral and ecological domain, organisms evolve ways of tracking favored foods or climate conditions and/or sophisticated behavioral mechanisms that can engender versatile responses to new environments. The latter may entail decoupling of the organism from any specific ancestral habitat, enabling it to diversify its behavioral and ecological options. Humans are perhaps the most extreme example, but there are others, as suggested by the case examined earlier, in which more versatile mammals replaced more specialized lineages in mid-Pleistocene East Africa. Levins (1968) refers to this decoupling process as one of "binding and unbinding" genetic, developmental, and phenotypic variables. Greater degrees of freedom among these variables may enhance the possibility of major restructuring of the organism in relation to its environment. These phenomena lie at the heart of human adaptive complexity; thus, the role of changing environments is central to the evolution of both adaptability and complexity.

Still, the question remains as to how adaptability has evolved in the face of the immense inconsistency of adaptive conditions encountered by Pleistocene lineages. The variability selection idea—which has been developed conceptually but not mathematically as yet—draws attention to the possibility that some genetic novelties prove favorable under shifting conditions. Yet the genetics of adaptability remains largely unknown, as do the ways in which developmental pathways mediate adaptability. Orr (2000) has suggested that increasingly complex organisms pay a larger cost for genetic adaptation. This implies that under certain circumstances, complex organisms may benefit by evolving systems of nongenetic adaptation—that is, systems of information processing and behavioral plasticity that enhance versatility without incurring the ongoing costs of genetic adaptation. It seems likely that the cost-benefit compromise between genetic and nongenetic adaptation has been shaped by environmental complexity and that this compromise has played a crucial role in human evolution.

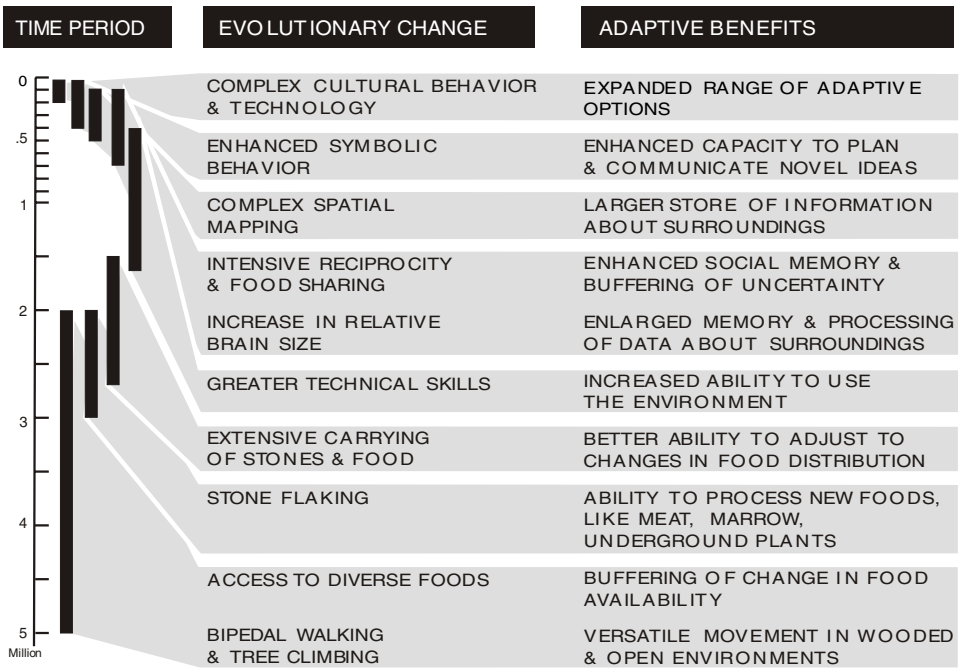
Human Evolutionary History

One consequence of evolving complex systems of adaptability is the persistence of lineages and phenotypes across large changes in environment. Over the course of human evolutionary history, certain behaviors resulted in greater adaptability and responsiveness to novel environmental settings. Previously, I have suggested that human evolution, stretching back through earlier hominin lineages within a diverse phylogenetic history, was characterized by a ratcheting up of adaptive versatility (Potts, 1998a). An evolutionary relay took place over time that involved (from earlier to later) greater mobility, expanded diet, enhanced cognition, and the potential for extreme

social complexity. A simplistic version of this trajectory is shown in Figure 6, along with the possible enhancements to adaptive versatility in the lineage resulting in *Homo sapiens*.

The earliest manifestation of habitual bipedal behavior was coupled with climbing ability and possibly dietary expansion, which offered the means of accommodating to vegetation change and unstable food sources. Later, stone flaking, extensive carrying of objects, and small enhancements in stone flaking over time expanded the means of buffering environmental uncertainty. Beginning about 700,000 to 400,000 years ago, a variety of other response systems became evident, indicated by more rapid encephalization; archeological evidence of hearths and shelters suggestive of more elaborate sociality, food sharing, and home base behavior; and evidence of longer distances of stone transport suggestive of more intricate spatial mapping. Over the past 100,000 years or so, the archeological record documents the enhanced use of symbols to refer to circumstances beyond those immediately apparent and the diversification of behavioral options—characteristics of cultures in the modern human sense. These latter changes denote a radical intensifying of social interactions.

Figure 6. Ten major developments in human evolution (Evolutionary Change), the main time period when these developments took place (read from bottom to top), and a brief summary of the potential adaptive benefits in the context of environmental instability.



The presentation of human evolutionary history in its appropriate environmental context (which becomes increasingly complex as it becomes better investigated) alters one's interpretation of these evolved features. This sequence can be considered an evolutionary history of rising adaptability in relation to environmental complexity.

CONCLUSION

Regarding the evolution of adaptability, we may conclude that some evolved characteristics in some organisms are geared toward solving problems of inconsistency in the adaptive milieu. These adaptations reflect the dynamic properties of settings in which those organisms' ancestors evolved.

An exceptional degree of environmental complexity has characterized the past several million years. Over the period of human evolutionary history, environmental complexity has included a graded spectrum of climatic variability, with greater variability over longer time periods; periodic climate fluctuation interspersed with periods of relative stability; interaction among climate cycles of different oscillatory periods, which has led to threshold-type environmental change; and nonlinear change due to interactions between climate variability and tectonic events.

Organisms would appear to mirror this environmental complexity at the genomal, developmental, behavioral, and ecological levels of biological organization—what I refer to as "the complexity response."

Understanding human evolutionary history may require far more sophisticated models of the adaptive process that focus on dynamics—the variability of environments over evolutionary time—rather than on the consistent properties of habitats and of the problems organisms are said to solve.

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CHAPTER 4

Culture is Part of Human Biology: Why the Superorganic Concept Serves the Human Sciences Badly

PETER J. RICHERSON AND ROBERT BOYD

Rates of violence in the American South have long been much greater than in the North. Accounts of duels, feuds, bushwhackings, and lynchings occur prominently in visitors' accounts, newspaper articles, and autobiographies from the eighteenth century onward. According to crime statistics, these differences persist today. In their book *Culture of Honor*, Richard Nisbett and Dov Cohen (1996) argue that the South is more violent than the North because Southerners have different culturally acquired beliefs about personal honor than Northerners. The South was disproportionately settled by Protestant Scotch-Irish people with an animal herding background, whereas northern settlers were English, German, and Dutch peasant farmers. Most herders live in thinly settled, lawless regions. Since livestock are easy to steal, herders (generally male) seek reputations for willingness to engage in violent behavior as a deterrent to rustling and other predatory behavior. Of course, bad men come to subscribe to the same code, the better to intimidate their victims. As this "arms race" proceeds, arguments over trivial acts can rapidly escalate if a man thinks his honor is at stake, and the resulting culture of honor leads to high rates of violence. Nisbett and Cohen support their hypothesis with an impressive range of data, including findings from laboratory and field experiments, attitude surveys, data on violence, and information on differences in legal codes.

Their laboratory experiments are most relevant to our argument here. Cohen and Nisbett recruited subjects with northern and southern backgrounds from the University of Michigan student body, ostensibly to work on a psychological task dealing with perception. During the experiment, a confederate bumped some subjects and

muttered "asshole" at them. Cortisol (a stress hormone) and testosterone (which rises in preparation for violence) were measured before and after the insult. Insulted Southerners showed big jumps in both cortisol and testosterone as compared with uninsulted Southerners and insulted Northerners. The differences in psychological and physiological responses to insults were manifested in behavior. Nisbett and Cohen recruited a 6'3" tall, 250-pound football player whose task was to walk down the middle of a narrow hall as subjects walked in the other direction. The experimenters measured how close subjects came to the football player before stepping aside. Northerners stepped aside at around 6 feet, regardless of whether or not they had been insulted. Uninsulted Southerners stepped aside at an average distance of 9 feet, whereas insulted Southerners approached to an average of about 3 feet. Polite but prepared to be violent, uninsulted Southerners take more care, presumably because they attribute a sense of honor to the football player and are normally respectful of others' honor. When their honor is challenged, they are prepared and willing to challenge the offender at considerable risk to their own safety.

Nisbett and Cohen's study illustrates the two main points we want to make in this essay:

> *Culture is fundamental to understanding human behavior*. The high rates of violence in the American South are a product of a social heritage. The southern culture of honor arose in, and was for a long time maintained by, an environment that made it an efficacious means of protecting a family's livelihood. Nowadays, few Southerners are pastoralists, and few Northerners are peasant farmers. Nonetheless, these striking differences in behavior persist.
>
> *Culture causes behavior by causing changes in our biology*. An insult that has trivial effects in a Northerner sets off a cascade of physiological changes in a Southerner that prepare him to do violent harm to the insulter and to cope with the likelihood that the insulter is prepared to do equal harm in return. We argue that this example is merely a single strand in a mass of connections that so thoroughly web culture into other aspects of human biology that any separation of them into distinct phenomena is impossible.

We can certainly make an analytical distinction between genetic and cultural influences on our behavior, and noncultural forms of environmental influences. However useful, this analytical distinction emphatically does not license an ontological separation of culture and

biology into discrete levels of organization, with only simple biological "constraints" on cultural evolution and diversity. Culture is as much part of human biology as bipedal locomotion, and cultural and genetic influences on human behavior are thoroughly intertwined.

Most of the important threads of twentieth-century social science have rejected one of these two principles. Some traditions within the social sciences—for example, those of rational choice theorists, many psychologists, and human sociobiologists—place little emphasis on culture as a cause of human behavior and sometimes view cultural explanations as limited to historical-descriptive accounts devoid of real explanatory power. While we sympathize with critics of current culture studies, this state of affairs is not inherent in the culture concept. The effects of culture on human behavior can readily be addressed with the methods of the so-called hard sciences (see Cavalli-Sforza et al., 1973, 1981; Lumsden and Wilson, 1981; Boyd and Richerson, 1985; Richerson and Boyd, 1989). We want to convince you that a Darwinian science of culture is a respectable and promising pursuit and that the easiest way to see why is to place culture squarely in the middle of human biology.

Many social scientists have objected to moves of this ilk for fear that the result would be to "reduce" culture to biology. Many biologists interested in humans have encouraged such fears. Edward O. Wilson (1975, 1998) argues that disciplines stand in a reductionistic relation to one another and that the ultimate fate of the social sciences is to be reduced to sociobiology. The project we champion differs significantly from Wilson's. Part of the payoff for locating culture in biology is that we can model the influence that culture has on genes, as well as the "reductionistic" influence of genes on culture. If we imagine that genes and culture are two inheritance systems that interact on the same level to produce human behavior, we can make coevolutionary, or dual inheritance, models of the basic processes by which this interaction takes place. These models have the virtue of reducing to more conventional positions such as rational choice theory, various kinds of human sociobiology, and, most interestingly, Marshall Sahlins's "cultural reason" (1976), under different simplifying assumptions (Boyd and Richerson, 1985, ch. 8). Under a broad and reasonable range of assumptions, evolving genes, evolving culture, and environmental contingencies all conspire to affect human behavior.

For some students of culture, locating culture in biology may still seem a risky strategy. The powerful theories and intimidating empirical methods of the natural sciences might overwhelm culture, as if science is somehow inherently biased against cultural explanations. We believe the opposite. Cultural explanations of human behavior are likely to prove exceedingly robust. The processes of cultural evo-

lution may substantially socially construct human nature itself, not just our ideas about it. Culture, in this hypothesis, has the fundamental role in human behavior long claimed for it by cultural anthropologists and many other social scientists and humanists. Cultural evolution can create social institutions that in the long run shape important aspects of even the innate components of human biology. Innatists run a real risk that some of their genes will be "reduced" to culture!

THE POVERTY OF SUPERORGANICISM

Most social scientists treat culture as a superorganic phenomenon. As A. L. Kroeber (1948, p. 62) put it in trying to explicate the superorganic concept, "particular manifestations of culture find their primary significance in other cultural manifestations, and can be most fully understood in terms of these manifestations; whereas they cannot be specifically explained from the generic organic endowment of the human personality, even though cultural phenomena must always conform to the frame of this endowment." To quote Theodosius Dobzhansky (1962, p. 20), an evolutionary biologist very sympathetic to the Twentieth-Century social sciences of culture, "In producing the genetic basis of culture, biological evolution has transcended itself—it has produced the superorganic." Social scientists have long used rhetoric like this to dismiss the need to incorporate biology in any serious way into their study of human behavior. Humans cannot fly by flapping their arms or swim naked in polar seas, but outside of obvious framing constraints of this type, things biological have had no explanatory role in explaining things cultural. In this view, biology is important, of course, because we need bodies and brains to have culture—but biology just furnishes the blank slate on which culture and personal experience write. This idea goes back to the turn-of-the-Twentieth-Century pioneers of sociology and anthropology. For example, French sociologist Gabriel Tarde's book *The Laws of Imitation* (1903) prefigured in many ways the ideas in this essay, but he rejected any considerations of biology as a practical matter of disciplinary specialization. Dobzhansky's usage was probably inspired by Kroeber and kindred influential social scientists of his period. Dobzhansky was recognizing a *fait accompli*, we believe. If biologists of his day wanted harmonious relations with social scientists rather than destructive nature-nurture disputes, they had to make obeisance to the superorganic concept—though Dobzhansky went right on to say, "Yet the superorganic has not annulled the organic." He never satisfactorily resolved the tension between his two statements on superorganicism. Ingold (1986, p. 223 ff) provides a discussion of three different senses of the term *superorganic* as used by social scientists over the years, concluding that "the superorganic has become a banner of convenience under which have paraded anthropological

and sociological philosophies of the most diverse kinds."

In our view, superorganicism is wrong because it cannot deal with the rich interconnections between culture and other aspects of our phenotype, as exemplified by the Southern culture of honor. Superorganicism may have served a useful function in helping the social sciences get on their feet. Today we believe it is best to grasp the nettle: *Culture is a part of human biology*—as much a part as bipedal locomotion or thick enamel on our molars. Because of culture, people can do many weird and wonderful things. But in all cases, the equipment in human brains, our hormone-producing glands, our hands, and the rest of our bodies play a fundamental role in how we learn our cultures and why we prefer some ideas to others. This is a minority, even heretical, position among human scientists, albeit one with a long pedigree. Freud was a defender of it (Sulloway, 1979), as are many modern psychologists, some of whom we discuss later.

Suppose we define culture as follows: *Culture is information capable of affecting individuals' phenotypes, which they acquire from other conspecifics by teaching or imitation*. In the taxonomy of definitions of culture, ours is in a category that emphasizes the psychological aspects of the phenomenon (Kroeber and Kluckhohn, 1952). Culture is taught by motivated human teachers, acquired by motivated learners, and stored and manipulated in human brains. Culture is an evolving product of populations of human brains. Humans are adapted to learn and manage culture by the way natural selection has arranged our brains. Human social learners in turn arrange features of their brains as they learn from others and the environment. Culture is a major aspect of what the human brain does, just as smelling and breathing are what noses do. Culture-making brains are the product of more than two million years of more or less gradual increases in brain size and cultural complexity. During this evolution, culture must have increased genetic fitness, or the psychological capacities for it would not have evolved. Indeed, anthropologists long interpreted much of culture in adaptive terms (e.g., Steward, 1955). Rather than a neat, narrow boundary between innate and cultural processes that can be characterize by a short list of simple biological constraints on human behavior, we imagine a wide, historically contingent, densely intertwined set of phenomena with causal arrows operating in both directions. If we think of human culture as a part of human biology in this way, we simply do not need to try to unpack what "superorganic" could possibly mean.

We are a bit sensitive on this point because the style of analysis of the cultural phenomenon we advocate has collected its share of brickbats from both sides of the superorganic divide. From the evolutionary biology side, Richard Alexander (1979, pp. 79–81) and others have supposed that the analysis of culture as an inheritance system is

an attempt to defend the superorganic concept against evolutionary analyses of human behavior. On the other hand, some social scientists have treated our work as yet another attempt to "reduce" culture to biology (e.g., Ingold, 1986, ch. 7). In our view, culture and the rest of human biology interacted in complex ways in the evolutionary past to produce an extraordinary ability to imitate. Genes and culture continue to interact in the everyday world of human behavior in most complex ways. Functional magnetic resonance imaging and the other brain-scanning techniques are even beginning to give us a real-time picture of how these interactions take place in the brain. In some ways these processes resemble the claims of the conventional social sciences, and in some ways they resemble the proposals of human sociobiologists and nativist psychologists. Very often, the processes do not resemble the proposals of either. There are some fascinating scientific puzzles to solve here.

CULTURE IS A DERIVED HUMAN TRAIT

We as yet know precious little about exactly how genes, culture, and external environment play upon the brain to produce our behavior. We do know that without a human brain, you cannot acquire human culture. Recent comparative primatology is beginning to describe the nature of our capacity for imitation relative to other apes in some detail. Groups led by Andrew Whiten and Michael Tomasello have studied the social learning of apes and human children in a comparative framework (Whiten and Custance, 1996; Tomasello, 1996). For example, Tomasello's group used human demonstrators of a raking technique to test the social learning of juvenile and adult chimpanzees and 2-year-old children. The demonstrators used two different techniques of raking to obtain otherwise unreachable desirable objects. Control groups saw no demonstrator. In both children and chimpanzees, comparisons of experimental and control groups showed that the demonstrator had a big effect on the use of the rake. But the interspecific difference was also large: the children tended to imitate the exact technique used by the demonstrator, whereas the chimpanzees did not. In similar experiments with older children, Whiten and Custance reported a rapid increase in the fidelity of imitation by children over the age range of 2 to 4 years, with adult chimpanzees generally not quite achieving the fidelity of 2-year-old humans. At quite young ages, human children are already far more imitative than any other animal so far tested, although a very few other animals, such as parrots, are also about as good as chimpanzees at imitative tasks (Pepperberg, 1999).

What is the biological underpinning of our hypertrophied social learning system? Tomasello (1999) gives an account based on a considerable body of observational and experimental evidence. He

argues that the most important unique feature of human cognition is what he calls "joint attention." Human children, beginning at about nine months of age, begin to pay attention to the attention of other people and to call the attention of others to things of interest to themselves. For example, in Western cultures, children interact with their caregivers in little word games in which both the child and the adult pay attention to the same object—typically a toy. The child may hand the toy to the adult and then look to the adult for some reaction, or vice versa. The adult often articulates the word for the "toy"—*ball, dolly, truck*. In this way children learn their first words and use the joint attention situation to try out their new words. Or the adult operates the toy—throws the ball, dresses the doll, runs the truck on its wheels making motor noises—and the child learns the demonstrated skills. Tomasello dissects joint attention into nine separate skills emerging between nine and twelve months of age. The early maturation of these skills and the apparent necessity of having them before substantial imitation can occur argue for a large element of innate specification of the joint attention system. All of these skills are specific to normal humans and are sufficient to account for the differences in imitative capacities of children and chimpanzees. Autistic children seem to have specific deficits in joint attention and are greatly handicapped in learning language and acquiring other culturally transmitted skills. At the end of the normal developmental sequence, children understand that other people are intentional agents with motivations like their own. Thus, the actions of other are cues as to how one can take advantage of the experiences and skills of others to accomplish one's own goals. From the age of one year onward, children are efficient imitators and begin to build their cultural repertoires rapidly. According to Tomasello's hypothesis, the same joint attention skills underpin the learning of all aspects of culture, from language to subsistence skills. Many evolutionary psychologists prefer modular hypotheses, imagining many separate mental "organs," most famously for language learning (Pinker, 1994). The evidence on these problems is far from conclusive. The very existence of a seemingly rather unusual and highly organized capacity (or capacities) for imitation does argue that an understanding of it (or them) is part of evolutionary psychology correctly considered.

EVOLVED HUMAN NATURE VERSUS GENE-CULTURE COEVOLUTION

Most evolutionary theories of human behavior inspired by Darwin underestimate the importance of culture in the evolution of human behavior, much as the theories of superorganicists underestimate the role of genes. Typically, biological theorists assume first that natural selection built human biology and then that this evolved biology

controls human behavior. In such theories, the ultimate determinants of human behavior are the products of selection on genes. Any role for culture is proximate and can be thought of as implementing structures that are built into the genes. The distinction between proximate and ultimate causation is Ernst Mayr's (1961) borrowing from Aristotle. Mayr argues that in biology, proximate causes are typically physiological. Birds migrate equatorward when day lengths shorten because their brains convert short day lengths into hormonal signals that activate migratory behavior. The ultimate cause of migratory behavior is natural selection. Migration is an evolved strategy to exploit the favorable season at higher latitudes while passing the harsh winter in undemanding habitats. Selection has shaped the reaction of the brain to day length as well as all the downstream physiological and behavioral machinery that works to accomplish the migratory adaptation. Much of the dispute over the role of culture in human behavior is understandable in terms of the proximate/ultimate distinction.

Most Human Sociobiology Unduly Neglects Culture

Most students of human behavior inspired by evolutionary biology, prefer to keep things simple by neglecting or denying the possibility that culture has a fundamental role to play in human adaptation, and especially that it has any component of ultimate causality. The classic 1974 paper by Richard Alexander and the final chapter on humans in Edward O. Wilson's landmark treatise *Sociobiology* (1975) caused considerable interest in applying evolutionary ideas to human behavior. Two traditions that grew up in the wake of Alexander's and Wilson's work are human behavioral ecology and evolutionary psychology. The bedrock of the evolutionary analysis conducted by scholars in these traditions is the concept of natural selection acting on genes. They argue that selection, over the course of human evolution, would have favored capacities to make decisions—including decisions about what cultural behaviors to adopt—that increased genetic fitness. How could our large, complex, physiologically expensive brain have evolved to support human capacities for learning, including the learning of culture, unless the resulting behaviors increased fitness? Natural selection is the only process of design operating in the world, and the complex capacities of the human brain must therefore have arisen by its operation.

We call this argument the "principle of natural origins." In our view, it is an exceedingly important idea. It has been attacked vigorously by critics from Darwin's time forward but has proved quite robust (Dawkins, 1985). Most Darwinians no longer think detailed defense of it is necessary and just use natural origins as a metatheoretical precept to use to discover adaptations. That is, Darwinians frequently use the principle of natural origins to formulate hypotheses

about what would be adaptive if it is true, rather than test the dominant role of selection as a hypothesis. This usage has famous critics among evolutionists, not to mention antievolutionists (Gould and Lewontin, 1979), but we are not among their number. The alternative metatheory of the evolutionist critics has not enjoyed much success (e.g., Carroll, 1997) compared, say, with the universal Darwinism of Campbell (1965), Dawkins (1976), Dennett (1995), Cziko (1995), and Sober and Wilson (1998). Universal Darwinists see selection as producing adaptations on diverse heritable substrates, including culture, and at diverse levels ranging from individual genes and memes to groups. Some of the most exciting recent work in population genetics is that showing how wide a variety of Dawkins's "selfish genes" exist in the genome. Given selection falling at different levels or on different sexes, intragenomic conflicts of various kinds arise, giving adaptationism a neat, built-in theory of maladaptations (Rice, 1994). Selection at one level can produce maladaptations at another. The creation of new levels on which selection might act occasionally leads to breakthrough adaptations like multicellularity, in which formerly intensely competing individuals are welded into larger units (Maynard Smith and Szathmáry, 1995).

In our view, the problem is not with the principle of natural origins itself but with its persistent misapplication in the human case. Human sociobiologists with otherwise diverse beliefs have taken certain contingent generalizations from evolutionary biology on board as metatheoretical presuppositions to guide the formation of hypotheses that we believe should be left in the realm of hypotheses to be tested (see Miller, 2000, for a view something like ours). Among the most problematical are the presuppositions that (1) we can deduce adaptations directly from what would maximize individual or inclusive genetic fitness, (2) cultural causes are always proximate, and (3) group selection plays no role in the evolution of human social institutions. We think the proper use of the principle of natural origins is methodological, not substantive. If culture itself has the attributes of an inheritance system, then it makes sense to apply Darwinian analytical methods to that system of inheritance as well as to the genetic and see where the exercise leads. Will cultural evolution generally lead to genetic fitness maximization? Can cultural variation itself create heritable variation on which selection can act? Can enough of this variation be expressed at the group level for group selection to be an important force? These are among the most interesting hypotheses that we want to use the analysis to address. To imagine that the principle of natural origins dictates certain answers to them is, in the human case, to badly mislocate the boundary of Darwinian metatheory and hypothesis. The human/chimpanzee comparative data on imitation, not to mention a mass of other data indicating how important culture is in humans, makes importing the

unvarnished adaptationist metatheory from evolutionary biology a dubious proposition.

Human behavioral ecologists start with the idea that natural selection ensures that humans act, to a decent first approximation, as general-purpose genetic fitness maximizers. Considerations of cultural evolution and gene-culture coevolution have a strictly secondary role, and for most practical purposes they can be neglected, in the view of most human behavioral ecologists. As Alexander (1979, p. 80) puts it, "Cultural novelties do not replicate or spread themselves, even indirectly. They are replicated as a consequence of the behavior of vehicles of gene replication." Or, as Betzig (1997) says in reaction to claims for the importance of culture, "Everything we think, feel, and do might be better understood as a means to the spread of our own—or of our ancestors'—genes" (p. 2), adding, "I, personally, find 'culture' unnecessary" (p. 17).

Often, the strategy of asking what behavior would optimize fitness leads to useful insights. For example, consider mating strategies. When should females mate polygynously with a male that already has a mate, and when should they seek an unmarried mate? In the case of species in which males defend territories with resources on them, females should mate polygynously if the extra resources available on an already mated male's territory exceed those available on the best available unmated male's territory. Such "polygyny threshold" models were first applied to birds and nonhuman mammals, and they often work quite well. Borgerhoff Mulder (1992) has shown that one human population, Kipsigis farmers of Kenya, also follow the polygyny threshold model. Women tend to select husbands on the basis of the land they can offer a new wife to cultivate rather than by other criteria. The success of such models should not surprise us. Humans are the Earth's dominant species, and much of our behavior must be pretty adaptive most of the time to account for this success. At minimum, fitness-optimizing models provide a convenient benchmark against which to judge competing hypotheses. Interesting competing cultural evolutionary hypotheses do exist. For example, the basic subsistence adaptations of humans have been evolving rapidly, relatively speaking, throughout the history of our species. Most of these adaptations seem to have a large cultural component, and how we get from one to another, optimally or not, is certainly of interest. To ignore our most dynamic system for achieving our adaptations on an "argument" such as Betzig's is stubborn and willful ignorance.

A second important branch of human sociobiology is evolutionary psychology. Proponents of the influential school of evolutionary psychology represented by the authors in *The Adapted Mind* (Barkow et al., 1992) contend that fitness-optimizing arguments are directed at the wrong target by human behavioral ecologists. In their view, the real adaptations to focus on are the attributes of the mind that

optimally adapted us to live in Pleistocene environments. Contemporary environments have changed so radically from the past that it is vain to hope that behavior will be fitness-maximizing today. Evolution is too slow to have readapted the human mind significantly in the past few thousand years. The human mind is best conceived of as a collection of adaptations designed to solve specific problems of Pleistocene life—our "environments of evolutionary adaptedness"—not as a general-purpose fitness maximization system. (The fact that people are even more successful in the Holocene than the Pleistocene is puzzling on this argument, but the fact that we did evolve under Pleistocene conditions is probably important.)

These scholars model the mind as a large collection of rather narrowly specialized content rich algorithms that solve a series of narrow problems. For example, human adaptations to the Pleistocene were social. To judge from contemporary hunter-gatherers and from archaeology, small bands of people collaborated to gain subsistence, with a great deal of sharing within and between the constituent families of each band. Bands were linked into a larger social sphere, the tribe, from which mates were sought and help elicited in emergencies. The exchange economies of even the simplest human societies were greatly expanded in comparison to those of ancestral primates. Among the adaptations to life in Pleistocene societies must have been the ability to detect violators of complex social contracts.

Evolutionary psychologists want to use this Pleistocene-limited version of the natural origins principle to inspire hypotheses about evolved cognitive architecture that can be tested experimentally (Tooby and Cosmides, 1989). As with the empirical program of human behavioral ecologists, the results of these experiments are often quite convincing. For example, the classic work of Cosmides (1989; see also Gigerenzer and Hug, 1992) showed that humans are much better at solving logical problems posed as violations of social rules than those posed as abstract logical problems—and better at solving the social-rule problems than those with other familiar, concrete content. Cosmides argues that these data are consistent with the hypothesis that humans' social adaptation has equipped them with a powerful innate mental organ for detecting cheaters.

From our point of view, the main problem with this form of evolutionary psychology is, again, that the principle of natural origins has been misapplied. Now it seems to be licensing as metatheoretical assumptions the innateness of the important adaptations as well as fitness optimization (in past but not in present environments). Several of the leading figures in evolutionary psychology are radical nativists who believe, like Betzig, that the role of culture is greatly exaggerated by most social scientists. John Tooby and Leda Cosmides, for example, argue that social scientists have failed to distinguish between what they call "evoked" and "transmitted" culture

(Thornhill et al., 1997, pp. 230–234). Transmitted culture is what we call culture here: the product of human social learning. Evoked culture is the innate information that resides in human heads and is expressed contingently in different environments. Tooby and Cosmides (1989) introduced the term evoked culture to make the point that innate mental organs can be environment-contingent rules and hence can produce patterns of variation in space (i.e., in diffferent physical locations) that would be difficult to distinguish from transmitted culture. As a hypothesis to explain any given pattern of human behavior, evoked culture is a perfectly good candidate. Undoubtedly, adapted genes play a large role in human behavior, much along the lines that such nativists suggest. For example, the impressive rate at which we can encode and decode speech is the product of specialized auditory and motor pathways (Friederici, 1996). In general, however, testing ideas about less peripheral aspects of speech processing and language learning, such as how grammar develops, has proven rather difficult, and hypotheses like Tomasello's (1999), giving a large role to transmitted culture, are currently as viable as much more nativist views, such as Pinker's (1994). Given that humans live in intensely social groups structured by culturally transmitted institutions, and given that culture and individual learning generally lead to adaptive behavior, the bare finding that people are very good at social tasks does not speak loudly about the proximal causes of social behaviors. The nativist interpretation of the results of Cosmides's experiments seems to based upon the assumption that, at least in the ultimate sense, the products of natural selection all reside in the genes, on the principle of natural origins. This application of the principle at the psychological level makes no more sense than at the phenotypic. Experimental work by psychologists such as Nisbett, Cohen, and Tomasello shows that culture is an important part of human psychology and that to attempt to marginalize it *a priori* is just not a good bet as a research strategy, much less a legitimate deduction from the principle of natural origins.

We think that psychobiology offers plenty of evidence to rule out an extreme *tabula rasa* hypothesis but not nearly enough to rule out an important role for culture. Cultural scientists bring plenty of evidence to the table to rule out a strong version of the evoked culture argument but not nearly enough to rule out a complex role for evolved mechanisms in the acquisition and management of culture. For example, even if the diversity of human behavior in space were to be somehow explicable on the basis of only an innate human nature and environment, its diversity in time is harder to account for in this way. Over the past 10,000 years, human subsistence behavior and social organization have changed quite radically, even though neither genes nor environments have changed much at all. Although we can easily reject extreme nativist and extreme *tabula rasa* hypothe-

ses, the evidence currently available is far from sufficient to specify the exact division of labor between genes, culture, and individual learning (Richerson and Boyd, 2000).

In the remainder of this essay, with the nettle of biology tightly in our grasp, we illustrate the consequences of taking both the principle of natural origins and the importance of culture seriously with two example hypotheses. The classic claim of mid-twentieth-century cultural ecologists (e.g., Steward, 1955) was that human adaptation has two basic components: technology and social organization. Humans adapt to environments by evolving elegant tools to exploit the most diverse sorts of resources Earth has to offer. Human adaptations are social. Human populations take advantage of the principles of cooperation, coordination, and division of labor to a degree otherwise known only among the social insects and a few other lineages. Even by the middle Pleistocene we were an unusually widely distributed species, and for the past 50,000 years or so, we have been fairly abundant over most of our range. Let us imagine our nearly acultural chimpanzee like ancestors. What sort of selective pressures would have led to the evolution of accurate imitation of food-gathering strategies? What sort of adaptation is technology? Why is it rare? In this example, we stick to the conventional sociobiological assumption that culture is a proximal system of adaptation. Even so, to understand how culture works as a genetic adaptation requires taking the properties of cultural evolution seriously. What of the evolution of the social component of our adaptation? How might we come to cooperate in groups composed of distantly related individuals? Evolutionary theory makes strong predictions about cooperation, and the standard sociobiological theory well predicts all but a handful of cases. We are perhaps the most glaring exception, cooperating in large groups of distantly (genetically) related individuals. Our hypothesis is that group selection has a stronger purchase on cultural than genetic variation and that the social component of our behavior is substantially the result of culture participating in evolution as an ultimate cause, not just a proximate one.

HOW TECHNOLOGY WORKS

The principle of natural origins encourages us to ask why natural selection might have favored our capacity for culture. The imitative capacity psychologists have described, and the cultural traditions the capacity apparently supports, could only have evolved if they were adaptive. The capacity to acquire, store, manage, and use technological practices is at least one of the functions of our large brain. Most accounts of human origins take our current ecological dominance as evidence of a qualitatively new and superior form of adaptation and ask what evolutionary breakthrough led to this revolutionary new adaptation. For example, Lumsden and Wilson (1981, p. 330) remark

that "[*Homo*] overcame the resistance to advanced cognitive evolution by the cosmic good fortune of being in the right place at the right time." Our current ecological dominance is undeniable, although perhaps precarious, but the principle of natural origins encourages us to ask quite detailed questions about just what selection pressures would have operated leading up to any breakthroughs.

Cultural Evolution Is Fast and Cumulative

The human brain is a serious adaptive puzzle. It is a very costly organ (Aiello and Wheeler, 1995). Human brains account for about 10% of our total energy budget, versus something like 1.5% for average mammals. Aiello and Wheeler argue that one consequence of our expensive brain is that to pay its overhead we evolved a smaller gut (gut tissue is also costly per unit weight). A short gut meant that we had to eat more energy-intensive foods than our ancestors did. Thus, humans had to hunt, gather, and conduct their social life with some efficiency to support their brains under quite hostile physical conditions, in competition with other predators, scavengers, and plant eaters with much more economical brains and more efficient guts.

We believe that culture is most likely an adaptation to the Pleistocene climate variation (Richerson and Boyd, 2000). During the last glaciation, and by inference during most of the rest of the Pleistocene, climate did not vary only on the 100,000 year time scale of the classic ice ages. Climates were also spectacularly variable on time scales ranging from a few years to a few thousand years. For example, the period from 80,000 to 10,000 years ago was punctuated by more than 20 abrupt (~ 1°C per decade) warmings to about half of interglacial temperatures, not to mention considerable variation at both shorter and longer time scales (Ditlevsen et al., 1996; Broecker, 1995).

Our mathematical modeling studies show that a likely adaptive advantage of culture is its ability to respond to changing environments more rapidly than genes (Boyd and Richerson, 1985). This ability comes from coupling adaptive decision-making systems to the transmission system made possible by accurate, fast imitation. Take the two simplest kinds of models. One feature of culture is that it is a system for the inheritance of acquired variation. Individuals can imitate the behavior learned by others. If the rules that guide learning tend to be adaptive, then two forces, natural selection and learning, act together to favor the accumulation of adaptations. In the world of models, at least, this system is especially suited to adapting to environments that vary a lot, but with an appreciable, though not too large, resemblance between parents' and offsprings' environments. If environments vary too fast, then parents' behavior may be out of date, and individuals should learn for themselves. If environments vary too slowly, selection on genes keeps up well enough, and

the costly overhead of brain-tissue-consumptive culture weighs against culture as a system of adaptation. The Pleistocene was rich in just the kind of variation that favors the inheritance of acquired variation.

A second trick we can do with culture is to use preexisting cultural variants rather than our own random trials or inventions. Suppose we observe not only how Mom gathers but also the techniques of several other gatherers. Suppose we observe two or three variants. As we begin to practice gathering, we can try each variant a few times and retain the one that seems best. Furthermore, throughout life we may continue to observe and try out any likely-looking new variant techniques that seem promising. Depending on how accurately people can discriminate among varied techniques and how many different techniques one has an opportunity to observe, the biasing of imitation can be a weak or powerful force.

The neat result of the models is that even when decision-making effects are weak at the level of individuals, they can be powerful at the level of the population. This finding is closely related to the fact that natural selection is a powerful force at the population level, even when so weak as to be impractical to measure at the individual level. When any directional force acts in the same direction in an entire population and consistently for more than a few generations, the evolutionary response is swift. For selective forces to operate, including both biased imitation and natural selection, variation to select upon must exist. However, coupling individual learning to social learning means that trial-and-error learning can act as a source of new, generally partly adaptive, variation.

We believe (Boyd and Richerson, 1996) that the evidence suggests that our adaptive success also rests decisively on our ability to create cultural adaptations that can accumulate complexity, eventually coming to rival genetic adaptations in the sophistication of their "design." Even relatively sophisticated social learners like chimpanzees get only a very general idea of a behavior using social cues. Using this general idea, they refine their actions to a functional behavior using individual learning. This limits the complexity of the socially learned behavior to that which can be supported by individual learning at the individual level. The human ability to imitate accurately means that we can adopt the precise variant of a previous innovator, perhaps tracing back to some long-dead genius, and then add a new wrinkle of our own, which can in turn be imitated and improved by our successors. Eventually, human populations heap innovation upon innovation until we reach the limits of human minds to be taught the result. Even the cultures of simple societies accumulate far more genius than even the most brilliant individual innovator could muster. Most likely, the invention of language increased the number and sophistication of abstract concepts we

could learn. In simple societies, memory places limits on complexity that more recently have been relieved by the invention of writing and numbers (Donald, 1991). At the cutting edge, we again push right up against human cognitive limitations. Most of us now live by skills dearly won in classrooms by great mental exertion on both our own and our teachers' parts. The relative rapidity with which we could build up and adaptively modify complex technology is one leg of the adaptation allowed us in the Pleistocene to chase the ephemeral niches left underexploited as other species lagged behind in adapting to the kaleidoscopic changes in resources caused by rapid climate change. In the Holocene, the invention of agriculture gave us the tools to deteriorate the environments of competing and pest species faster than they could adapt to our modifications (Richerson et al., 2001).

Thus, we suppose that the environmental deterioration of the Pleistocene is the specific environmental factor that humans exploited to support their large, costly brains (Richerson and Boyd, 2000). Interestingly, many mammalian lineages show increased brain size in the Pleistocene. Other species may also have been using social learning to adapt to variable environments. However, no other mammalian species has developed the ability to use rapidly evolving complex tools to exploit variable environments. Our bipedal posture, which freed the hands to specialize in creating and using tools, was probably, a decisive preadaptation (Tobias, 1981). Coupling the capacity to imitate with the capacity to make tools allowed us to develop rapidly adaptations that would otherwise have required slow anatomical modifications. Lacking a flexible way to implement a diversity of cultural adaptations, no other species came to support such a radically enlarged and costly brain.

The promise of explicitly modeling and measuring the processes of cultural change is immense. For example, why has the Holocene witnessed a 10,000-year-long, raggedly progressive trend toward fancier technology and larger societies? What currently regulates rates of change in various components of various cultures? Are current anthropogenic climate changes likely to stress our ability to adapt to them? Ice-age climates will presumably return. Can complex societies adapt enough to cope with the very noisy climates that have prevailed during the past couple of million years? The extraordinary dynamism of human societies means that understanding our species using assumptions about equilibrium adaptations to given environments will be less productive than in other cases (Nelson and Winter, 1982).

WHY HUMANS ARE ULTRASOCIAL

Many critics of the orthodox schools of human sociobiology have argued that the problem is that their adherents leap to adaptation

without considering the complexities raised by development. Our own critique above is of this form if we take social learning to be a developmental process linking the evolving genes to the adaptive phenotypes. While true, this objection bites less sharply than it might otherwise because adaptationists commonly, and often successfully, neglect the details of genes and development when studying the evolution of adaptations. The tactic of taking genes and development lightly in the hope that progress can be made without needing to understand proximate causes is called the "phenotypic gambit" (Grafen, 1991). The phenotypic gambit is generally necessary when one studies adaptations. Development is a complex and difficult topic all its own, and usually the only practical way to proceed is to assume that selection has managed the developmental processes well enough that adaptations have been close to what we would predict from gross functional considerations. We endorse the judicious use of the phenotypic gambit; if we could not use it, we would have to wait until developmental psychologists had delivered a Mercedes model of the imitation process rather than a pick-your-own collection of Amsterdam bicycles. Related scientific programs typically have to cope with weaknesses in their partners and with the intimidating complexity of even well-known phenomena. The phenotypic gambit and allied strategies are necessary to finesse ignorance and complexity.

A critique that bites deeper is that human sociobiologists have generally neglected the ultimate role culture has played in human evolution. The coevolutionary concept of an ultimate-cause role for culture is very simple. Culture, like genes, creates patterns of heritable variation. Natural selection will inevitably play upon any pattern of heritable variation that arises in the world, as Richard Dawkins (1976) noticed and as Donald Campbell (e.g., 1965) had argued earlier. If cultural variation can respond to selection, it is just as ultimate a cause as genes. Of course, culture does not stand in isolation; it lives in brains and is no doubt heavily shaped by influences having their roots in genes and selection on genes. But the proximal causal arrow runs both ways, as we have already seen. Our psychology is shaped by our culture. Culture acts as a selective environment to which our genes will, in the long run, adapt. The term *coevolution* classically derives from the interacting evolution of pairs of species, such as predators and prey, diseases and hosts, and mutualists. We imagine that our culture is something like a symbiont. It lives in the same body as our genes but has a different life cycle and thus responds somewhat differently to natural selection. In our species, culture and genes are obligate mutualists; an individual cannot even survive without tolerably good genes and tolerably good culture.

We hope that the gene-culture coevolutionary idea seems perfectly intuitive to most of our readers. Be warned, however, that you are

being invited down what many evolutionary social scientists believe is a garden path. The issue is whether or not gene-culture interactions in humans are fully or only partially coevolutionary. The more prominent hypothesis is that the gene-culture system is a degenerate example of coevolution. Genes have no doubt evolved to constrain the evolution of cultural variants in ways that favor the fitness of the evolving gene. This dynamic is what Lumsden and Wilson (1981, p. 303) called the "full coevolutionary circuit." They emphasized evolution of evolved genetic "leashes" on cultural evolution. We think Lumsden and Wilson's dynamic is really the full circuit because selection also exists on the cultural variants and, thus, evolved cultural institutions can cause changes in the genome that favor cultural fitness. Culture is on a leash, all right—but the dog on the end is big, smart, and independent, not a well-trained toy poodle. On any given walk, who is leading whom is not a question with a simple answer. (See Durham, 1991, pp. 223–225, for a similar argument.)

Mechanisms by which culture might exert forces tugging in this direction are not far to seek. Cultural norms affect mate choice, and people seeking mates are likely to discriminate against genotypes that are incapable of conforming to cultural norms (Richerson and Boyd, 1989). Men who cannot control their testosterone storms end up exiled to the wilderness in small-scale societies and to prison in contemporary ones. Women who are an embarrassment in social circumstances are unlikely to find or keep husbands. We believe that with (at minimum) tens of thousands of years in which to operate, natural selection on cultural variation could easily have had dramatic effects on the evolution of human genes by this process. Some of these effects no doubt just energize Lumsden and Wilson's limb of the coevolutionary circuit, favoring better genetic leashes. Humans are still in part wild animals; our cultural adaptations often still serve the ancient imperatives of genetic fitness. However, we think the evidence supports the hypothesis that the coevolutionary circuit is "doubly full." The leash works both ways. Humans, we might say, are a *semi-domesticated* species. Cultural imperatives are built into our genes. Not only can culture act proximally to constrain behavior via institutions, skills, and values; by constraining behavior in similar ways over hundreds of millennia, it is also a major source of ultimate causes of human "nature."

Group Selection on Cultural Variation Selected New Social Instincts by Coevolution

The other major leg of human adaptation is our complex social organization—and our form of social organization is potentially a result of selection on cultural variation and coevolutionary adjustments on the genetic side. The coresidential bands that most ethnographically known hunter-gatherers lived in are only a little larger

than those of chimpanzees (Dunbar, 1992), but human social organization includes a tribal level that is unique to our species. In the simpler human societies, a few hundred to a few thousand people, typically occupying several residential units, speak the same dialect, participate in a common ceremonial system, maintain a level of internal peace and security against hostile groups, and aid one another in subsistence emergencies.

Other ultrasocial animals—including to one other mammalian example, the naked mole rats of Africa—create large societies by multiplying the number of close genetic relatives. The creation of reproductive and sterile castes by the social insects offers examples of several independent origins of this system. Humans have taken a quite different route to ultrasociality. As Campbell (1983) observed, human societies have reproductive competition among the cooperators, leading to societies that exhibit considerable self-sacrificial altruism (e.g., heroism in war) and considerable within-group conflict (e.g., feuding). Some societies exhibit extremes of both warrior self-sacrifice and internal conflict rooted in subtribal-scale loyalties—a trick that seems to defy the evolutionary law of gravity (Hamilton, 1964) as it applies to all other species. The proximal mechanisms by which cultural institutions can harness phenomena like Southerners' touchy sense of personal honor to functional large-scale organizations like the excellent armies of the Confederacy in the American Civil War are tolerably well understood (Boehm, 1984; Salter, 1995).

We have proposed what we call the "tribal social instincts hypothesis" to account for our peculiar pattern of social organization (Richerson and Boyd, 1998, 1999, 2001). The tribal social instincts hypothesis is based on theoretical analyses suggesting that group selection plays a more important role in shaping culturally transmitted variation than it does in shaping genetic variation. In our simplest model of the process, we imagine that humans come to use conformist biases in acquiring culture (Boyd and Richerson, 1985, ch. 7; Henrich and Boyd, 1998). Conformity is adaptive under a wide range of conditions because the commonest thing people are doing in a given environment is frequently a very good thing to do relative to most easy-to-discover alternatives (When in Rome, do as the Romans do). As a byproduct, conformity has the effect of preserving between-group variation and suppressing within-group variation. Most evolutionists doubt that group selection on genes is very often important, because it is so hard to maintain variation between groups—particularly variation for traits such as altruism, which are selected against within-groups.

Almost everyone agrees that human material culture was of essentially modern levels of sophistication by the Upper Paleolithic transition, 50,000 years ago (Klein, 1999). Even if the cultural group selection process did not start until that time, human minds have been

selected for 2,000 generations in social environments in which the innate willingness to recognize, aid, and, if necessary, punish fellow group members has been favored by coevolution. That is, cultural group selection has produced traditional institutions that have penalized genotypes that hewed too tightly to individual selfishness, Hamilton's kin-selection rules, or reciprocity strategies to deal with non-relatives. If cultural institutions can generate sufficiently costly punishments for deviations from their rules or provide the benefits of group cooperation mainly to cooperators, any genetic variation underlying behavioral dispositions will fall under selection favoring genotypes that avoid the punishments and earn the rewards. We suppose that the resulting tribal instincts are something like principles in the Chomskian linguists' "principles and parameters" view of language (Pinker, 1994). The innate principles furnish people with basic predispositions, emotional capacities, and social skills that are implemented in practice through highly variable cultural institutions (the parameters). People are innately prepared to act as members of tribes, but culture tells us how to recognize who belongs to our tribes, what schedules of aid, praise, and punishment are due to tribal fellows, and how the tribe is to deal with other tribes—allies, enemies, and clients.

Because the tribal instincts are of relatively recent origin and because our genes still fall under selection pressures obeying Hamilton's rule, they are not the sole regulators of human social life. The tribal instincts are laid on top of more ancient social instincts rooted in kin selection and reciprocal altruism. These ancient social instincts conflict with the tribal. We are simultaneously committed to tribes, family, and self, even though the conflicting demands often cause us great anguish, as illustrated in Freud's *Civilization and Its Discontents* (1930) and in many novels, including Graham Greene's *The Honorary Consul*. So long as reproductive competition among the cooperators exists, people still have to look out for their personal fitness interests, even as they try to do their civic duty.

We (Richerson and Boyd, 2001) argue that a considerable mass of evidence from a number of domains of knowledge supports the tribal social instincts hypothesis and calls into question competing evolutionary explanations. Nevertheless, much more work needs to be done before any hypothesis regarding the evolutionary origins of human sociality should be accepted as well verified. What we do claim, on the basis of the evidence we review, is that the tribal social instincts hypothesis, with its active, ultimate role for the process of group selection on cultural variation, is at least as attractive as any current competing hypothesis.

CONCLUSION

The fast and cumulative hypothesis to explain the original adaptive advantage of imitation in humans is a straightforward application of adaptive analysis. It is a simple argument from the principle of natural origins. However, if it or hypotheses like it are true, culture plays, and has long played, a central role in human evolution and cannot be marginalized. For example, the time scale of cultural evolution is rapid but not instantaneous. Indeed, although 10,000 years have passed since the end of the last big shift in Earth's environmental regime, the Pleistocene-Holocene transition, human cultural change has apparently not yet equilibrated with that change. The processes of cultural evolution are fundamentally important to understanding human behavior but have been studied relatively little, especially with sophisticated quantitative methods.

The coevolutionary tribal instincts hypothesis, if it or anything in its genre are correct, means that coevolution with culture has driven the evolution of genes in directions genes would never have gone if left to their own devices. Cultural institutions achieved the tribal (and now larger) scale of organization by partly domesticating genes. The human achievement of ultrasociality, tenuous and conflict ridden though it may be, seems to be one of those rare evolutionary transitions in which a new level of organization emerges because some form of group selection unites (no doubt *always* tenuously and conflictually in the beginning) previously fiercely competing entities into a larger-scale cooperative system (Maynard Smith and Szathmáry, 1995). This hypothesis is also perfectly consistent with natural origins. Large-scale human societies are (so far) extraordinarily successful because, on average, they increase the fitness of both genes and culture, quite like other successful coevolved mutualisms.

The principle of natural origins is the fundamental building block of Darwinian metatheory. No competing metatheory has much promise of giving us a truly deep and synthetic theory of human behavior. The trouble is not with the principle but with its misapplication in the human case. It especially does not imply what cultural scientists have come to fear: a trivialization of the role of culture in human behavior. Culture, its evolutionary processes and its coevolutionary effects, are all straightforward topics for Darwinian investigation. A mass of evidence argues that we cannot understand human behavior without adequate analysis of culture. This same evidence argues against using concepts like the superorganic to separate the study of culture from the rest of human biology. The superorganic concept was a tribal ploy used by twentieth-century social scientists to create and maintain disciplinary boundaries with biology (see Campbell, 1978, on the functions and dysfunctions of disciplinary boundaries). If we are correct, it never served a truly useful analytical role. Regardless of whatever useful function the concept and its

boundaries served in the twentieth century, they are now utterly senescent. The task for twenty-first-century human science is to put culture back into human biology.

Culture operates through biological mechanisms—brains, hormones, hands—and the causal pathways by which it acts are certain to prove densely tangled with genetic causes. The difficulty we have in following the threads of genetic and cultural influences on human behavior is the best evidence we have on this point. If the relationship between genes and culture were simple, the case would have been cracked long ago. Scientists should not be fainthearted in the face of complexity if that is where the real problem lies. Darwinism is rich in techniques for making progress in the face of intimidating complexity. The last, "tangled bank" paragraph of *The Origin of Species* is a lyrical passage that combines a downright mystical appreciation for the complexity of nature with a scientist's optimism that useful understanding is possible nonetheless. The extremes of superorganicism and innatism are useless simplifications that lead human scientists to avoid the hard but central problem of the human species: the natural origin of the cultural system of inheritance and all the things that people can create because their biology includes the capacity for imitation.

Cultural scientists should not be timid about being reunited with biology. Culture is a brawny phenomenon in no danger of being "reduced" to genes. Evolutionary biologists should not be timid about welcoming cultural scientists either; as biologists they command the methods cultural scientists neglected because superorganicism especially stigmatized Darwinism. All sorts of borrowings and interchanges across the biology-social science divide are likely to prove fruitful (Weingart et al., 1997). The only people with legitimate reason to fear a unified human biology with culture and genes playing their appropriate roles are those who want easy answers to hard questions.

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CHAPTER 5

Armchair Delusions Versus Empirical Realities: A Neurological Model for the Continuity of Ape and Human Languaging

ROGER S. FOUTS AND MARY LEE A. JENSVOLD

Truly discontinuous, all-or-none phenomena must be rare in nature. Historically, the great discontinuities have turned out to be conceptual barriers rather than natural phenomena. They have been passed by and abandoned rather than broken through in the course of scientific progress. The sign language studies in chimpanzees . . . have neither sought nor discovered a means of breathing humanity into the soul of a beast. They have assumed instead that there is no discontinuity between verbal behavior and the rest of human behavior or between human behavior and the rest of animal behavior—no barrier to be broken, no chasm to be bridged, only unknown territory to be explored.

—Gardner et al., 1989, p. xvii

INTRODUCTION

Our perspective on the world determines how we behave in the world. If we thought the world was flat, we would certainly avoid trying to sail around it. If we thought the Earth was the center of the universe, we might try to explore other planets, but without much success. While geocentric models are now regarded as an embarrassing part of our scientific history, we are in the middle of a major change in perspective with regard to our species' place in nature and our relationships with other organic beings. In the more than 150 years since Darwin wrote *The Origin of Species*, a great deal of evi-

dence has accumulated to stimulate a change from an outmoded, delusional view that "man" is superior to and different in kind from our fellow beings to a view emphasizing continuity for both the mind and body (Darwin, 1859/1991).

In stark contrast to the Darwinian worldview, the Platonic and Cartesian worldviews saw "man" as superior to all other beings, including woman. In the Greek view—the more traditional and ladderlike "chain of being" model—the inferior creatures were placed below the superior Greek male human. Descartes's view was slightly different, maintaining that a definite gap or difference in kind existed between man and the defective automata below him. It still assumed a chain of being that ordered our fellow animals in descending fashion on a scale of imperfection, but these imperfect automata were considered quite distinct and different in kind from man because they lacked reason and, being machinelike, were incapable of thought and feeling.

Plato's notion of the "ideal" was the basis of man's arrogant assumption of superiority, which implicitly carried with it the notion of "not ideal" as one descended the chain of being below man. From Plato's student Aristotle arose a companion concept in the law of contradiction. It stated that A cannot be both B and non-B; therefore A must be either B or non-B. This bivalence provided us with a false sense of "certainty" and "absolute prediction." True and false became our absolutes. Plato's "ideals" and Aristotle's "excluded middle" led to "essentialism," which was one of the main barriers to the theory of evolution.

Essentialism held that each species was completely distinct from all other species and was based on an eternal static essence. Variations were nothing more than imperfections in the underlying essence. This model placed permanent gaps in the phylogenetic scale. Today this archaic, superstitious notion still survives, and science still implicitly clings to the concept that humans are somehow different from, and superior to, the "have-nots."

Darwin's principle that all biological functions vary in degree rather than kind is certainly accepted with regard to blood and bone. However, application of this principle to the mind still remains embattled, and at the center of the battle is language. Long tradition has perpetuated the armchair claim that language is the defining trait of human primates. This tradition has persisted through the rise and fall of many a paradigm, despite a surprising lack of scientific study concerning its place in nonhuman animals (Seyfarth and Cheney, 1997). Even today, many armchair theorists hold to the uniqueness of human language in spite of strong empirical evidence to the contrary (Gardner et al., 1989).

It is next to impossible to begin a discussion on language without

a mention of the Cartesian linguist Noam Chomsky. His influence is apparent in the fact that he currently ranks as one of the ten most-cited writers in the humanities (Pinker, 1994). Chomsky (1975) claims that grammar is innate and that the structures responsible for it can be conceived as a language "organ." Because of this, one might think to place him squarely in the ranks of Darwinian theory. After all, Darwin himself stated that natural selection ". . . can act on every internal organ, on every shade of constitutional difference, on the machinery of life" (1859/1991, p. 61). This would seem to be a strong base on which Chomsky could place his theory. On the contrary, however, Chomsky is a vocal opponent of Darwin, not only in regard to language but also in regard to natural selection as a mechanism for evolution. Chomsky goes so far as to say that natural selection ". . . amounts to nothing more than a belief that there is some naturalistic explanation for these phenomena" and that ". . . the laws that determine successful mutation and the nature of complex organisms are as unknown as the laws that determine the choice of hypotheses" (1972, p. 97).

Some of Chomsky's epigones have tried to correct his disdain for natural selection by incorporating evolutionary ideas into his work. However, the queasiness felt by a Darwinian after reading Chomsky's statement will not quickly fade upon this synthesis. Steven Pinker (1994) criticized Chomsky's elimination of natural selection in an attempt to show his theory of innate grammar in light of instinctual behavior. Unfortunately, while praising Darwin's theory for eliminating the theological "chain of being" argument, Pinker systematically refutes the related concept of continuity of organic beings.

Pinker's assertion is based not on observation but on an armchair misunderstanding of the distinction between species. Darwin's conception of species was not based on static, well-defined distinctions. Instead, Darwin saw *species* as a term

> arbitrarily given, for the sake of convenience, to a set of individuals closely resembling each other and that it does not essentially differ from the term variety, which is given to less distinct and more fluctuating forms. The term variety, again, in comparison with mere individual differences, is also applied arbitrarily, for convenience' sake. (1859/1991, p. 40)

Why is the continuity of ape and human language so vehemently and emotionally rejected by some quarters of academe when Darwinism maintains that the cognitive difference between apes and humans is one of degree? It is because many academics still adhere to Aristotelian superstitions and the Cartesian Dark Ages notion that humans are outside of nature and different in kind from our fellow

animals. While this arrogant position may be popular and handy for justifying exploitation and abuse, it is out of touch with biological reality and serves little purpose other than puffing up arrogant, delusional human pretensions.

SIGN LANGUAGE STUDIES OF CHIMPANZEES

Cross-Fostering

While chimpanzees have great difficulty adapting their vocalizations to human speech (Hayes and Hayes, 1951; Hayes and Nissen, 1971), chimpanzees can freely move their hands, which means that a gestural language is well suited to their abilities. R. A. and B. T. Gardner recognized this in their sign language studies with young chimpanzees. In 1966 the Gardners brought 10-month-old Washoe to the University of Nevada at Reno when they began their cross-fostering study. The Gardners (1998) described their approach as follows:

> Cross-fostering a chimpanzee is very different from keeping one in a home as a pet. Many people keep pets in their homes. They may treat their pets very well, and they may love them dearly, but they do not treat them like children. True cross-fostering—treating the chimpanzee infant like a human child in all respects, in all living arrangements, 24 hours a day every day of the year—requires a rigorous experimental regime that has rarely been attempted. (p. 292)

The Gardners and students involved in the cross-fostering project used only American Sign Language (ASL) in Washoe's presence (Gardner and Gardner, 1971, 1974, 1989; Gardner and Gardner, 1969).

> In teaching sign language to Washoe [and to other, later cross-fosterlings] . . . we imitated human parents teaching young children in a human home. We called attention to everyday events and objects that might interest the young chimpanzees, for example, THAT CHAIR, SEE PRETTY BIRD, MY HAT. We asked probing questions to check on communication, and we always tried to answer questions and to comply with requests. We expanded on fragmentary utterances using the fragments to teach and to probe. We also followed the parents of deaf children by using an especially simple and repetitious register of ASL and by making signs on the youngsters' bodies to capture their attention. (Gardner and Gardner, 1998, p. 297)

In 1970 Washoe left Reno with Roger and Deborah Fouts for the Institute of Primate Studies (IPS) at the University of Oklahoma. The

Gardners began a second cross-fostering project with four other infant chimpanzees. Moja, Pili, Tatu, and Dar were born in American laboratories, and each arrived in Reno within a few days of birth. Moja arrived in November 1972, and cross-fostering continued for her until winter 1979, when she left for IPS. In 1980 Washoe and Moja moved with the Foutses to the Chimpanzee and Human Communication Institute (CHCI) on the campus of Central Washington University in Ellensburg. Tatu arrived in Reno in January 1976 and Dar in August 1976. Cross-fostering continued for Tatu and Dar until May 1981, when they left to join Washoe and Moja in Ellensburg. Pili arrived in Reno in November 1973, and he died of leukemia in October 1975.

Size of vocabulary, responses to *Wh* questions, number of utterances, proportion of phrases, variety of phrases, length of phrases, complexity of phrases, and inflection all grew throughout five years of cross-fostering (Gardner et al., 1992; Gardner and Gardner, 1974, 1989, 1998). "Washoe, Moja, Pili, Tatu, and Dar signed to friends and strangers. They signed to each other and to themselves, to dogs and to cats, toys, tools, even to trees" (Gardner and Gardner, 1989, p. 24). Signing was a robust behavior in the chimpanzees.

Process Versus Stasis: Language Development in Ape and Child

Watching my first grandchild (R. S. F.), Marley Grace, take the first steps in her development of language is very exciting. Her clear turn-taking at the breast, her engaging eye contact, and her prosodics and gestures are truly amazing. But at four months of age she demonstrates only the beginnings of a long process of language development. If we were to plot this as a curve over a lifetime, it might be more of an inverted U, and a rather bumpy one at that. In his last years, my father (R. S. F.), who was noted in his youth as a good debater and quick thinker in an argument, struggled to find words or even to remember my name. Certainly, if we were to create a family of curves, we would fill a scatter plot, with some individuals rising quickly to the zenith and others barely leaving the abscissa. But for most humans, language development is an orderly process: children can enjoy the ride to the heights their parents have attained, while at the same time the parents may begin to worry about their own slide down the other side.

The human companions to the cross-fostered chimpanzees maintained meticulous field records of the signed output of Moja, Tatu, Pili, and Dar. From the field records, the Gardners plotted vocabulary and phrase development for the 60 months of the cross-fostering project (Gardner and Gardner, 1994, 1998). A phrase is two or more different signs within two utterance boundaries. Utterance boundaries are defined by a pause, marked by a relaxation of the hands within the signing area or a removal of the hands from the signing

area altogether. In the field records, the observers indicated utterance boundaries with a slash. A reiteration—that is, a repetition of a sign for emphasis—did not count as a phrase because it did not consist of two different signs. Pili died at 24 months, and his records were plotted only to the 18th month. The vocabulary of the chimpanzees grew robustly to the 60th month. The volume and variety of phrases also increased steadily. The growth of phrases with three or more signs increased steadily after the 18th month. The Gardners found that vocabulary and phrase development in the cross-fostered chimpanzees, like that in human children, showed degrees of change rather than Cartesian discrete junctures.

Cultural Transmission: Project Loulis, or the Chimpanzee Who "Got It"

In 1979 Washoe adopted a 10-month-old son, Loulis. To demonstrate that Loulis would learn signs from Washoe and other signing chimpanzees without human intervention, we restricted human signing in Loulis's presence to seven specific signs: WHO, WHAT, WHERE, WHICH, WANT, SIGN, and NAME. Other than these signs, humans used vocal English to communicate in his presence. Loulis began to sign in 7 days; at 15 months of age he combined signs; and at 73 months of age his vocabulary consisted of 51 signs (Fouts, 1994; Fouts et al., 1989; Fouts et al., 1982).

The human observers maintained written records of Loulis's signing and behavioral development. From these records we plotted the growth of Loulis's phrases. We used all of the records from his 10th month (the first month of the project) to his 72nd month. After the third year of the project, Loulis showed a steady increase in the variety of his phrases. This pattern was similar to that seen in Moja, Tatu, Pili, and Dar (Gardner and Gardner, 1998). After the fourth year of the project, there was a sharp increase in the variety of Loulis's phrases of three or more signs, such as HURRY YOU TICKLE. His phrase development paralleled that of the cross-fostered chimpanzees and children in that it grew gradually. Loulis's acquisition of phrases is particularly impressive because it occurred in the absence of human signing and because his only signing models were other signing chimpanzees.

Remote Videotaping

In June 1984 the signing restriction around Loulis ended, and we turned our attention to observing him through the use of remote videotaping (RVT)—a technique used to record behaviors of chimpanzees with no humans present. In the original method, three cameras were focused on the chimpanzees' enclosure. Later, a fourth camera was added. The cameras were attached to television monitors and a videocassette recorder (VCR) in another room. Only one cam-

era recorded at a time, and the VCR operator could control which camera was recording.

D. H. Fouts (1994) made 45 hours of RVT recordings to examine Loulis's interactions with Washoe, Moja, Tatu, and Dar, the other chimpanzees at CHCI. Loulis initiated 451 interactions, both signed and nonsigned, with the other chimpanzees. Of those interactions, 40% (181) were directed to his male peer, Dar. Loulis used 206 signs in his interactions, and 114 of those were directed toward Dar. Fouts also reported 115 private signs that Loulis made when his face and body were not oriented toward another chimpanzee.

The other chimpanzees signed to each other as well. A later study by Cianelli and R. S. Fouts (1998) found that the chimpanzees often signed emphatically during high-arousal interactions such as fights and active play. One example captured on videotape occurred after a fight between Dar and Loulis, and all the chimpanzees were still screaming. Loulis and Dar separated, and Washoe signed COME HUG to Loulis. He signed NO and continued to move away from her. These results indicate that the chimpanzees' signing is a regular part of their interactions.

Bodamer (1987) looked for instances of private signing by the other chimpanzees in the 45 hours of RVT recorded by D. H. Fouts (1994). He found 90 instances of private signing—that is, signing done in the absence of interactive behaviors such as looking toward another individual. He classified these into the categories of private speech that humans use (Furrow, 1984). We later recorded 56 more hours of RVT and found 368 instances of private signing (Bodamer et al., 1994). In both samples, one of the most common categories of signing was "referential" (59% of the signs in the 56-hour sample). In this category, the chimpanzees signed about something present in the room—for example, pictures in a magazine. The "informative" category, consisting of utterances that refer to objects or events that are not present, accounted for 12% of the signs in the 56-hour sample and 14% of the signs in the 45-hour sample. For example, Washoe signed DEBBI to herself when Debbi was not present.

One category of private signing was "imaginative" (Furrow, 1984) and accounted for 17 instances in the 56 hours of RVT. We later recorded 15 hours of RVT while the chimpanzees' enclosure was filled with toys. We found six instances of imaginary play. We classified these into categories of imaginary play that human children use (Matthews, 1977). There were four instances of animation, in which a chimpanzee treated an object as if it were alive. For example, Dar signed PEEKABOO to a stuffed bear. There were four instances of substitution, in which a chimpanzee treated one object as if it were another. For example, Moja wore a shoe and signed SHOE. She then removed the shoe, put a purse on her foot, and zipped it up (Jensvold and Fouts, 1993).

Williams (1995) used RVT to examine the five chimpanzees' nighttime behavior. The chimpanzees were more active at night than we had previously assumed. There were even a few instances of signing in their sleep.

The research on Project Washoe demonstrated that chimpanzees can acquire and communicate with American Sign Language. Chimpanzees can pass their signing skills on to the next generation, demonstrating cultural transmission of acquired language. They use their signs to converse spontaneously with each other when no humans are present, they sign to themselves, and they use their signs during imaginary play. Ape language behavior is rich enough to provide texts that could be analyzed for a number of linguistic traits that are shared with human language.

NEUROLOGICAL CONTINUITY

The "Language Organ"

Cartesian linguists continue to insist that language is a uniquely human behavior in spite of the continuity of linguistic behavior in cross-fostered chimpanzees and humans. The armchair conjecture made by Cartesian critics of the chimpanzee sign language research is in direct conflict with the empirical data. For example, Pinker (1994) claims that

> Even putting aside vocabulary, phonology, morphology, and syntax, what impresses one the most about chimpanzee signing is that fundamentally, deep down, chimps just don't "get it." They know that the trainers like them to sign and that signing often gets them what they want, but they never seem to feel in their bones what language is and how to use it. (p. 349)

Later, Pinker puts his academic foot even farther down his throat when he states, "The chimps seldom sign spontaneously; they have to be molded, drilled, and coerced" (p. 348).

The empirical evidence presented in this paper demonstrates that the difference between chimpanzees and humans is one of degree, just as it is with all of our fellow animals. This evidence is consistent with the Darwinian notion of continuity. The chimpanzee and other fellow apes just happen to be our next of kin in our phylogenetic family.

The explanation offered by many Cartesian linguists (Pinker, 1994) for the uniqueness of language in humans is that lucky mutations are responsible for the emergence of language in modern *Homo sapiens*. This is despite the lack of evidence to show that such a lucky mutation has ever occurred in the evolution of any other species. These mutations become luckier yet when one realizes not only that

one must develop the ability to produce language but also that someone must be around who possesses another mutation that confers the ability to understand it. In addition, both of these mutations must remain in the gene pool until these two lucky individuals meet. When the complexities of language, which include ". . . sequential and simultaneous development, coordination, reorganization, and elaboration of social, cognitive, symbolic, gestural and sound systems" (Parker, 1985, p. 618) are set against this model, it appears simplistic rather than parsimonious (Hewes, 1973).

Current Cartesian language theories build their foundations on Chomsky's notion of the language organ. Chomsky's language organ is essentially a metaphor for the structures within the brain that facilitate speech (Chomsky, 1975; Pinker, 1994). By utilizing the concept of an organ, Cartesian linguists evoke an image of innateness for human language that has consequences as to how one views both the ontogeny and phylogeny of language development. For example, by viewing language as the function of an imagined organ, one can postulate that humans are born with language. This view eliminates the need for intermediate stages of grammar and proposes a distinct gap between lexical and grammatical aspects of language. According to this model, a child only has to be exposed to language in order to acquire language; this leaves little room for diachronic language change between categories. There is no room in this view for interaction between language use and language form (Bybee et al., 1994) or for the social interaction between the child and her linguistic environment, which is necessary for language acquisition outside of an idealized theoretical situation (Stokoe, 1983).

The closest thing that researchers can currently identify as a language organ is a loose collection of areas within the brain that manifest similar language deficits when damaged in adults. These structures have been synthesized by Geschwind (1970) into a neurological language processing model that includes Broca's area in the frontal lobe, Wernicke's area in the temporal lobe, and the angular gyrus, which acts as an intermediate between the visual cortex and Wernicke's area. Cartesian theories of language evolution predict that these areas are the result of a mutation that either produced them or transformed them from nonlanguage areas into parts of a uniquely human language organ. However, far from being neuroanatomical adaptations exclusive to humans, the components of Geschwind's model have been found in nonhumans.

Gannon et al. (1998) found asymmetries in the planum temporal (part of Wernicke's area) in the left hemisphere in 17 of 18 chimpanzee cadaver brains. These results and reports of asymmetries in the angular gyrus were later confirmed with the use of MRI (Hopkins et al., 1998). The comprehension of human speech by chimpanzees (Fouts et al., 1976; Shaw, 1989) suggests that these areas

may also have a homologous function.

Evidence suggests that the structures of Geschwind's model are not specific to language. An early example of nonlanguage functions within these structures came from patients with brain injuries in the left hemisphere specific to the areas traditionally associated with language production. These individuals displayed language production disabilities and trouble sequencing different manual actions, such as turning a knob followed by flipping a switch. While they could repeat single syllables and do repetitive hand movements, the troubles were associated with sequential movements in general (Kimura, 1976).

Bischoff-Grethe et al. (2000) found neural activity in Wernicke's area when subjects were presented with nonlinguistic sequential visual patterns. These results were the same whether the subject was or was not made aware of the pattern. This suggests that the function of Wernicke's area is associated with general pattern recognition and prediction—traits central to the survival of most species.

Area F5 in monkeys, the rostral part of the ventral premotor cortex, is proposed to be homologous to Broca's area in humans and is also active when monkeys perform manual actions. Rizzolatti and Arbib (1998) reported that area F5 in monkeys is active when they observe the motor behavior of other monkeys. The observing monkey's neurons in area F5, mirror neurons, appear to map the motor neurons that are active in the actor. Neural activity in the mirror system fires in a way that is sympathetic to the action observed. This system is a neurological bridge between the observer's perceptions and the actor's actions. What is remarkable about the mirror system is that different patterns of neural activity in the mirror neurons occur for different actions. For example, the pattern of neural discharge in a monkey grasping a raisin and in the observer of that action will be different from the pattern in a monkey grasping a larger item and the observer of that action. Furthermore, these patterns would be different from that in a monkey grasping at nothing and in the observer of that action.

Signs of ASL and gestures have grammatical properties. Armstrong, Stokoe, and Wilcox (1994) illustrate how arm movements involved in visible gestures contain the framework on which a grammar can be built. Since a gesture occurs in three-dimensional space, the directionality of a single sign can serve as the syntax of an entire sentence. For example, the sign CATCH is made by moving the fist of one arm across the body, then catching that fist with the other hand. This single sign has syntax and contains a subject (the fist), verb (the movement), and direct object (the contact between fist and open hand). Wilbur (1980) further explains that

> the key to understanding ASL syntax, particularly word order, is the recognition that locations in space are used

> for inflectional purposes. Within the "signing space" (the allowable area in which signs may be made), signs may be moved from one location to another to indicate differences in subject and object. (p. 19)

The cross-fostered chimpanzees also showed evidence of this behavior. The Gardners (Rimpau et al., 1989; Gardner and Gardner, 1998) report that the cross-fostered chimpanzees changed the places where signs occurred. For example, Dar placed signs such as BRUSH on himself and on a person to show who was to receive the action. Here again we see a noun, a verb, and a direct object within a single gesture.

It may be the case that language is the function of a language "organ"—one based on the social interaction of an actor and an observer. This language organ would be one that does not exist in humans alone for the purpose of a universal grammar; rather, it also exists in many other social animals for the adaptive purpose of understanding and predicting one another's actions. The behavior of chimpanzees who have been immersed in a rich linguistic environment parallels behaviors used in human language. Results such as these threaten a philosophy that clings to a vast gap between human and nonhuman animals.

Reexamination of the traditional language structures proposed in Geschwind's neurological model suggests that these structures are responsible for cognitive abilities that are more general than those proposed in a language-specific model. These general abilities appear to be more closely related to the production of sequential motor actions and the perception of sequential patterns. The sequential nature of spoken language is described more parsimoniously as the evolutionary result of encoding and compressing a system into one that is expressed in time from one that was originally expressed in time and space (Stokoe, 1980). Motor and mirror neurons within a structure homologous to Broca's area in nonhuman primates could be the biological bridge between observed action and motor production. Further, Wernicke's area may contain a mechanism for parsing the inherent properties of gestures into the actor, action, and object components of sentence structure.

The implications of this neurological model are twofold. First, the study of the biological foundations of language can be expanded to include social communicative contexts that are absent in the study of language using the Cartesian worldview. This can be accomplished by examining language as a system of communication driven by cognitive abilities necessary for survival in many social animals, such as predicting and understanding the actions of others. Further research can expand to such diverse areas as motor memory and theory of mind, thus making the rules of social interaction the rules of language use. Second, the scope of research can be expanded to include

the capabilities of other species by examining language as a suite of behaviors congruent with social behavior. Such a scope accounts for the ability of chimpanzees raised in a linguistically rich environment to functionally communicate via the symbols and rules of human discourse and to transmit these abilities to a second generation.

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Contributors

ROBERT BOYD is a professor in the department of anthropology at the University of California, Los Angeles. His research focuses on culture as an essential human adaptation equally important as, for example, bipedal locomotion. Studies have included the evolutionary psychology of the mechanisms that give rise to and shape human culture. With Peter J. Richerson, he is the author of *Culture and the Evolutionary Process* (University of Chicago Press, 1985).

ROGER S. FOUTS is a professor of psychology and co-director of the Chimpanzee and Human Communication Institute at Central Washington University in Ellensburg, WA. Since 1967 he has been instrumental in Project Washoe, which studies chimpanzees that use American Sign Language and live together as a social group. His book *Next of Kin* received the "Best 100 Books of 1997 Award" from both *Publisher's Weekly* and the *Los Angeles Times*.

MORRIS GOODMAN, distinguished professor at Wayne State University School of Medicine in Detroit, is both a contributor and co-editor of this volume. He is a pioneer in the development of a molecular approach to better understanding the evolutionary origins of humans and other primates. His honors include the 2002 Charles R. Darwin Award for Lifetime Achievement from the American Association of Physical Anthropologists and, in 1996, election as a Fellow of the American Academy of Arts and Sciences. He is the founding editor of the *Journal of Molecular Phylogenetics and Evolution*.

LAWRENCE I. GROSSMAN is professor and associate director in the Center for Molecular Medicine and Genetics at Wayne State University School of Medicine in Detroit. His research focuses on the regulation and function of genes that code for enzymes involved in cellular respiration. He serves on the Editorial Boards of *Biochimica Biophysica Acta, Mitochondrion*, and the *McGraw-Hill Encyclopedia of Science and Technology*, and he was previously a contributing editor for *Science*.

DEBORAH L. GUMUCIO is an associate professor in the department of cell and developmental biology and co-director of the Center for Organogenesis at the University of Michigan. Her work in the field

of hemoglobin switching, which involves the study of how the human fetus switches from production of fetal hemoglobin to production of adult hemoglobin shortly after birth, is known for the use of evolutionary clues to infer changes in the regulation of the globin gene. Her collaborators on the paper include post-doctoral fellow David M. Thomas, graduate student Philip Schaner, senior research fellow Neil Richards, undergraduate Wesley Martus, and undergraduate Anish Wadhwa, all at the University of Michigan, and Morris Goodman, whose biography appears above.

MARY LEE A. JENSVOLD is an adjunct faculty member in the Primate Behavior and Ecology Program of the departments of anthropology and psychology at Central Washington University. She has taught courses on primatology, anthropology, psychology, and on chimpanzee husbandry and research.

ANNE SIMON MOFFAT is a co-editor of this volume. As Executive Associate of the Midwest Center of the American Academy of Arts and Sciences, she helped to organize the July 2001 National Science Foundation-sponsored "biocomplexity" meeting that commissioned the papers in this volume. An independent science writer, she has contributed to several books and has published more than 100 articles in periodicals such as *Science*, *The Sunday Times of London*, *The New York Times*, *Technology Review*, and *Genetic Engineering News*.

RICHARD POTTS is head of the Human Origins Program at the Smithsonian's National Museum of Natural History in Washington, DC, where he develops international collaborations to study the ecological aspects of human evolution. His research spans the evolution of early apes to the origin of modern humans and he has been involved with excavations at early human sites in the East African Rift Valley and in southern China, comparing evidence of early human behavior and environments from eastern Africa to eastern Asia. His most recent research focuses on the effects of past environmental instability on human origins.

PETER J. RICHERSON is a professor in the department of environmental science and policy at the University of California, Davis. His research interests include cultural evolution and the scientific study of the life and physical phenomena of lakes, ponds, and streams. In recognition of his work on Lake Titicaca, the largest freshwater lake in South America, an endemic fish *Orestias richersoni* was named for him. He has been a Guggenheim Fellow and, in 1989, won the J. I. Staley Prize from the School of American Research (with Robert Boyd).

DEREK E. WILDMAN received his Ph.D. from New York University in 2000 for research on the evolution of baboons from the Arabian Peninsula and is now a post-doctoral associate at Wayne State University.

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