

Will Genomics Do More For Metaphysics than Locke?

“Origin of man now solved. He who understands baboon would do more for metaphysics than Locke”
Charles Darwin, Notebooks

Darwin’s claim is probably guilty of pardonable exaggeration. After all he didn’t prove the origin of man, and Locke’s greatest contributions were to political philosophy, not metaphysics. But it may turn out that Darwin’s twentieth century grandchild, genomics, vindicates this claim both with respect to metaphysics and political philosophy. Here I will focus on the latter claim alone, however.

From the year that William Hamilton first introduced the concept of inclusive fitness and the mechanism of kin selection, biologists, psychologists, game theorists, philosophers and others have been adding details to answer the question of how altruism is possible as a biological disposition. We now have a fairly well articulated story of how we *could have* gotten from there—nature red in tooth and claw-- to here—an almost universal commitment to morality. That is, there is now a scenario showing how a lineage of organisms selected for maximizing genetic representation in subsequent generations could come eventually to be composed of cooperating creatures. Establishing this bare possibility was an important turning point for biological anthropology, for human sociobiology, and for evolutionary psychology. Prior to Hamilton’s breakthrough it was intellectually permissible to write off Darwinism as irrelevant to distinctively human behavior and human institutions. The unchecked contempt with which defenders of the autonomy of the social from the biological operated in their attacks on naturalistic approaches to social processes was both breath taking and without effective rejoinder.¹

The major components of the research program, the models and simulations, the comparative ethology, are well known. Once Hamilton showed that inclusive fitness maximization favors the emergence of altruism towards off-spring, a virtual riot of ethological activity began to identify previously known cases of off-spring care as kin-selected, and to uncover new examples of it. Once Hamilton was joined by Axelrod in identifying circumstances under which reciprocal altruism between genetically unrelated beings would be selected for, the community of game theorists began to make common cause with evolutionary biologists in the discovery of games in which the cooperative solution is a Nash equilibrium. This led in turn to the development of models of evolutionary dynamics for iterated games like cut-the-cake, ultimatum, hawk v. dove, which show how a disposition towards equal shares, private property,

and other norms among genetically unrelated beings may be selected for. An independent line of inquiry at the intersection of psychology and game theory developed an account of emotions which suggests that they too may have been selected for in order to solve problems of credible commitment and threat in the natural selection of optimal strategies in single games.²

But in a sense all this beautiful research remains what Lewontin and Gould once characterized as a “Just so Story”³. There was no evidence for it and it seemed unlikely that there ever would be. This is unsurprising; after all, behaviors, dispositions, norms, and social institutions are not among the hard parts preserved in the fossil record. There is of course comparative ethology, neurophysiology and neuroanatomy. But at most these provide data from which we can reverse-engineer our way into, ...well, into just so stories—hypotheses among which we cannot choose on the basis of independent *evidence*.

There is only one evidential source that stands a chance of doing any better: genomics. In this paper I want to explore what evidence genomics can provide about the actual evolution of human cooperation, when combined with phylogeny, comparative ethology, neurophysiology and neuroanatomy, and paleontology. To see its potential however, first, let’s consider how it can choose between competing accounts of human prehistory. This will give us an idea of how genomics can turn questions hitherto supposed to be purely speculative into matters open to testable answers.

By genomics I mean the comparative and often computational study of the nucleotide sequences and the functional organization of the human genome and the genomes of many other species of animals, plants, and fungi. The Human Genome Project has given us a first and second draft of the 3 billion base pair DNA sequence of the human genome. It has so far given us a little more information about the human genome. For instance, it appears that even more of it is “junk” DNA than molecular biologists have thought; “junk” DNA has no role in development or normal human function, and is just along for the ride, so to speak. And it now appears that there are only about 30,000 to 60,000 genes in our genome, which makes it a small multiple of the size of the fruit fly’s genome. But at an accelerating rate genomics--the comparative study of the human and the DNA sequences of other organisms--will begin to give us the sort of detailed information about our genomes we never dreamed of, and will give it to us as the result of methods we can automate and turn over to computers. Learning about our genomes and their protein products will cease to require genius, and at most demand ingenuity. Learning exactly which DNA sequences among the 3 billion nucleotide bases express genes, and which genes they express is matter of “annotation” of the DNA sequence the Human Genome Project has provided. Even before the whole sequence came into our hands, comparative genomics was providing evidence about large tracts of history about which only informed speculation had hitherto been possible.

We are inclined to think of history as having begun when written records did, about 3000 years ago in the near east, and a thousand years later in Mesoamerica. But DNA sequence data already in hand extend our knowledge of the general lines of human history so far back as to turn the Inca empire, the fall of Rome, the building of the Great Wall of China, or the founding of Sumerian Ur into matters of recent history. DNA sequence data can answer derelict perennial questions about human origins and prehistory that have hitherto been the domain of pure speculation. Like the bar-code on a can of beans on a supermarket shelf, our DNA sequences are labels from which we can read off date and place of manufacture not just in geological time, but over the last 200,000 years with resolving power that already approaches only a few thousand years, just beyond the reach of carbon-14 dating. Seeing how fine-grained is the resolving power of the genetic bar-code in these cases should give us some confidence it can answer countless other questions hitherto beyond the reach of evidence. But to see this requires a little of the science of DNA sequences.

Everyone inherits their cell's mitochondria and the genes they contain only from their mothers, because the mitochondrion genes aren't in the nucleus of any cell--somatic or germline, and so don't make it into the sperm, which contains only DNA from the nucleus. But since mitochondria are in every cell, they are in every ovum, and so in every ovum fertilized by a sperm. By contrast, all males and everyone else who has a Y chromosome inherits it from his (or her) father (the parenthesized here accommodates the rare XXY females). Mitochondrial genes' DNA (mtDNA) and Y-chromosome DNA can be sequenced. Since individuals differ from one another in gene sequence, it is easy to order a sample of individuals for greater and lesser similarity in DNA sequences--whether in the nucleus or the mitochondrion. The more similar the sequences the more closely related two people are. Given an ordering of similarity in mtDNA and Y-chromosome DNA among people living today, and compared to some mtDNA and Y-Chromosome DNA sequence in another species, whose age is known, geneticists can work backwards to identify an mtDNA or Y-chromosome sequence from which all contemporary sequences must have mutated and descended; in effect they can draw a family tree of all the main lines of descent among mtDNA or Y-chromosome sequences, and they can date the age of various branches in this family-tree. mtDNA sequence data was available much earlier than Y-chromosome data; it led to the conclusion that every human being now living is descended from one particular woman living in eastern Africa--current Kenya and/or Tanzania, approximately 144,000 years ago. She alone, of all women then alive, has had an unbroken line of female descents from that day to this. Every other woman has had at least one generation of all male descendants, and so her mitochondrial sequences have become extinct.

Moreover, the narrowness in sequence variation among extant people reveals that we are ten times more similar to each other in sequence data than, for example, chimpanzees are similar

to one another in sequence data. It can also be established that this woman, called “Eve” by biological anthropologists, lived among a relatively small number of *Homo sapiens*, who must have gone through some sort of evolutionary bottleneck—i.e. most of our ancestors were killed off at some point in the recent past. As a result there were only about 2000 (+/- 1000) women altogether alive at the time Eve lived. Subsequent sequencing of a portion of the Y-chromosome has confirmed these conclusions. Indeed, as more and more sequence data comes in, about single nucleotide polymorphisms, and microsatellite loci, the conclusion has become inescapable (in spite of Chinese reluctance to accept it) that all present *Homo sapiens* are descended from this one African Eve and a relatively small number (about a dozen) of African Adams alive at the same time as Eve.⁴ This explains why intra-racial gene sequence differences are larger than interracial ones, why polygenetically (many gene) coded traits have not had sufficient time to assort into separate lineages, and so explains why race is not a biologically significant explanatory concept. The genetic similarity among humans suggests further that the obvious visible differences among us in skin color, hair color, facial characteristics, etc are both of relatively recent origin, and are most probably the result of natural selection in local environments and sexual selection.⁵

Besides telling us where and when we started from, following out differences in more and more available DNA sequences, geneticists have traced the details of early human migration out of east Africa both into western and southern Africa, and northward, dating the arrival of *Homo sapiens* on each of the continents to within a few thousand years, and explaining in some detail the peopling of Micronesia, Melanesia and Polynesia within the last 6000 years.⁶ And beyond chronology, sequence data provides other startlingly detailed revelations about matters of prehistorical narrative hitherto thought forever beyond answers. For example, consider the question that concerned novelists like Auel and Goulding, and many others: what became of the Neanderthals? Well, Neanderthal DNA is available in bones from the Neander valley in Germany. By comparing mtDNA and the ALU gene sequence—a bit of junk DNA sequence that repeats a distinctive number of times in chimp, *Homo sapiens*, and Neanderthal DNA—it can be shown that these three lines of descent don’t share these genes at all, as they would have to if there were any interbreeding among them. This is not surprising in the case of Chimpanzees and *Homo sapiens* of course. But that there was no interbreeding between our species and Neanderthal at all is very significant. That means that either *Homo sapiens* killed off the Neanderthal or gave them all a fatal disease, or otherwise out-competed them in a common environment. Probably, Cro-Magnon out-competed them, because there is archeological evidence that both populations existed side by side in Europe over many thousands of years.⁷ Similarly, the absence of any non-African Y-chromosome sequences among 12,000 Asian males from 163 different populations shows that the migrants out of Africa replaced any earlier Asian

populations, and did not interbreed with them either⁸

Further research will employ DNA sequence data to uncover the detailed narrative of events we never dreamed of reconstructing, and of other events our non-genetic records have misrepresented to us. For example, consider the origin of Agriculture in Europe about 10,000 years ago. How did it happen? There is some archeological evidence to show that farming spread from the near east northward and westward in Europe. But how? By cultural evolution one might presume: farming must have spread as people in one European valley noticed the success of those farming in the next valley to the south east, and copied their discovery. Others have held that the farmers came out of the near east, and like Cro-magnon out competing or extirpating Neanderthal, displaced—pushed out or decimated local populations, took over their territory and thus expanded the farming regions. Which hypothesis is right is not a question we could ever have expected to answer since these events took place before any *recorded* history, indeed before writing!

But recent studies, first of mtDNA, and now of Y-chromosome sequence differences in contemporary near-eastern and European populations, substantiate the latter scenario, the so-called “demic-defusion” model, a euphemism for the displacement of one whole population by another. MtDNA and Y-chromosome sequence data shows that the earliest migration from the near east into Europe occurred about 45,000 years ago, and its descendants now account for only about 7 % of contemporary European mtDNA., but earliest immigrants provide twice that proportion of mtDNA among the isolated Basque, Irish and Norwegian populations, and only half that frequency in Mediterranean populations. The next wave of migration about 26,000 years ago provided about 25 % of current mtDNA in Europe, while the third wave, 15,000 years ago account for about 36 % of contemporary European mtDNA. Agriculture arrived with a diffusion from the middle east about 9,000 years ago, and despite their recent arrival the mtDNA sequences these immigrants brought with them account for 23 % of the mtDNAs of current European populations, 50 % when we exclude the extreme Basque, Irish and Scandinavian populations. And this wave of migration provides mtDNA and Y-Chromosome DNA sequences in a “cline”—a gradient of change in proportions-- that moves in the direction from south-east to north-west.⁹ What the sequence data tells us is that near-eastern populations displaced indigenous ones year after year in wider and wider arc of expansion from the middle east, either driving them west eventually to the extremities of the European continent, or killing them off so that the only survivors of the original population of Europe were those inhabiting agriculturally marginal territories.

The question arises then, why didn't the earlier inhabitants acquire farming either independently or by imitation of their neighbors' practices? Surely there is no gene for farming which they lacked. Did farming and the social organization it produced make the near-easterners

that much more formidable than the hunter gatherers. If so, why? Further thought about this displacement should at least enable theorists of the evolution of cooperation among hunter/gatherer egalitarians to set some constraints on their models. The pay-offs to cooperation cannot be so strong as to prevent defeat by less egalitarian groups with storable commodities.

More recent population events, besides revealing who settled Melanesia, Micronesia, Polynesia, and the Western Hemisphere and when they did so, will tell us who arrived later, what groups went back the other way to settle Madagascar (where mtDNA sequences are quite different from mainland African mtDNA)¹⁰ and why the current residents of the Andaman islands east of the Indian mainland have mtDNA sequences far closer to those of east Africans than even the inhabitants of their neighboring islands or the Indian subcontinent.

Nonhuman DNA sequence data will be able to tell us still more about human prehistory. Sequencing the domesticated plants and animals and their extant undomesticated relatives can tell us where and when hunting and gathering first gave permanent way to farming, and thus to the beginnings of hierarchal social, political and cultural institutions. And they can date these events well before or with much greater accuracy than does the archeological evidence now available. In fact, what DNA sequence research thus far has shown is that both wheat and cattle were probably domesticated at least twice independently and at roughly the same time. Among the earliest domesticated cereals is emmer wheat, which however reflects two different sequences which diverged two million years ago, one traceable back only to southern and central Europe, including Italy, the Balkans and Turkey, while the other is ubiquitous to all regions of emmer cultivation. This suggests a double expansion from domestication in the middle east. There are two distinct types of cattle—the humped breeds of India and the humpless ones of Africa and Europe. They were both domesticated two thousand years after wheat, but their DNA sequences are sufficiently different to support the hypothesis of separate domestication.¹¹

Our question is whether gene sequencing can provide evidence that will distinguish among the alternative hypotheses about how, why and when human cooperation emerged? If the emergence of cooperation happened in several places independently, then it was presumably a matter of cultural and not biological evolution. Would it follow that we can immediately excuse genomics from the task of choosing among hypotheses about when, where, why and how it did so? Even if gene sequences can shed light on cultural evolution, there are other problems here, however, that we need immediately to face. First, if the just-so-stories have it right, cooperation, reciprocal altruism, a sense of justice as equal division, the emotions and the norms which enforce and express these institutions long antedate agriculture. Moreover, if anything, we should expect agriculture to begin to provide an environment in which many of dispositions we seek to explain would cease to be selected for. Once agriculture kicks in, the inclination to equal effort and equal shares becomes much less adaptive for individual survival. Storable

commodities and capital investment emerge, and the pay-offs to cooperating, sharing, reciprocation, defecting, hoarding, and free-riding, not to say domination, become quite different, hard to model, and probably produce unstable equilibria.

But, in fact the gene sequence data may be relevant to the evolution of cooperation in spite of all these caveats. The mitochondrial DNA sequences strongly suggest that sometime at or before 144,000 years ago, there was a bottleneck through which *Homo sapiens* came. This was long before the advent of agriculture, and presumably cooperation was already well established at that point. If *Homo sapiens* is the sole species in which substantial cooperation emerged, and if we could compare gene sequences between extant and extinct hominid lineages, then there would at least be a chance of uncovering a genetic difference relevant to this phenotypic difference. There are a lot of ifs here. But even if sociality is a forced move written into our gene sequence and not those of extinct hominids (a tendentious assumption yet to be discussed), it is obvious from gene sequence data that these other hominids left no representatives for us to sequence and compare. Or did they?

Recall that DNA has been extracted from Neanderthal bones upwards of 40,000 years old. This work is part of a new subdivision of biological anthropology which styles itself the study of *ancient DNA*. Quantities of DNA to be found in burial ground bones, around cave- and camp-fire detritus (and coprolites for that matter), fossil skulls, etc. are minuscule in quantity; proportions of the full sequence are low, and no particular portion—say functional genes as opposed to junk DNA—is preferentially preserved. Nevertheless, the prospects of worthwhile data are not entirely unfavorable. The optimism here as elsewhere in the genetic revolution is in the power of a molecular process: PCR—polymerase chain reaction for the amplification of DNA. This is a process employing a reagent that can catalyze the amplification (reproduction) of a single nucleotide sequence of any length into a million copies in only thirty rounds of replication. This means if a molecular biologist can extract just a simple molecule of the DNA from any specimen, an unlimited number of copies will shortly be available for sequencing, comparing to other sequences, and functional annotation (identifying the part of a gene, if any, it codes for). Naturally the older a specimen the smaller amount and the shorter the DNA molecules recoverable. Moreover, in sequencing hominid DNA, the greatest stumbling block is contamination with contemporary human DNA, which literally spews from the figure-tips of the investigator running the PCR procedure. But (as yet unreplicated) claims of successful amplification and sequencing include 80 million year old dinosaur bones, and 130 million year old insects trapped in amber.¹² So, if a) sociality is encoded in the genes, and b) we can find the right specimens, gene sequencing holds out the prospect of answering questions that are otherwise open only to speculation.

But what reason is there to suppose that either of these two assumptions obtain? What

indeed would we be looking for, were we to seek genes for cooperation? The first problem is to characterize the phenotype with sufficient precision. Identifying immediate protein products of particular genes is relatively easy. But identifying anatomical phenotypes is no trivial matter. Identifying behavioral ones, assuming they exist, is much trickier. In some cases identifying phenotypes has however become much simpler in the advent of “positional gene cloning”. The strategy involves identifying a trait—usually a disease or deficiency that appears to assort in accordance with Mendelian principles, locating a chromosomal marker in victims that does so as well, and then by automated means zeroing in on the specific gene sequence whose mutation or rearrangement is closely correlated with the trait. We thus establish the normal sequence as the gene for the normal trait in the “normal environmental range”. (It’s worth noting of course that the expression “gene for” is both widely misunderstood and misused. As I use it here, it can at least be quasi-operationally defined as the sequence that would be identified as the wild-type in a positional cloning exercise. The expression “gene for” must be understood as always relativized to a population and an environment). But the opportunity to employ positional gene cloning will be limited to behaviors that (in the normal environmental range) are rendered dysfunctional by a single gene error. It is very unlikely that the behaviors we seek, or the dispositions to such behaviors will be so controlled.

Current inquiry into the genetic basis of behavior begins with the assumption that behavioral dispositions which are statistically heritable or disproportionately represented in some genetically homogeneous groups, are matters of degree and dependent on a large number of genes. For example, the search for a genetic basis of criminality or intelligence—both taken to be dispositions measurable by criminal records or performance on a test—treats the disposition as a “quantitative trait” and seeks a “locus” in the genome statistically associated with that trait in the populations who manifest it in a high degree. These QTL (quantitative trait loci) studies are both politically and scientifically controversial. Few such studies reveal even a .20 correlation between the quantitative trait and some region of the genome on which a detectable marker can be found QTL studies face two scientific problems. A) most traits of interest are hard to operationalize so that individuals who instantiate them to the greatest degree are hard to identify; in effect, the traits of interest are not themselves phenotypes, but at most packages of phenotypes or the result of phenotypic and environmental interaction. B) At best QTL studies will identify a set of loci—perhaps 10 or more relatively large stretches of DNA--which are jointly highly correlated with the instantiation of a high degree of some quantitative trait in a “normal environmental range.” Nothing will be revealed by such studies about the biosynthetic pathways from these genes to the actual behavior they are supposed to be the “genes for”. It is easy to see how these problems will bedevil the attempt to employ genomics as evidence to test alternative theories about how human cooperation emerged.¹³

To make matters concrete suppose the behavioral disposition we seek to explain as an evolutionary adaptation is something as specific as “the disposition to engage in tit-for-tat strategies in iterated prisoner’s dilemma games”, or “the disposition to ask for $\frac{1}{2}$ in iterated cut-the-cake games”, or again, “the disposition to reject anything less than $\frac{1}{2}$ in an ultimatum game”. Call the first of these dispositions “TFT” for short. Now, no one supposes that any of these dispositions is a single gene-controlled phenotype like tongue-rolling. Genes just don’t seem likely to code for recognition of a complex environmental conditional set-up in which an abstractly described strategy is to be employed. Rather, if anything like TFT behavior is actually evinced by humans, then it may simply be the result of a package of genes for much simpler disposition that the TFT behavior supervenes on, without there being a phenotypic TFT disposition to be genetically encoded at all.

Here is a striking example of this sort of thing: the male mouse is disposed to kill all mouse pups not its own off-spring—a highly adaptive bit of environmentally conditional behavior that maximizes its genetic representation. But how could nature have programmed the male mouse with the power to make the required genealogical discriminations, given the similarity in look, smell, or other feature a mouse can detect in pups? It didn’t have to. In stead, nature found a “quick and dirty” substitute that does just about as well. Male mice have a genetically hardwired pup-killer disposition. But mice do not live in large colonies, and nature equips the male mouse with a further package of genes that automatically switches off the mouse’ pup-killer disposition from day 18 to day 22 after its last ejaculation. This period happens to be the gestation time for female mice. So pups the male encounters during this period have a high probability of being its own pups and have a chance to escape before the pup-killer instinct returns.¹⁴ For all the world it looks like male mice show a complicated strategy requiring considerable genealogical knowledge, when in fact the behavior is hard-wired, and the gene which produces it is a quick and dirty solution to hard problem.

Similarly, TFT behavior will be indistinguishable from behavior generated by some much simpler genetically encoded dispositions. In particular, a gene for unconditional kin-altruism will produce behavior indistinguishable from TFT strategy in iterated prisoner’s dilemma circumstances, when all players are close kin. That there is a gene for kin-altruism, or any preferential treatment of kin, or for that matter some quick and dirty substitute for it (a gene for altruism towards anything that secrete a certain odor, for example) among the mammals is a pretty safe bet. But if there is a “gene for” kin-altruism or even any quick and dirty available substitute for it, there is also some considerable evidence that such a gene either never figured among the genotype of primates, or that if it did, it made no significant contribution to cooperation among them. This is due to the fact that long before our last common ancestor with the chimps (about 5 million years ago), all the primates had ceased to live in groups in which

kin-altruism would be selected for. Or at least that is what a comparative analysis of our closest primate relatives suggests. The social structure of almost all extant ape groups reflects female (and often also male) dispersal at puberty, high uncertainty of paternity (except for gibbons), an abundance of weak social ties and a lack of strong ones. Paleontology reveals that the number of ape species underwent a sharp decline about 18 million years ago, while monkey species proliferated. If this was the result of competitive exclusion of apes towards marginal tree-limb niches, it would explain many of their and human anatomical similarities. Unlike humans, chimps and gorillas remained in these restricted niches to the present. Humans, and chimps are highly individualistic, mobile across wide areas, self-reliant and independent. By contrast the monkey species reflect matrifocal social networks that would strongly encourage the selection of kin-altruism.¹⁵ At a minimum the pattern of sociality we and the other primates inherited from our last common ancestor makes it highly probable that cooperation among us is not written in the genes, even imperfectly, approximately, by some quick and dirty exploitation of an already available gene for some form of kin-altruism, still less by direct natural selection for the disposition to TFT

All in all, it seems more reasonable to assume that TFT and other cooperative behaviors are the results of the collaboration of a number of different behavioral dispositions all simply reinforced by their environments, i.e. dispositions ontogenetically selected for, though not phylogenetically selected for. If so, it would be worthwhile seeking a package of genes which produce the dispositions and capacities that are individually (non-trivially) necessary but not jointly sufficient for these sorts of cooperative behavior. (A gene is non-trivially necessary for a phenotype roughly if it is not also necessary for a large number of other traits, including respiration, metabolism, reproduction, survival, etc.) In this scenario a great deal of the burden of explaining the exact shape of cooperation is shouldered by the environment in which hominids must have survived for hundreds of thousands of years. And the degree to which our genomes are explanatorily relevant to cooperative dispositions will turn on whether the genes that subserve cooperative behavior were selected for owing to the fact that they make overwhelmingly likely hitting on TFT or one of the other strategies and make it easy to learn these strategies from others. If they merely make it easy to discover and learning any complex behavior, the notion that cooperation is an evolutionary adaptation naturally selected for will be undercut. The former case on the contrary would go some way towards vindicating evolutionary scenarios for cooperation.

Exponents of an evolutionary account of cooperation will favor an account of the matter according to which dispositions that specifically subserve cooperation are selected for owing to the pay-off cooperation provides for fitness. Indeed, some will hold that dispositions and capacities useful for other purposes beside fostering sociality, capacities like memory, speech,

and reasoning, have been selected for owing to their contribution to solving the design-problem presented by iterated prisoner's dilemmas, and other competitive games. Suppose the genes for a suite of widely useful capacities such as speech, memory, and a theory of mind, were all selected for because together, they made an agent's seeing and choosing the cooperative strategy a "no-brainer" obvious move in appropriate circumstances. We might be tempted to say that together the sequences do constitute "a gene for cooperation".

Which of these possibilities obtains is something gene sequencing may illuminate. We suppose that the genes needed for the evolution of cooperation will include those which subserve general capacities such as memory, reasoning, speech, and ones specific to cooperation such as the emotions like anger, shame, resentment, guilt, love, jealousy, and revenge. One of the ways to begin to identify the relevant phenotypes and genotypes on which cooperative behavior supervenes is to examine hereditary and genetic defects in humans.

For an example of a defect more directly tied to the specific dispositions involved in cooperation consider high function autism and Asperger's syndrome, which prevent normal cooperative behavior, are associated with anatomical and neurological abnormalities in the brain, and (in the case of autism at least) have a substantial hereditary component. There is reason to suppose that autism results from the interactive effects of at least three micro-rearrangements on genes some of which produce a serotonin transporter. These genes are probably located on Chromosomes 7 and 15, and they are implicated in some other rare genetically caused retardation. We know that normal children develop a "theory of mind"—the attribution of intentional states to others between the ages of two and four, and there has been some empirical investigation and an good deal of debate about whether the primates show a similar capacity. If the capacity to treat others as having intentional states is one lost in autism, then we are on the way to locating the genes that are either non-trivially necessary or perhaps even sufficient for the capacity in humans.

For another example, it has recently been shown that certain significant defects in speech assort in genetically familiar patterns, and positional cloning has enabled geneticists to locate the particular genes responsible for the defect, and *mutatis mutandis*, the genes whose normal function is necessary for normal speech.¹⁶ It occurred to the researchers making the discovery of the "gene for" a hereditary speech disorders, almost immediately, that genomic comparisons to chimps could reveal important information about the evolution of language-competence, a vital necessity for the emergence of complex cooperative dispositions. We know that chimps and gorillas have show substantial communicative behavior in domestication, and ethological study of vervet monkeys continues to increase our knowledge of their lexicon well beyond the well known calls for eagle, leopard and snake. What they appear to lack is syntactic skills, and these skills are genetically hard wired in us is suggested not just by Chomsky's speculations but by

Derrick Bickerton's studies of the transition from pidgins to Creoles.¹⁷

The sorts of sequence data available from mitochondrial DNA, or for that matter the Y-chromosome sequence data, is however, completely inadequate to test hypotheses about human/primate genetic differences and similarities. As is well known, to begin with the sequence similarity between *Homo sapiens* and Chimpanzees is something over 98 %, and the size of the genomes is immensely greater than that of the mitochondria their cells bear. Moreover, approximately 95 % of the sequences in both genomes are "junk" DNA which does not code for any gene products and whose function if any is unknown. Presumably, the differences between *Homo sapiens* and Chimps is to be found among the 5 % of coding sequences, in the regulatory sequences which control the expression of structural genes identical between humans and chimps. But where these coding sequences are across the 3 billion base pairs, and how they differ is the issue. Something else is needed as a source of evidence, and a way of analyzing it.

However, it is at this point that the next generation of genomic data comes into play. For even in the last five years, genomics has moved from comparisons of relatively small amount of extra nuclear DNA to the comparison of entire chromosomes employing automated "gene-chip" or "microarray" technology. A gene chip is a small piece of glass on which a huge number of gene sequences can be arrayed. These will preferentially bind to sequences that are closely similar to themselves, when a sample of such sequences are washed over the chip. Those sequences which have bound a similar sequence from the sample can be detected. If the sequences on the chip are known, it is trivial to read off the differences between genes originally placed on the chip and those of the sample. So, once we have located some or all of the genes on, say, a human chromosome, we can array sequences from these genes on a chip, wash the sample with DNA sequences from the homologous Chimpanzee chromosome, and read off the sequence differences. If enough is known about how the sequences on the human chromosome realize particular genes, we can identify the presence or absence of the same genes in Chimps, differences in their structure, number, and location on the chromosome. If enough is known about the biosynthetic pathways into which these genes enter, we are in position to identify the genetic bases of differences in anatomy and behavior between *Homo sapiens* and Chimp.

Such a program of research has already begun to be carried out for the human chromosome 21 and the homologous Chimpanzee chromosome 22. Human chromosome 21 is the shortest and was the second to be fully sequenced (by a German-Japanese consortium). It contains only 225 genes (of which 98 are identified only through computer gene prediction) within 33.5 million base pairs, and is of particular interest owing to its duplication in Trisomy 21 (Down's syndrome), and the role of genes it carries in Alzheimer's disease, some forms of epilepsy, auto immune disorders, a form of manic psychosis, and deafness. Within the last month (March 2003), a microarray comparison between the Human chromosome 21 and the

homologous Chimp chromosome 22 has been undertaken.¹⁸ What this work so far shows is that here are not just individual polynucleotide differences, but substantial genomic rearrangements—both insertions and deletions—between the two genomes, that these rearrangements account for about 50 % of the total sequence differences between chimps and humans on these chromosomes; that the deletions at least appear to be random in origin, and both deletions and insertions are randomly distributed across the chromosomes (except for one 250 kilobase region).

Let's apply these evidential breakthroughs to the study of the evolution of sociality. Assume that cooperative behavior does not result from a single genetically coded behavioral disposition, but rather that it is taught, learned and culturally selected for, once it appears. However, there is a suite of hereditary phenotypic dispositions on which it depends. What will these look like? Most skeptics about “genetic determinism” will claim that these phenotypes are likely to be at most anatomical structures and in many cases mediate and immediate protein products of regulatory and structural genes at best causally necessary for the behavior, not sufficient for it, even in relatively restricted circumstances. If these skeptics are right, genomics can do little for our inquiry, nor much for human behavioral biology, evolutionary psychology, biological anthropology or the rest of social science.

But whether they are right or whether there are gene sequences sufficient in normal environmental circumstances for complex behavior is of course an empirical question, and on it some interesting light has already been shed by genomic studies of other model systems. In mice normal nurturant behavior includes creating nests, cleaning pups, retrieving them to the nest, crouching over them to provide warmth and nourishment. Nurturance in mice reflects a capacity normally acquired by males and females after exposure to similar retrieval, cleaning, warming and feeding behavior in other mice. When the mouse genome is subjected to a “knock-out mutation” of *FosB*, a gene which codes for a 4.5 kilobase messenger RNA, the result is that mothers ignore their pups, do not gather them, retrieve them, warm, them or feed them, although they do approach and sniff them. This behavior remains unchanged through several pregnancies and in the presence of appropriate modeling behavior by wild type (“normal”) maternal mice. So we can exclude learning and experience as causes of infant nurturance. Indeed, wild type mice which have never been pregnant will show nurturant behavior when exposed to new born pups, while *FosB* mutant mice that have never been pregnant show the same non-nurturance defect. Nor is the defect even limited to females: wild type males will nurture and *FosB* mutant males will not. When subject to tests for cognitive, olfactory, or hypothalamic related abnormalities (hypothalamus defects are known to influence nurturance), the *FosB* mutant mice show no behavioral deficits or abnormalities. Studies of *FosB* gene expression in normal mice brains have led researchers to conclude that exposure to pups triggers the *FosB* gene in cells of the preoptic

area of the hypothalamus to produce a protein which appears critical to nurturing. The FosB protein is expressed elsewhere in the brain and may have functions additional to its role in nurturance. However, research has excluded many more basic, and non-specific roles for *FosB*—in olfaction, general cognition, perception, learning, which might lead to defects in nurturance (and other capacities as well).¹⁹

It is hard to escape the conclusion that this is a “gene for” nurturing in mice. Why should there not be genes for similarly complex behavior in other mammals, up to and including chimps and humans. Unfortunately, the best way to tell whether there are such genes is simply not open to us. The regulations under which both institutional review boards for human subjects, and animal care committees operate, make it unlikely that the protocols under which knock-out and gene-insertion experiments proceed, will ever be approved for humans or chimps. Nevertheless, it is worth considering what such experiments could show. Take for example, the “grammar gene”, as Pinker calls it.²⁰ described above: many of the affected humans show normal intelligence, “they have trouble identifying basic speech sounds, understanding sentences, judging grammaticality and other language skills.” and a genetic marker at a locus called the SPCH1 segment of chromosome 7, at a specific regulatory gene *FOXP2*, disrupted in their case by a translocation. The translocation results in the substitution of guanine by adenine in the nucleic acid sequence, and arginine by histidine in the gene product. Two forbidden experiments immediately suggest themselves: locate the homolog of *FOXP2* in the chimp (it must be there, since it is already known to be expressed in the developing mouse cerebral cortex), and either insert a normal human *FOXP2* or some portion of it so that the same regulatory product is produced in the chimp. It is well known that the sorts of regimes already employed to test linguistic competence among chimps reveal a lack of grammaticality in their performance which is required for complex schemes of cooperative behavior. Will the gene-insertion make a difference either to the individual chimp’s language learning capacity or the enhancement of complex communication among chimps? A second experiment, even less permissible, is to locate the homologous gene in chimps and insert it in human infants, and to follow development to determine what sorts of linguistic deficits result.

The same sorts of experiments will repeatedly suggest themselves as positional cloning identifies more and more specific DNA sequences implicated in the blockage of development and exercise of human capacities and dispositions we suspect are necessary for complex behavior such as TFT (playing tit for tat in iterated prisoner’s dilemmas). Beyond the limitations imposed on experimentation with human and primate subjects, the real problem with this strategy seems to be the sheer number of genes and gene products that are implicated in these complex dispositions. The gene for nurturance in the mouse is more likely the exception than the rule, among mammals. But even if it is common, the number of gene products involved in

complex behavior may well be beyond current computational limits. If upwards of 60 % of the coding regions of the genome are devoted to the production of proteins and enzymes expressed in the brain, then even to identify a significant portion of the “genes for” something as complicated as cooperation will be a vast undertaking. But this fact does not detract from the in principle possibility of employing gene sequencing to illuminate the evolution of cooperation.

The gene chip, applied to gene expression in heritable human behavioral deficits, and to chimpanzee brain function, enables us to begin to identify the genes which are necessary for the sort of complex behavior that constitutes social cooperation, and eventually to decide whether they are also sufficient for it in normal environmental circumstances. The comparison will have to be three way, including gene expression in the normally functioning brain, hereditarily malfunction brains, and chimp brains. Begin by using a microarray to identify the chromosomal locations of gene sequence differences between the normal and large range of humans with hereditary neurological malfunctions. Given the location on the normal chromosome of this candidate, use the same gene chip method to establish chromosomal locations of the homologous sequences, if any, in chimps. If the sequence is quite similar in size, copy number, relative location, etc., assume that it is not among those interestingly necessary for a distinctive human behavioral disposition. If the gene is absent, different in number, location, introns, etc. in the chimp, then it is a candidate for being interestingly necessary for distinctive human dispositions.

It will take a very long time to identify all the genes non-trivially necessary for complex cooperative behavior, and to learn what they do: the biosynthetic pathways from them to behavior. But it will not take as long to simply provide a list of locations, alternate sequences, introns, copy numbers, for these genes without details about their biosynthetic consequences, macromolecular, anatomical, and ultimately combined behavioral consequences. At this point it should become possible to construct a number of macromolecular scenarios for how linkages, cross-over events, mutation, gene-duplications and translocations, and other events, were selected for, to produced these nucleotide sequences from the common ancestor of humans and chimps.. That such genetic alterations which hold the key to our distinctive capacities and dispositions were selected for, or at least were selected because they were carried along by some other gene sequences, is reflected in the differential adaptation of the primate species. Despite the tiny quantitative nucleotide difference between us, the chimps and the gorillas, they are both relatively unsuccessful species, still restricted to a narrow and endangered niche geographically close to the one we started out in, while we bestride the globe. The sequence differences between our ancestors and theirs must have been selected for in the environments we shared.

Once the list of locations and sequences for genes without a known function, but nevertheless implicated in distinctively human behavior, are given, the methods employed to date mitochondrial and Y-chromosome sequences can be employed to give the order of

emergence and perhaps even the ages of these genes. The comparison of human chromosome 21 and chimp chromosome 22 provides evidence that the genetic differences include rearrangements and duplications, and thus there is reason to think that within homologous sequences there will be the single nucleotide polymorphisms—neutral point mutations-- that can provide a molecular clock to date the emergence of each of these distinctively human gene before we know much more about them than that they produce a protein that functions in the brain cells. With the right hominid fossils—Neanderthal, and older for that matter-- and a great deal of good fortune, PCR amplifications can add important data about chronologies or dates of first appearance to our evidential base. (And those sequences, if any, which are entirely missing or diverge beyond random point mutations, may tell us even more, once we have annotated the genes they figure in) .

What will the chronology so established show us? It depends on what the chronology looks like. The alternatives are obvious: each gene interestingly necessary for distinctively human dispositions emerges at a different date, all the genes emerge at roughly the same date, different sub-sets emerge together. Any one of these outcomes will drive a significant research program in the evolution of human cooperation.

A) None of the gene sequences uniquely expressed in human brains are of the same age.. Then probably each emerged as the solution to a separate design problem and their joint result, distinctive human cooperative behavior, cannot be mainly attributable to natural selection, but rather to cultural selection. In this case it will be worth while investing in annotating these genes roughly in the order of their age, in order to shed light on stages of hominid cognitive evolution, by determining older genes' roles in cognitive neuroanatomy and behavior, employing reverse engineering to determine what design problem if any each solves, and what design problems it may make possible for later genes to solve. If ancient DNA from other hominid lines can be recovered in sufficient quantities, then we will learn something about the evolutionary distance between our branch of the hominid tree and other branches, and from the absence in them of some genes that are expressed in our cognitive anatomy and behavior perhaps what design problems their failure to solve led to their extinction.

C) The gene sequences can be divided on or more sets with roughly the same age.

This result would be extremely significant. It would be empirical evidence for the hypotheses that the genes of equal age were selected for contributing to the solution of the same set of design problems. Accordingly, identifying the function of genes in a package of the same age, could provide evidence to test hypotheses about what design problems that humans and/or their hominid ancestors faced and solved.

Assume that working from chimpanzee DNA hereditary human defects, employing positional cloning and microarray technology, we have identified and located the group of genes

for linguistic communication, or grammaticality, and the group of genes for a theory of mind, and the group responsible for the emotions crucial to commitment in iterated strategic games, and the group of genes for memory and reidentification of fellow-players in competitive and cooperative games, or simply assume that we have identified many of these gene families and some of the genes in each family. If these genes and groups of genes are about equally old, it is reasonable to believe that they were all selected because they were all involved in solving a design problem, or a small number of connected design problems. If we can correlate the concerted emergence of these genes with what we know about environmental changes, paleo-archeology and demography, the conclusion that they were selected for solving one or a small number of connected problems is further strengthened. It is pretty clear that such evidence would strongly support the hypothesis that the emergence of human cooperation was, if not a forced move, a neat trick with a Darwinian explanation.²¹

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Notes

¹ [Cf Marshall Shalins, The Uses and Abuses of Biology, U.; Michigan Press, 1974] The problem of how even to reconcile the theory of natural selection with the possibility of cooperative institutions was so grave that E.O. Wilson insisted that Camus was wrong: it was not suicide that is the only philosophical question, but rather altruism [Sociobiology: the New Synthesis, p. 3].

² Frank, Passion within Reason

³ “The Panglossian Paradigm and the Spandrels of San Marco”

⁴ [for an introduction to the African “Eve” hypothesis and supporting data see R. Boyd and J. Silk, How Humans Evolved, New York, Norton, 2000, pp. 477-483, Hedges, B., “A start for population genomics”, Nature, 408 (2000): 652-653, and articles there cited, especially Stoneking, M., and Soodyall, “Human evolution and the mitochondrial gene”, Current Opinion in Genomics and Development, 6 (19996): 731-736. For Y-chromosome sequence confirmation and amplification see Renfrew, C., Foster, P., and Hurles, M., “ The past within us”, Nature

Genetics, 36 (2000): 253-254 and papers there cited, Stumpf, M., Goldstein, D., “Genealogical and evolutionary inference with the human Y-chromosome”, Science, 291 (2001): 1738-1742

⁵ For an account of the natural selection of skin colors see Jablonski, N.G. and Chaplin, G. (In press.) “The evolution of human skin coloration.” J. Hum. Evol.

⁶ See R.L. Cann, “Genetic clues to dispersal in the human populations: retracing the past from the present”, Science 291 (2001): 1742-1748.]

⁷ Boyd and Silk, 2000, pp. 484-485, Science, 11 July, 1997, Gibbons, A., “The riddle of co-existence”, Science, 291 (2001): 1725-1729.

⁸ Key et. Al., Science 292 (2001); 1151-1153.

⁹ Richards, M., Mccaulay, V., et al. “Tracing European flounder lineages in the near eastern mtDNA pool”, American Journal of Human Genetics, 67 (2000): 1251-1276.

¹⁰ Gibbons, A., “The peopling of the Pacific”, Science, 291 (2001): 1735-1737, and papers cited therein.

¹¹ “Ancient DNA in charred wheats: Taxonomic identification of mixed and single grains.” Brown, Terence A.; Allaby, Robin G.; Sallares, Robert; Jones, Glynnis; Ancient Biomolecules, May98, Vol. 2 Issue 2/3, p185-184; “Patterns of genetic diversity in extant and extinct cattle populations: Evidence from sequence analysis of mitochondrial coding regions”, Turner(a), Catherine L.; Grant(b,*), Annie; Bailey(c), Jill E; Dover(b), Gabriel A.; Barker(a), Graeme W. W.; Ancient Biomolecules, May98, Vol. 2 Issue 2/3, p235-250]

¹² Paabo, S., “Human evolution”, TCB 9(1999): m13-m16, Hoss, M., Nature, 404 (2000): 453-454..

¹³ For an introduction to these QTL studies see R. Plomin, et. Al., Behavioral Genetics, Forth Edition, New York, Worth, 2001.

¹⁴ G. Perrigo, et. al., “A unique timing system prevents male mice from harming their own offspring”, Animal Behavior, 39 (1990): 535-539

¹⁵ Maryanski and Turner, Social Cage, Stanford UP, 1992

¹⁶ Cecilia S. L. Lai, Simon E. Fisher, Jane A. Hurst, Faraneh Vargha-khadem, and Anthony P. Monaco, “A forkhead-domain gene is mutated in a severe speech and language disorder” Nature 413, 519-523 (4 October 2001).

¹⁷ Bickerton, Derek, ‘How protolanguage became language’, in Chris Knight, James R. Hurford and Michael Studdert-Kennedy (eds), The Evolutionary Emergence of Language Cambridge: Cambridge University Press, 1998.

¹⁸ Frazer, K., Chen, X., Hinds, D., Krishna Pant, P.V., Patil, N., and Cox, D., “Genomic DNA insertions and deletions occur frequently between humans and nonhuman primates”, Genome Research, 13: 341-346, March 2003. See also Locke, D.P., Segraves, R., Carbone, L., Archidiacono, D., Pinkel, D., Eicler, E, “Large-scale variation among human and great ape genomes determined by array-comparative genomic hybridization”, Genome Research, 13: 347-357, March 2003

¹⁹ Jennifer R. Brown, et. al. “A defect in nurturing in mice lacking the immediate early Gene *fosB* Cell, Vol. 86, 297-309, July 26, 1996]

²⁰ Pinker, S. “Talk of genes and vice versa”, Nature, 419 (2001):465-66] identified by Fisher SE, Vargha-Khadem F, Watkins KE, Monaco AP and Pembrey ME (1998) Localisation of a gene implicated in a severe speech and language disorder. Nature Genet. 18:168-170, et al,

²¹ The distinction is first drawn in D. Dennett, Darwin’s Dangerous Idea, New York, Simon and Schuster, 1995.