Genetics and Social Behavior

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Abstract

We focus on the effects of gene differences on social and behavioral differences among individuals and among larger groups of individuals. Many specific genetic markers are known that influence aspects of personality and behavior. The focus on single genes and groups of genes is giving way to quantitative genetics, the statistical study of transmission of characteristics viewed as the outcome of the effects of very large numbers of genes. While traditional social science largely ignores the effects of genetically transmitted influences, the subject persists and grows in importance. Classical quantitative genetic methods may give much insight into human behavioral diversity and they provide the "right" way to measure and assess variation in rates of threshold traits. We discuss examples, trends, and possibilities for the incorporation of genetic data and models in the social and behavioral sciences without advocating major changes in practice.

INTRODUCTION

There are two distinct ways we could approach the task of reviewing genetics and human social behavior. One is to review aspects that are genetic in the sense of hard-wired in the human genome and universal. For example, we all learned spoken language early in our lives and most agree that the trait "learn to speak the language(s) in your social environment" is such a universal, while the specific language(s) we learned were entirely dependent on our rearing environment. In spite of the existence of people who never learned to speak, we do not hesitate to regard learning speech as "genetic." However, things are not so simple: speech does not have to be taught while literacy does. We are all literate, but many of us struggled to learn, say, calculus, in a way we never struggled with literacy. This would lead to a consideration of the nature of cognitive modules, a lively area of current research.

Another way to proceed is to consider how genetic differences among people or among groups of people lead to or have been generated by individual or group differences in social behavior. Are individual differences in social behavior determined by gene differences? Do group level gene differences

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modulate group differences in social behavior? A century ago, the response of most educated people would have been "of course" to questions such as these, whereas today, the response of many educated people and scientists is "of course not." We suggest that this change has gone too far, motivated in many cases by political undercurrents, and that an important task for social science it to understand and investigate gene differences.

In the past century, many anthropologists were interested in temperament, for example, and culture and personality was a prominent subfield. It is striking that in all that literature, the possibility of such things reflecting gene differences was never on the table. Roughly between 1900 and 1950, there had emerged a new cluster of academic disciplines, social science. The vision was that this would be a third kind of knowledge, somewhere between the humanities and the sciences, a branch that would stand between and perhaps unite human knowledge. The optimism and ambition of practitioners of the new disciplines were great and they were contagious so, for example, Yale University created the Institute of Human Relations in the 1930, "directly concerned with the problems of man's individual and group conduct" (Morawski, 1986). Harvard University created what was practically a whole new division of the university with its Department of Social Relations in 1946, a hybrid of sociology, anthropology, and psychology.

A simple, indeed simplistic, statement of the origin of social science in its early years was that it insisted on the separation of the body and soul, the soul being the locus of social behavior and personality. If social science was to be the scientific study of the human soul, it was necessary that there must be a universal human soul separate from the human body. The result was a denial of human biological diversity's relevance to social behavior and, in its strong form in Anthropology due to Boas and his followers, a denial of ongoing evolution in humans of that soul. There was in universities something like a truce between the biological sciences and the social sciences so that even the great biologist Ernst Mayr could say that "We cannot escape the conclusion that man's evolution toward manness suddenly came to a halt" (Mayr, 1963).

FOUNDATIONAL RESEARCH

There have always been cracks in the strong social science stance but in the past decade or so it has been noticeably weakening. A prominent example has been psychiatry converting essentially to biological psychiatry. Advances in biochemistry such as radioimmunoassay, allowing assays of hormones and other chemicals at reasonable cost, led to a lively literature on the effects of hormones and other chemicals on behavior.

Much of the study of genetic influences on behavior and personality relied for decades on the similarity between genetic relatives, especially identical

and fraternal twins. Twins separated at birth were of special interest because it was presumed that they did not share a common environment and their resemblance reflected shared genes. A not unreasonable criticism of such studies was that (i) they shared a common prenatal uterine environment and (ii) foster families were not random families but were often chosen to match the adoptees in terms of race, social class, and so on.

In the past several decades, new technology from genomics has allowed genotyping of hundreds of thousands to millions of genetic markers in humans, directly measuring similarity (i.e., kinship) between individuals. This means, for example, we can look at similarities between third cousins and compare those similarities to those between fifth cousins, cryptic cousins as the individuals are complete strangers to each other. The possible errors from looking at close genealogical relatives in quantitative genetic studies are now bypassed (Purcell *et al*., 2009; Visscher, 2010), as the new methods for estimating heritability have replicated the old estimates. An old nagging source of doubt and controversy in behavior genetic studies is gone.

CUTTING EDGE RESEARCH

Single Locus Traits

Much of human behavior is universal and adaptive, or used to be in past environments. We eat when hungry, fear spiders and snakes, love our children. Individuals without these behaviors failed to reproduce, or to reproduce at rates comparable to those with the behaviors. Such patterns can be thought of as strategies, patterns or rules of conduct that, on average, led to reproductive success in the human past. However, many aspects of our behavior vary. The differences show up early in life and do not seem to be affected much by parental rearing style.

What are the sources of these differences between individuals? Genetic differences must contribute, as personality is heritable, in humans as in other species (Kendler & Greenspan, 2006). To say that some trait is heritable is to say that it is genetically transmissable to offspring. More precisely, heritability is a fraction between 0 and 1 that specifies the amount of trait diversity in a population that reflects gene differences.

Environmental insults contribute as well, such as neurotropic viruses, toxins, and other influences. However, here we are going to talk genetics. Genetic influences on behavior fall into two fundamental categories: those that are the product of natural selection, and those that are not.

To what extent is existing variation in human behavior adaptive? For example, some people are scrupulous and hyper-moral while, at the other extreme, some people are without apparent conscience. (We call them

sociopaths.) There is apparently a continuum from one extreme to the other, with most people falling somewhere between the two extremes. Why should not there be a single best strategy, that is, why is not everyone the same?

Often there is a single optimum, but not always. It depends on whether the payoff in terms of fitness of a particular course of action is frequency-dependent—in other words, whether it depends on the actions of other individuals. Running from a forest fire pays off whether anyone else does or not, but the payoff of running for tribal chief decreases as the number of candidates increases. If everyone is passive (a "dove"), aggressive individuals (hawks) prosper, but as hawks becomes more common, they increasingly run into and fight other hawks, so their payoff decreases. If the cost of fighting among hawks is high enough, the equilibrium solution is a mixed state consisting of both hawks and doves (Smith & Price, 1973). Thus there is no single best solution, no single optimal behavior. This is often the case with social interactions. It is worth remembering that hawks do not benefit the species as a whole: The species would do better if everyone just got along. However, when the strategy benefits individual hawks and the genes they carry, they increase in frequency.

A related issue is whether adaptive variation in behavior and personality is fixed or flexible, heritable or not. Sometimes an individual can pick one of several possible life strategies based on exterior clues. If a female bee larva is fed royal jelly, she becomes a queen; otherwise, she becomes a sterile worker. The capability to assume those different roles is adaptive and presumably a result of natural selection, but it is not noticeably heritable, because *all* female bees have it. In other cases, like those pesky fire ants that exhibit single-queen and Los Angeles-style multiqueen colonies, the two morphs are determined by the two alleles of a single supergene (Wang *et al.*, 2013), and the behavior difference is completely heritable. In the former case of queens and workers the two outcomes reflect environment effects with no contribution from gene differences while in the latter case of colony structure the outcomes reflect simple gene differences with no contribution from the environment.

As we are arguably a lot smarter than ants or bees, one might think that most adaptive personality variation in humans would be learned (a response to exterior cues) rather than heritable. Certainly some is, but much variation looks heritable. People do not seem to learn to be aggressive or shy—they just are, and in those tendencies resemble their biological parents. There are models that may explain this. One is that jacks of all trades are masters of none: If you play the same role all the time, you will be better at it than someone who keeps switching personalities. It could be the case that such switching is physiologically difficult and/or expensive. Moreover, in at least some cases, being predictable has social value. Someone who is known to be implacably aggressive will always win at chicken (Kahn, Aligică, & Weinstein, 2009).

Being known as the sort of guy who would rush into a burning building to save ugly strangers may pay off, even though actually running into that blaze does not.

In addition, if a particular role or personality type only became viable relatively recently—say, 10,000 years ago, in the early Neolthic—a mutation that induces that personality may have become fairly common, but would not yet be part of a precise and flexible system. The required modifier genes that would turn those tendencies on when they pay and off when they do not, would take longer to evolve. Evolution is ongoing, so many new adaptations are likely to be imperfect and incomplete.

John Tooby and Leda Cosmides, leading founders of evolutionary psychology, have argued strongly against the possibility of heritable, adaptive behavioral variation in humans (Tooby & Cosmides, 1990). Their arguments imply that no such thing should exist in any species, but we have found genetic morphs in lizards, birds, crustaceans, ants, and butterflies. They are widespread in nature. However, one of their arguments is especially interesting: they argue that adaptations, including behavioral adaptations, are usually generated by complex, coadapted sets of genes (true), and that such gene complexes would be broken up by sexual reproduction (also true): Even if the dad had a set of genes that made him a natural blacksmith or tap dancer, his kids would never inherit that whole set and the talent should disappear.

They are on to something with this argument, but nature seems to have found solutions. In some cases, a set of genes that determine the alternative phenotype are arranged as a supergene, a group of genes that are physically close and strongly linked. That keeps the gene complex from being broken up. In other cases, the alternate forms are determined by a single gene that acts as a switch. Different alleles of that gene specify different morphs. This kind of mechanism is also largely unaffected by recombination.

Perhaps the heritable variant is in some way a simple trait. For example, what if simply losing a particular complex adaptation was adaptive when rare? It is easy to see that a complex behavioral adaptation could be stopped cold by a single mutation. Alternatively, for that matter, what if greatly intensifying a particular behavior or drive—turning up the volume knob—was adaptive when rare? It is possible to imagine a simple mutation that turns up the volume in some way? Surely. In addition, of course, such simple initial changes can be gradually refined by natural selection.

We know of many clear examples of distinct behavioral morphs or strategies in other species. We mentioned the two different kinds of fire-ant society. Many of the examples are of male morphs with different reproductive strategies. One of the most interesting and well-known example is *Uta stansburiana*, a species of lizard studied by Barry Sinervo (Sinervo & Lively, 1996). The

species has three male morphs with distinct color patterns: orange, yellow, and blue. Orange males are large, aggressive, and control a territory with several females. They dominate blue males, but yellow males, which mimic females, often cuckold them. Blue males have a smaller territory with one female, which they can effectively defend against yellow males. Orange beats blue, blue beats yellow, and yellow beats orange. It is scissors-paper-rock.

Within-group adaptive behavioral variation is possible, as we know of many examples in other species. Behavioral differences between geographically separated populations of the same species are also possible, as selection pressures often vary with location. That means that genetically induced adaptive behavioral variation could exist in humans—but does it?

The usual measures of personality all show significant heritability (Turkheimer, 2011), which is consistent with adaptive personality variation but also with nonadaptive genetic influences, such as mutational load. For example, aggressive individuals could be present in a population as evolved players of a "hawk" strategy in their interpersonal relations, or they could simply be unfortunate bearers of deleterious genetic mutations leading to their aggressive behavior. The case that would be clearest, easiest to prove, would be one in which different variants of a single gene have a significant influence on behavior. If such variants existed, they would almost certainly be the product of adaptive evolution. Mutational pressure would create a very different picture, with rare deleterious mutations of many different genes, rather than two or three common variants of a single locus.

The most promising possible case of adaptive genetic variation in humans is in MAOA, an enzyme that degrades several neutrotransmitters. The gene, located on the X chromosome, contains a 30-base sequence that is repeated different numbers of times in different MAOA variants—2R, 3R, 3.5R, 4R, and 5R. These repeats are in a regulatory region and affect gene activity.

We know that complete loss of MAOA activity has a strong impact on human behavior. A null mutation of MAOA has been seen in large Dutch kindred (Brunner, Nelen, Breakefield, Ropers, & Van Oost, 1993). Males with this mutation show impulsive aggressiveness and mild mental retardation. Low levels of the gene products (rather than any particular variant) are associated with trait aggression in men (Alia-Klein *et al.*, 2008).

In mouse models, inactivation of MAOA causes increased aggressiveness (Cases *et al.*, 1995; Scott, Bortolato, Chen, & Shih, 2008). In rhesus monkeys, there is an association between low activity MAOA alleles that is dependent on early environment. There seems to a similar interaction effects in humans: low activity MAOA alleles (the 3R and 2R variants) seem to increase vulnerability to environmental stresses such as abuse in childhood. In this case, the environmental effect of being abused as a child, like the jelly to the female bee larva, leads to behavior change in adulthood only if a specific genetic variant

is present. With this level of complexity, spreading of a trait as learned or not loses its meaning.

Despite the special difficulties of performing these investigations in humans, it appears there really are genetically induced behavioral variants that are the product of selection. MAOA is not the only locus for which we have evidence of this, but it is probably the best-understood, and makes the point.

There are important aspects of human behavioral variation that are almost certainly maladaptive, such as mental retardation and mental illness. They too are heritable, and to a significant extent genetic in origin. That is not to say that environmental insults and pathogens do not play a role. Prenatal starvation doubled the incidence of schizophrenia in the cohorts affected by the Dutch famine of 1944 and the Chinese famine associated with the Great Leap Forward (St Clair *et al*., 2005; Susser & Lin, 1992). Neurosyphilis once accounted for about half of the patients in psychiatric hospitals (Barondes, 1990). However, we have reduced the impact of syphilis and other pathogens over the past century, as well as famine. Genetic causes have not diminished, and therefore account for a larger fraction of cases than they once did.

Personality variations generally do not drastically reduce reproductive fitness, but mental illness and mental retardation do. For example, in schizophrenia, fitness is drastically lowered in men (about 25% of normal) and significantly lowered in women as well, about 50% of normal (Bassett, Bury, Hodgkinson, & Honer, 1996). As schizophrenia is quite heritable (Tsuang, 2000) there is an apparent paradox: how can schizophrenia (and other forms of mental illness) persist over time at moderately high frequency, 0.5–1%? (Saha, Chant, Welham, & McGrath, 2005).

Increasingly, it looks as if the cause is ongoing mutation. Our understanding of mutation in humans and its negative consequences for human health is developing rapidly, because of advances in sequencing technology. It is now possible to identify mutations by high accuracy whole-genome sequencing of family triads: if you see a sequence in the child that does not exist in his parents, it is a new mutation. (Of course it could also be parental assignment error, but that is easily detected because there would be thousands of mismatches between the parental genes and those in the child.)

There are a number of recent reports that have found evidence for de novo mutations as an important cause of schizophrenia and autism (Awadalla *et al*., 2010; Girard *et al*., 2011). A high rate of ongoing mutations also explains why a significant fraction of schizophrenia cases are sporadic: they appear in families that do not have previous cases of schizophrenia (Xu *et al*., 2011). These techniques have also confirmed that most mutations originate in the father, and that the mutation rate increases linearly with post-pubertal father's age.

Many problems are more common in the children of older fathers, but disorders that result from impaired brain function, such as autism, schizophrenia, and reduced intelligence, are particularly common (Kondrashov, 2012). This seems to be a consequence of the great complexity of brain development and function, making use of the majority of all genes.

Schizophrenia must be made up of many different mutational disorders. At the same time, a given mutation often seems to increase risk for several different mental disorders. (Cross-Disorder Group of the Psychiatric Genomes Consortium, 2013). Clearly, progress in this area has been hampered by simultaneously lumping together causally unrelated syndromes and splitting a syndrome with a single cause into several different categories.

On the other hand, if this new picture is correct, it may suggest new and effective approaches for fighting mental illness. In the short run, finding ways to reduce the mutation rate might pay off, especially if any factors other than paternal age are found to materially influence that rate. In the longer run, gene therapy may offer hope.

Quantitative Traits

Many regard the origin of evolutionary biology as a science to be the publication by R.A. Fisher (1918) of a paper titled "The correlation between relatives under the supposition of Mendelian inheritance." In it he showed that if a quantitative trait, such as height or blood pressure that is measured on a continuous scale, was determined by an environmental effect and a large number of genes, each of small effect, the trait should follow a Normal or Gaussian distribution in the population. The model predicts correlations between relatives of different degrees. Before this publication there was widespread doubt about Darwin's theory and conflict between two traditions—Mendelians, who sought to understand variation using Mendel's principles, and the Biometricians such as Galton and Pearson, who doubted that Mendel's insights were relevant to quantitative traits. With the two schools reconciled, biologists stopped throwing stones at each other, and by the 1930s modern evolutionary theory had essentially taken shape.

Fisher defined the heritability of a quantitative trait as the fraction of the population variance of the trait was due to genetic variation, in particular to additive genetic variation, additive variation being variation that is transmitted from parent to offspring. By convention, heritability is written as h^2. The model led to the "breeder's equation" giving the predicted response of a trait to selection as $r = h^2 s$ where r is the response to selection, the difference between the average value in the population before selection and the average value of the offspring of selected parents. This equation is the workhorse of quantitative genetics as it relates the intensity of selection on a trait to the

rate of change in the character that would result. It seems too simple, yet in general it works very well.

A general finding is that nearly everything one can measure in humans is partially heritable, from stature and IQ on the high end (~0.80) to personality traits from questionnaires on the low (~0.25) (Turkheimer, 2011). Such estimates are obtained from correlations between relatives, most famously from the extent of similarity between pairs of identical twins. The implication from the generally positive findings is that many of the differences among us that have generally been assumed to be entirely learned, such as how religious we are or whether we are politically liberal or conservative, reflect in part gene differences. This suggests that group differences without our species also reflect in part gene differences, but there is little direct exploration of that possibility yet in the literature.

Heritability studies in humans have always been subject to doubts and criticisms. For example, similarities between identical twins separated early in life are a staple of the literature, but there remained doubt about whether their environments were really different. Further, even if separated at birth they had shared a uterine environment during gestation and this shared environment might account for similarities. These shadows of doubt, together with the old idea that gene differences are not the proper domain for the social sciences, have kept the impact of heritability studies low, or at least lower than they ought to be.

Most of these doubts and reservations are now resolved with the use of SNP (single nucleotide polymorphism) chips that provide for any individual his or her genotype at several hundred thousand or more genetic loci. These loci are not necessarily or even usually functionally significant, but these chips allow us to measure overall genetic similarity, called *kinship*, between any two individuals. "Kinship" in common usage means degree of relationship computed from a pedigree, while in genetics, kinship often means simply overall genetic similarity. The meanings are equivalent in practice, but kinship from chips is more accurate. For example, from a pedigree my kinship with my child is 1/4 or 0.25, but in fact my kinship with my child might be as low as 0.2 or so or as high as 0.30 or more. (Social scientists may be more familiar with what is called the coefficient of relationship rather than kinship: roughly relationship is twice kinship.)

In a series of papers, Peter Visscher and colleagues have computed kinship between all pairs of individuals from large longitudinal studies and looked at distant kinship and interesting quantitative traits such as stature (Yang *et al*., 2010), and intelligence (Davies *et al*., 2011) and schizophrenia and bipolar disorder (Purcell *et al*., 2009). In order to avoid the possibility of shared environments any pair more closely related than third cousins were not used. They showed that the new method using computed genetic

similarity yielded that same heritability estimates that had been known for years, essentially falsifying the doubts and quibbles that had plagued human quantitative genetics for decades. These papers also showed quite clearly that Fisher's original model of 1918 was in fact the right description of the inheritance mechanism: for both stature and intelligence our differences are caused by the small effects of a very large number of genes scattered over the genome, ending hopefully the futile search for "genes for" one thing and another. The details are somewhat technical: a lucid explanation is given by Visscher (2010) in a commentary on his earlier paper.

Epigenetic transmission has received much attention in the past several years. In the simplest case, some environmental effect causes DNA to be modified so that a gene is turned off or turned on, and this modified gene is transmitted to later generations. The modification is not permanent and the modification can be reversed (reset) in the future. We have so far no clear picture of its importance in human social behavior. The new SNP technology will certainly be an important tool in following this up, as a correlation between, say, parent and child would be inflated by shared epigenetics while correlations between more distant relatives would not. The SNP approach informs about correlations between, for example, sixth cousins, who have no idea of their relationship and who would not share epigenetic effects as parent and child or siblings would (Tal, Kisdi, & Jablonka, 2010).

An important category of model is the threshold model in which there is an underlying Normal distribution of some unobserved trait. Individuals with more than the threshold value of the underlying trait exhibit the phenotype under study. In an important series of papers in the 1970s Robert Cloninger and others proposed that sociopathy, defined by rigid explicit criteria, followed such a model in which the thresholds were different in males and females (Cloninger, Reich, & Guze, 1975a, 1975b; Reich, Cloninger, & Guze, 1975): the threshold for sociopathic behavior was higher in females. It required a higher concentration of the underlying trait to cause outright sociopathy in women.

Threshold models provide useful insight even without assuming anything about a transmission mechanism. For example, Eisner (2001) discusses the decline in homicide victimization rates in Europe from the Middle Ages to the present. A typical comparison in his data is from 50 homicides per 100,000 population in the year 1200 to 2 per 100,000 today, a 25-fold decline. This seems to be a big decline, but is it really? How does it compare to a decline from 100 to 4 per 100,000?

A model, perhaps a poor model but better than no model initially, is to imagine that homicide is a reflection of some underlying normally distributed genetic trait that has varied over time, perhaps because of selection against violence. A rate of 50/100,000 implies that this fraction of

the population, 0.05%, is homicidal. If the underlying trait has a standard normal distribution, 0.05% corresponds to the fraction of the population that is >3.3 standard deviations from the mean. A decline to 2/100,000, 0.002%, corresponds to 4.1 standard deviations from the mean. Assuming that selection has simply shifted the underlying distribution of violence proneness to the left without changing the variance, which is a consequence of Fisher's model and often seen in experiments with animals, the amount of shift is 4.1–3.3 or 0.8 standard deviations. In more familiar terms this 0.8 standard deviations would correspond to about 2 inches in height or 12 IQ points, not a very impressive change at all over 900 years.

What if were studying another social indicator, 100 times as common, that changed from in the same time from 50 to 2 per 1000 rather than 100,000. With the same assumptions about an unchanging variance the implied change in the underlying distribution is 1.3 standard deviations over 900 years, nearly double the change in proneness to homicide, corresponding to a change in height of 3.5 inches or a change in IQ of nearly 20 points. If this were due to selection, the implied selection intensity against the latter trait is much greater.

None of this is to claim that homicide or drunk driving or anything else is "genetic." If we had more information we could investigate the heritability of the trait(s) and build more accurate and more useful models. Even so, thinking in terms of a threshold and an underlying Normal distribution provides an important and useful way to understand changes in discrete traits over time.

ISSUES FOR FUTURE RESEARCH

Genetics and genetic approaches will become more and more important in social science research and understanding. Specific loci such as MAOA that we discussed earlier, and numerous others, are of limited interest as few of these loci are very significant in determining everyday human differences. This means that the practice of looking for "genes for" one thing and another will lose salience in social science. While the occasional locus may remain salient and important, it is increasingly apparent that most of our diversity reflects our small differences at large and very larger numbers of genetic loci, the effect of each locus so small as to be essentially undetectable. While the old paradigm of looking for "genes for" one thing and another will persist it will surely fade in the face of the even older quantitative genetic models as they come back for their place in the social sciences. The recent use of SNP chips has essentially resolved old doubts and controversies about biases and inaccuracies in heritability estimates. The standard model

of genetic influence being the combined small effects of a large number of genes scattered throughout the genome, fits most data quite well.

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Henry Harpending is Professor of Anthropology at the University of Utah. His academic webpage is http://harpending.humanevo.utah.edu. He has spent several long fieldtrips in the northern Kalahari studying genetics and family organization of foraging groups there as well as ranchers. He has also had a parallel career working on human biological and cultural diversity, human genetics, and modern human origins. Cochran and Harpending published *The Ten Thousand Year Explosion* (Basic Books) in 2010.

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Gregory Cochran is a physicist and an adjunct Professor of Anthropology at the University of Utah. His early career was in optical physics working in our aerospace industry. A decade or so ago, his interests turned to theoretical biology and anthropology. He has written about an infectious model of human male homosexuality, immune system diversity, and immune system evolution among human continental populations, and acceleration of the rate of evolution in humans, especially since the end of the ice ages and the origins of agriculture.

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