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Complexity, Genetic Causation, and Hereditarianism

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Abstract

Hereditarians have claimed that recent advances in psychological and psychiatric genetics support their contention that individual and group socially important aspects of behavior and cognition are largely insensitive to environmental context. This has been countered by anti-hereditarians who (correctly) claim that the conclusion of genetic ineluctability is false. Anti-hereditarians, however, sometimes use problematic arguments based on complexity and the ignorance that comes with complexity and a demand for mechanistic, as opposed to variational, explanations for the ways in which genes affect phenotype. I argue here, as a committed anti-hereditarian, that the complexity gambit and the demand for mechanisms open anti-hereditarian

arguments to counter-attack from hereditarians. Re-focusing the argument onto issues about to which uses heritability, genotypic scores, and genome wide association studies may be appropriately applied and reemphasizing the point that context matters are stronger measures to counter hereditarian claims.

Accompanying the latest round of genome wide association studies (GWAS) results and the increasing use of polygenic scores — indices of genomic variants used to predict phenotypic values — to predict phenotypic outcomes is an un-shelving of hereditarian prescriptions for society calling for interventions ranging from hailing the prospect of genetic testing for use in school tracking (Plomin, 2018) to outright eugenical measures (Hsu, 2014). Enthusiasm for the application of heritability estimates and polygenic scores to problems in social sciences has spilled over into the academic dark web and more extreme hereditarian sources who seek to apply them to find proof of immutable genetic causes of presumed racial and class differences in a variety of cognitive and behavioral features.

While outright denial that genetic influences on behavior are possible has faded, two other problematic arguments are used in anti-hereditarian replies to hereditarianism in social media and the academic literature. One is characterized by the highlighting the complexity of behavior in the context of the general failure of behavior genetics to yield convincing associations between genes and features of behavior or cognition. This complexity argument is used both to characterize genetic accounts of behavior as inadequate in their simplicity and to suggest that the evolution of behavioral characteristics will be constrained by genetic complexity in ways other aspects of phenotype will not. The other argument is based on claims of how dissatisfying explanations of genetic effects are in the absence of clear mechanistic links between genome and phenotype. In this view, genuine genetic explanation takes the form of a gene to protein to biological process to end phenotypic outcome account of traits.

Here, I argue that both the complexity argument and the insistence on molecular explanation do not do the kind of work anti-hereditarians would like them to do. While human behavior and cognition are doubtlessly complex, the complexity argument corners anti-

hereditarians in both theoretical and empirical ways. The insistence on molecular explanation is a needlessly reductionist move that risks casting genetics in a deterministic light. In both cases, these arguments are superfluous to the core of the classic, and correct, anti-hereditarian argument.

Some Responses to Recent Hereditarian Work

I focus on two anti-hereditarian responses to claims about the genetics of behavior and cognition. In the first place, there is a tendency to claim that GWAS, polygenic scores, and heritability analyses do not do justice to the complexity of human existence and that genetic complexity can hinder the evolution of traits. Whatever these new efforts to link genome to phenotype reveal, they are nested within a deeply multifaceted and interactive dynamical system that is not amenable to what is supposedly simplistic analysis, which explains the historical inability of behavior genetics to produce convincing gene-behavior associations. "Simplistic" is a common epithet applied to hereditarian accounts of human variation. Second, with respect to what constitutes a good genetic or biological explanation, anti-hereditarians sometimes demand the delivery of a gene through development and physiology to behavior account to accept that an explanation as being truly biological and convincingly genetic. I will demonstrate the use of these arguments by drawing on sources ranging from cases in the the academic literature to those in more popular venues.

Fuentes cites both the simplicity of genetic analyses and their failure to render results linking genetic variants to behaviors as critical shortcomings in behavior genetic research (2016).

> [W]hile there are a few cases of being able to tie specific genetic variants tied to specific outcomes with some diseases, it has not proven effective in complex behaviors and

> complex genetic systems, even in the extensive twin studies. The GxE approach to understanding behavior gives us overly simplistic, and incomplete, answers. [p. 301]

Similarly, in a review of recent book with a hereditarian outlook (Plomin, 2018), Comfort (2018) de- scribes the volume as "yet another expression of the discredited, simplistic idea that genes alone control human nature seems particularly insidious."

Stronger claims about the ways in which we might expect complexity to enable or inhibit certain evolutionary or otherwise variational outcomes are likewise used. As Evans claims in his recent critique of race science (Evans, 2018),

> Intelligence — even the rather specific version measured by IQ — involves a network of potentially thousands of genes, which probably takes at least 100 millennia to evolve appreciably. Given that so many genes, operating in different parts of the brain, contribute in some way to intelligence, it is hardly surprising that there is scant evidence of cognitive advance, at least over the last 100,000 years.

While couched in evolutionary terms, this speaks to within-human variation as we know it today as most of the differences among humans today and in the recent past evolved on the several tens of thousands to a few hundreds of thousands of years time scale. In this case, genetic complexity alone should keep all individuals roughly the same phenotypically within and among groups, however defined.

Grauer makes a similar argument (Graur, 2018) when replying to Reich's (2018) claim that there was sufficient time for evolution to work to differentiate complex traits between different populations of humans today:

> Unfortunately, the long-term effective population size for all the humans in the world is barely 10,000 — lower than that of chimpanzee. By necessity, the effective population

> size of each race separately is much smaller. So, the chances that 74 loci will experience significant changes in allele frequencies simultaneously in each of the four populations is zero.

In this case, it is the polygenic nature of complex traits that limits their evolutionary potential, or at least evolution by random genetic drift.

Fuentes (2015) bridges the complexity argument with one about what it means to be biological in a concrete sense by outlining a set of critical questions one should ask of claimants of genetic influences on behavior.

> So, trying to connect genes and behavior is not at all simple. We know there are genetic components to all aspects of life, but how or whether specific alleles affect complex behavior patterns is far from obvious. If you do hear an assertion about a relationship between genes and behavior, you need to think critically and ask a few basic questions. What is the gene or genes? How many alleles are there? What protein or proteins are coded for? How do these proteins affect the organism so that a specific behavior is performed? [p. 49]

What constitutes a satisfactory genetic explanation, in this view, is a step by step account of the ways in which genes act through development and physiology to cause individual differences.

In a similar vein, Turkheimer (2016) holds up Huntington's disease and a hypothetical genetic etiology of divorce as examples of what constitutes a compelling genetic explanation:

> The co-occurrence of dementia and choreiform movements in Huntington's disease has a strong genetic explanation: Both are the result of a mutation in a single dominant gene. Strong genetic explanations do not have to refer to single gene mechanisms, however. If it turned out that divorce was the result of a network of countable genes with specifiable

neurological and then behavioral consequences, eventually compelling people to dissolve their marriages, our conception of divorce would have to change.

Across these cases, we see a combination of two different arguments from some critics of hereditarianism. One appeals to inherent complexity and a history of the empirical shortcomings of behavior genetic investigations as a reason to not accept hereditarian claims about genetic influence on behavior and casts doubt that features like intelligence might evolve quickly. The other makes a claim that a good genetic explanation involves genetic mechanism. I contend these arguments do not do good work toward anti-hereditarian ends.

Complexity

Everyone agrees that life, behavior, and the genetic influences on them are complex in the sense that they involve many influences ranging from genes to environments and interactions thereamong. Indeed, quantitative genetics, the discipline spanning no-man's land separating the forward trenches of both parties to the conflict over genetic determinism, is the science of complex traits (Lynch et al., 1998). I identify problems with the argument from complexity at a broadly conceptual level, at an empirical level, and from an argumentative standpoint.

At the conceptual level, too warm an embrace of complexity is a denial of the possibility of theory. All understanding relies on abstraction and simplification. If life is so very complex and we ask that every last detail be accounted for before an explanation is accepted, any attempt at a science of human behavior or anything else that includes genes or environment is an exercise in futility. Like Borges' cartographers who bankrupt their fictional kingdom by building a 1:1 scale map of it (Borges, 1999), many anti-hereditarians risk demanding a map of life of such precision and accuracy that it would be impossible to understand because of its extreme accuracy

and precision, and thus useless (see Bunzl (2008) for an analogous problem in the social sciences). Good scientific explanations, certainly at the biological and societal levels, are both simplistic and wrong in important ways. Focusing on the technical questions of precision and accuracy misses the larger point in that what constitutes an *adequate* explanation. Explanatory adequacy is a larger question informed by what is at stake and our aims and aspirations, issues not directly related to precision and accuracy.

Versions of the complexity argument forward empirical claims and have hidden and strong theoretical commitments that run contrary to our best evidence and theory. The claim that genetic complexity inhibits the rates of evolution of a characteristic is a good case in point (Evans, 2018; Graur, 2018).

Complex traits like morphology, however, have high rates of evolution in humans, and a mode and tempo of evolution often consistent with random genetic drift, and sometimes directional selection (Lynch, 1990; Ackermann and Cheverud, 2004; Schroeder et al., 2014). Indeed, the rates of cranial evolution among recent human groups is much higher than those found among groups of chimpanzees (Weaver and Stringer, 2015). In small populations undergoing random genetic drift, we expect rapid evolutionary change in characteristics whose variation is governed by many environmental and genetic influences and their interactions. These morphological characteristics are the products of developmental processes that are as complex as they come (Hallgrimsson et al., 2014) and the rapidity of their evolution decidedly refutes this part of the complexity argument. It may be that environmental effects swamp out genetic effects or otherwise stymie random genetic drift and natural selection, but there is nothing inherent to complex traits that will slow their evolution over short time spans. In contrast to the constraint view of complexity, it is much more likely that the complexity of these kinds of traits are

practically invitations for variation. The array of perturbations to developmental systems leading to subtle variations in phenotype are vast for most complex characteristics and it is exceedingly rare to find complex traits that do not exhibit some kind of variation somewhere or another in the grand diversity of life (Hansen and Houle, 2004). While there may be other reasons — evolutionary, environmental, or interactive — to suppose that some aspects of phenotype should not have much evolved among populations in the recent past, genetic complexity is not one of them on its own.

From a more rhetorical standpoint, critiquing and rejecting hereditarian arguments about the relation-ships among genome, organism, and environment by pointing to a past set of failures behavior genetics and accusing them of oversimplifying a complex situation is a self-defeating strategy. What was not possible during the debates over the *Bell Curve* (Herrnstein and Murray, 2010) — the mapping of genetic effects on behavior and cognition to regions in the genome — is now central to psychological and psychiatric genetics (Savage et al., 2018). While the deliverances of quantitative genetics do not solve biological problems on their own (no result in biology does, irrespective of its disciplinary origin), the kind of merging of systems and variational perspectives that will allow for more mechanistic understandings of the complex relation- ships between genome and phenotype in environmental context beginning to be used for morphological and developmental characteristics (Jamniczky et al., 2010) may be applied to behavior. If genetic accounts of behavior begin to mimic their morphological and physiological cousins and as more complex studies of genes and behavior are lavishly funded, ground erodes from beneath the palisades of the complexity argument and it is not clear where anti-hereditarians of the complexity stripe can further retreat.

Mechanism and Biological Explanation

Turning to the issue of what kind of explanation constitutes an adequate explanation for behavior, some anti-hereditarians are inclined to be dismissive of variational approaches to genetics in the form of the study of heritability and genetic association/linkage in favor of mechanistic accounts of the ways in which genes affect behavior or other aspects of phenotype. This distinction fits neatly into Turkheimer's framework of "weak genetic explanation" and "strong genetic explanation" (Turkheimer, 1998, 2016).

Strong genetic explanation matches with Fuentes' recommendations (Quoted above. Fuentes, 2015) for the critical acceptance of a genetic effect on behavior: A molecule to phenotype cascade of identifiable causation. Among the strong cases for genetic causation is Huntington's disease in which a known genotype leads to the production of a known toxin that has phenotypic effects ranging from the physiological to the behavioral and cognitive (MacDonald et al., 1993).

Weak genetic explanation involves the observation that genes are involved in some way in the development and physiology of any aspect of an organism's phenotype (Turkheimer, 2016). This applies with equal force to variational quantitative genetic studies and GWAS. In the former case, almost every variable behavioral or cognitive property of humans is heritable to one degree or another, an observation dubbed by Turkheimer as "the first law of behavior genetics" (Turkheimer and Gottesman, 1991; Turkheimer, 2000), disagreements on the biases associated with heritability estimates aside (Feldman and Ramachandran, 2018). Similar, more modestly labeled conjectures about variation have been proposed for a variety of characteristics across wide swaths of the diversity of life (Cheverud, 1988; Roff, 1995). In the latter, we expect nearly every variable property of behavior or cognition to travel with a multitude of genetic

associations, each of small effect and perhaps low frequency (Boyle et al., 2017). In all of these cases, if it varies, it is probably heritable to some degree and will harbor extensive genomic associations, albeit few that are easily explained in a way satisfying the conditions for strong genetic explanation. This near universality of genetic influence on just about every variable trait, behavioral or otherwise, arises from the fact that genetic influences can manifest themselves in ways that do not conform to our standard conception of genes acting at the molecular level. Association studies have shown that genes outside of traditionally conceived genetic pathways play an important role in generating variation in complex traits (Boyle et al., 2017). Maternal effects in mice can be the product of maternal genotypes varying the environments of the pups (Wolf et al., 1998). In this case, the genetic effects transcend physiology and development to ricochet about the environment before being re-internalized by the offspring. Phenotypic characteristics can arise through the action of and interaction among processes at multiple traditionally understood levels of organization ranging from the spatial arrangements of tissues during development to the ways in which the concentration in chemical gradients plays out over wide areas of a developing organism (Gawne et al., 2018). Mechanical forces acting at the level of the whole organism or structure level can also cause individual differences in phenotype (Wallace et al., 2017). The fact that genetic information can flow through systems via multiple channels and can cascade across traditionally defined levels of organization ranging from the molecular to the social means that genes can affect just about anything.

Do we need to have a molecule by molecule account of how a gene works to be confident that there are genetic effects on phenotypic variation? The answer to this depends on the question. Questions at the organismal and population level, do not always demand mechanistic genetic explanation. It is indisputable that nutritional deprivation stunts growth in children and

adolescents and we knew that well before we knew anything about the molecular machinery of growth plates (James, 2006). Similarly, the links between smoking and lung cancer were well established before the details of the physiological operations of any cancer were understood (Hoffman et al., 1929). We can tell similar stories for phenomena as disparate as the realization that sewers and clean water supplies were important for health (Chadwick, 1842) and the efficacy of vaccination (Lombard et al., 2007). In all of these cases, the big picture of how causal influences were flowing through the world were clear before the molecule- or microbe-eye view of the mechanisms of causation were worked out.

It is undoubtedly correct that, beyond demonstrating that the genetic influences on most variable traits are probably influenced by many loci of small effect, heritability and GWAS give us very limited insight into the mechanisms underlying trait development and individual differences. No amount of manipulation or calculation performed on covariance matrices will glean any more understanding of developmental or mechanistic process (Bookstein, 2016; Mitteroecker, 2009). The uses to which these techniques are suited include predicting phenotypic outcomes in particular environments supposing that the environments do not change, how successful selection (natural or artificial) will be in effecting evolutionary change in particular environments, and serving as a control on relatedness when searching for environmental causes of individual differences in contexts in which there study subjects are related (e.g. family studies. Turkheimer and Harden (2014)). The usual provisos apply here. These studies do not provide evidence that individual or group differences are immutable and inevitable across environments. Nor do they give any indication of how developmentally determined a trait is. They do, however, tell us whether a trait is affected by genes in the most general sense and no further understanding of mechanism is necessary to substantiate this kind of claim. I contend that the distinction

between weak and strong genetic or variational and mechanistic explanation is not as clear as we might like when it comes to classifying the characteristics we study. We can illustrate this by evaluating Turkheimer's claim that "[w]eak genetic explanation of complex individual differences does not imply that those differences have genetic mechanisms for scientists to discover" (Turkheimer, 2016).

A quick inspection of the diversity of life makes the difficulty with this position clear. A comparison of *Ankylosaurus* clubs (Arbour and Currie, 2015), *Stegosaurus* spikes (Cobb, 2009), and the tailless Manx (Todd, 1961) shows that tails can evolve in elaborate ways. The genetic basis of these tail characteristics (outside of the Manx) are as obscure as that of cognitive characteristics as are the vast majority of traits that have ever evolved. Evolution takes place nonetheless, showing that there are mechanisms that generate genetic variation amenable to sustaining evolutionary responses even if our understanding of relationships between genotype and phenotype in any one generation is less than concrete. This is a simple extension of the Darwinian observation that there is no difference in kind between variation within and among species.

Over evolutionary time, shifts in, and elaborations on, developmental processes become more easily identifiable as they become differences between species. While strong accounts of the genetic basis of facial morphology are elusive and look to remain so over the near term (Hallgrimsson et al., 2014), differences in facial shape among amniotes have been located in the regulation of the differential contributions of growth in the frontonasal prominence and the maxillary component of the first branchial arch, among other developmental mechanisms (Young et al., 2014). In contrast, developmental systems drift leads to situations in which homologous, even more or less identical, characteristics may have appreciably different

mechanistic underpinnings (True and Haag, 2001). Evolution discovers or produces genetic mechanisms that generate differences within and among species. None of the diversity of life would be possible without this propensity for uncovering and tinkering with genetic mechanisms.

When we build scenarios of how things might vary across contexts in any one time and through evolutionary time as opposed to narrowly restricting ourselves to what might be in front of us at the moment, we can see how situations amenable to explanation in strong genetic terms may turn into ones approachable only by weak genetic explanations and vice versa. In a context in which factors (e.g. medications) kept toxins from building up during the life course of someone with the Huntington's disease allele, we can easily imagine the causes of Huntington's to become as obscure as those influencing divorce or personality. In a world in which many treatment options for Huntington's have been discovered and a population exhibits genetic variation in responses to drugs, acquiring Huntington's disease may be the effect of adverse interaction with a medication, which would be subject to innumerable environmental and genetic influences. We can also imagine a world in which the only permissible grounds for divorce was contracting Huntington's disease. Out of evolutionary context, and supposing that inhabitants of this world hew to a static view of society much in the same way that hereditarians do (see below), divorce would look like it had a strong genetic explanation.

So, while our understandings of divorce and Huntington's disease may be very different on account of the fact that the genetic influences on the former are inscrutable and those on the latter obvious from our perspective in our present context, there is no difference in the underlying principles that structure the causes of and variation in these different life outcomes. Some classes of causes are more relevant than others in different contexts, but the differences are in degree

rather than in kind. Where there is genetic variation in phenotype, there are always genetic mechanisms and all will depend on context in one way or another. The only distinctions lie in the kinds of questions we are asking and the degree to which our research faculties are up to the task of uncovering answers to them.

It is not clear what the demand for strong genetic explanation is supposed to deliver for anti-hereditarians. A mechanistic explanation is no less context dependent than statements about variation at the population level. As with results from heritability studies, a mechanistic understanding of a trait gives us little indication of the amount of change we might expect when context is varied. Irrespective of the level of analysis, for the purposes of the anti-hereditarian argument, it is the emphasis on the context dependency when asking questions about heredity and the appropriateness of the tools for approaching the question that matter.

My fear is that choosing to only accept extreme and well-characterized clinical phenotypes or simple Mendelian characteristics with good molecular level mechanistic explanations as genuinely genetic casts genetics in biased ways. While these cases may draw our attention because they are either obvious or cause suffering we wish to ameliorate, they are comparatively quite rare (Antonarakis and Beckmann, 2006). These can be the most difficult characteristics to change via environmental intervention (in our present context), sometimes requiring multiple surgeries or life-long use of drugs to manage a condition. By insisting these (presently) difficult to change conditions are the only legitimate cases of genetic causation, we anti-hereditarians falsely give the impression that the relationship between genotype and phenotype is highly deterministic. In effect, we anti-hereditarians are tacitly agreeing with hereditarians that genetics blesses or damns inevitably.

Context and Change

It is worth emphasizing that these two issues with hereditarian arguments do not damage the strength of the core anti-hereditarian critique, which has been on firm ground since its inception (Lewontin, 1970). The genetics of complex traits are highly context dependent and there are strong environmental effects on behavior and cognition (Turkheimer et al., 2003; Trahan et al., 2014). Heritability and the newer polygenic scores say little about how traits would be distributed in different contexts. The effects of genetics on many features in humans are probably overstated because of estimation bias (Feldman and Ramachandran, 2018; Morris et al., 2019). Statements about ineluctable race and class differences in behavioral and cognitive features and ability to participate in societies are nefarious speculation. Indeed, it is difficult to say what a convincing test of this kind of hypothesis would look like much less how one could be considered ethical. The classic argument is worth repeating here to emphasize the importance of context and the ways in which the attitude of hereditarians toward context reveals things about their intent.

Contrast the attitudes of hereditarian genetic determinists, particular those interested in behavior or cognition, with those of practitioners of pharmacogenomics (Whirl-Carrillo et al., 2012). Both concern themselves with the ways in which genes influence life outcomes. Pharmacogenomics, in its own way how- ever, has a diametrically opposed set of theoretical outlooks and ambitions to behavior genetic hereditarians. Where hereditarians are content to allow their results stand uncritically analyzed as though they were the natural, proper, and unalterable order of things, pharmacogenomicists seek out drug targets so that they may change life outcomes by changing an organism's environment through the use of chemical medicines. The difference here is the prevailing attitudes of the practitioners of the different realms of inquiry toward context. Both parties agree genes are at work, but the hereditarian stops there

with the notion that heritability or well-mapped out genetic causation means inevitable and wholly determined outcomes. One seeks to stifle imagination about possible ways of being while the other seeks to navigate possibility and the evitability inherent to life, albeit through what may be needlessly narrow chemical approaches.

What is particularly telling about this distinction is the fact that I have not met a hereditarian who would argue against the utility of pharmacogenomics. So, when a hereditarian is making a statement about how a trait cannot be changed through environmental intervention, they are really saying that there is a class of traits for which, and a class of contexts in which, we should not bother to try. That these characteristics are those hereditarians identify as being socially important reveals that theirs is an ideology meant to cement a particular social order into place.

Estimates of heritability and genetic associations have very limited uses on their own. They indicate whether a characteristic might evolve in response to evolutionary processes such as selection (natural or artificial) and random genetic drift and give a sense of what phenotypic outcomes are more likely for individuals given genotype or information on related individuals in given environmental contexts. Other applications include serving as a control of relatedness when searching for environmental effects on traits (Turkheimer and Harden, 2014). Since hereditarians rule out change to a social order and the prevailing set of environments that come with it, the only remaining uses for heritability or polygenic score estimates are to fit existing people by virtue of their genotypes into an existing order or to breed new people for fit to an order. Plomin's (2018) suggestion to use genetic testing as a part of evaluation for employment is a good example:

For selection for the purpose of employment, it is again an empirical issue how much

> polygenic scores can add to the prediction of success on the job. It seems likely that polygenic scores can help because tests and interviews are notoriously poor at predicting job success, predicting just a few per cent of the variance. Psychological polygenic profiles might be especially useful in considering patterns of strengths and weaknesses that predict success at particular jobs. Similar to the example of dating websites, a password-protected link to a direct to consumer company could make available a certified set of polygenic scores relevant to specific jobs. [p. 181]

This echoes Herrnstein and Murray's "A Place for Everyone" argument from two and a half decades past (Herrnstein and Murray, 2010), proposing that supposedly genetically determined intelligence scores be the proper way by which we evaluate the fairness of life outcomes and to ease the placement of people in their genetically rightful places in a society. The notion that polygenic scores and other genetic quantities might be context dependent is not unfamiliar to hereditarianism, it is simply ignored when they make social prescriptions. The difficulty with hereditarianism is not that it advocates too strongly for biology. Nor is it because it is simplistic in its explanations — some hereditarian studies are plenty complex — or insufficient because they often do not include a molecule to behavior account of psychology. Rather, hereditarianism's primary failing is the kind of biology it represents. It is a set of restrictions on the reach of the imagination, limiting us to questions of how to best fit people into, or produce people to fit, particular social orders.

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