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NATURE, NURTURE, AND INDIVIDUAL CHANGE

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ABSTRACT: Determining the degree to which persistent human behaviors and traits are the result of genetics or environment is important for a host of theoretical reasons in psychology. This article asks whether the results of such determinations are relevant to the practical tasks of individual change as attempted, for example, through therapy, parenting techniques, or self-transformation. Examples from the psychological literature on happiness or "subjective well-being" illustrate the common idea that a trait being largely genetic implies that it is more difficult to modify than one that is largely environmental. The most widely known theoretical approach to disentangling genetic from environmental effects in humans is behavioral genetics, with its central concept of heritability (h^2). This article argues that measures of h^2 do not predict the ease or difficulty of modifying behaviors or traits of individuals. A concept different from h^2, that of innateness, is explicated, but it, too, is found not to be a useful predictor of individual modifiability. The general conclusion is that knowing only the degree to which a human characteristic results from nature or nurture has no implications for the ease or difficulty of altering that trait.
Key words: heritability, behavioral genetics, nature–nurture, parenting, behavior therapy, innateness, norms of reaction, personal transformation

Therapists seek change in the behaviors and attitudes of their clients, individuals attempt the same on their own, and parents seek to instill personal traits in their developing children. In all these cases the intention is to alter either what already is (e.g., agoraphobia in an adult) or what is thought likely to develop (e.g., a secular outlook in an offspring). One issue that has been considered to be relevant to these attempts at individual change is whether the behaviors and traits are more a result of nature or nurture. This is considered to be relevant under the assumption that the likelihood of achieving human change is diminished to the degree to which a trait is genetically caused and enhanced to the degree that it is environmentally caused.[1] For example, it is taken as important to gay rights proponents that sexual orientation is hereditary (so straight sex cannot be viewed

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[1] There is an additional idea that what is genetically caused is more closely related to one's personal identity, but this will not be treated here.

as a legitimate option) and to opponents that it is chosen (so that it can be unchosen) or nurtured (so that it can be un-nurtured).

The position of this article is that there are no implications for the ease or difficulty of individual human change that follow from alternative conclusions concerning whether a trait is more nature-based or environment-based. The first part of the argument shows that the degree of heritability of a trait, as represented by behavioral genetic analysis, has no implications for the question of whether the trait is easier or more difficult to modify. The second part of the argument shows that heritability and innateness are separate concepts, and finally, that the degree of innateness has no implications for modifiability. Examples are provided from relatively recent work that conflates modifiability with both heritability and innateness.

Heritability

No human trait can be solely genetic or solely environmental. "Having two thumbs" is strongly genetic yet requires a certain range of environments for its existence (e.g., those below a particular level of thalidomide). "Speaking fluent Latin" is strongly environmental yet requires certain basic genetic structures for its existence. Behavioral genetics is the science most adept at quantifying the relative influence of genetic and environmental factors upon individual differences in levels of a trait in a population. For example, behavioral genetics can calculate the body height variation in a population then show that this population's genetic variance accounts for 80% of the height variance. It can also, though with less success, sort out the relative importance of the factors of gene/environment interactions, nonrandom gene/environment correlations, and error.

It has been conceptually straightforward, though logistically formidable, to show that genes affect individual trait differences in a population. Genetic influence is typically diagnosed using studies of identical twins reared together and apart, family studies comparing identical and fraternal twins, and adoption studies comparing early-adopted children with their biological vs. their adoptive parents. Turkheimer and Gottesman have proposed that "h^2 (heritability) $\neq 0$" should be enshrined as the first law of behavioral genetics (Turkheimer, 2000). Maccoby (2002), not at all a fan of some of the stronger claims of behavioral genetics, wrote, "Behavior geneticists have made their case. Genetic factors do clearly make a significant contribution to individual differences. . ." In the interest of foreshadowing, one should keep an eye on "individual differences" as the subject matter of heritability studies.

Behavioral genetics has sought to show not only that genes are consequential but also the comparative influence of genes and the environment. To accomplish this quantification it employs the concept of heritability (h^2). In a widely reported study, Cherkas and colleagues (2004) studied the heritability of female infidelity among a population of women in the United Kingdom. Lead researcher Tim Spector is quoted in *Medical News Today* (2005) as saying ". . .we found that around 40 percent of the influence on the number of sexual partners and infidelity

were (sic) due to genetic factors." What should the reader understand is being measured by the decimal .40? What if it was .90? Should husbands be any more watchful of their wives? For clearer examples, Silventoinen and colleagues (2003) report that the heritability of body height of Swedish male adults is approximately .80. The heritability of IQ has been calculated between .50 and .80 depending upon factors such as age and socioeconomic status, and Rowe (1994) reports the following:

Personality Traits	**h^2**
Extraversion (gregarious, sociable, dominant)	.49
Agreeableness (kind, affectionate, friendly)	.39
Conscientiousness (reliable, organized, planful)	.40
Emotional stability (calm, not worrying, stable)	.41
Intellectual openness (original, insightful, inventive)	.45

Attitudinal Traits	**h^2**
Authoritarianism (rigid adherence to authority)	.60
Religiosity	.30
Prejudice	.26

Of course, there are challenges to these numbers. For example, conscientious people might, because of their conscientious natures, select social environments that reinforce their conscientiousness. Authoritarian people might gravitate to groups that reinforce similar tendencies. In these two cases the resulting behaviors and traits will be affected by these gene/environment interactions, yet they are methodologically attributed to genetic factors (in all but a few cases), which inflates the h^2 number (Sesardic, 2002). In the other direction, Rowe (1994) points out that shared religiosity is a strong marriage attraction factor. This produces greater genetic effect upon children's religiosity, but that effect will methodologically mimic the consequences of family environment. For the purposes of this article these and other reservations will be set aside.

It is essential to this article that the meaning of a heritability claim is understood clearly and that common misunderstandings are avoided. Where "genotype" denotes the structure of an organism's genes and "phenotype" denotes the organism's observable traits (e.g., body height), the phenotypic variance (PV) of heights of Swedish male adults will be a function of both the genotypic variance (GV) within the population and environmental variance (EV) for the population. Thus the formula PV = GV + EV. Put simply, the degree of variation in this population's height is a function of the degree of variation in both the population's genes and in its environment.[2] The intuitive idea of how to accomplish the measurement of h^2 is described by the following hypothetical case:

[2] Gene/environment interactions, nonrandom gene/environment correlations, and error are ignored here.

1) Calculate the mean height of a sample of 100 Swedish men, unrelated by blood, between ages 20 and 40 years. The PV in height of this set of unrelated men (PV^U) is a measure of the dispersion of individual heights around this mean. For statistical reasons the variance is calculated as the mean of the squares of the distance of each member's height from the sample's mean.

2) Select a sample of 50 *pairs* of Swedish male identical twins between ages 20 and 40 years who were raised apart from birth (as were the members of the unrelated population). Calculate the PV of this group of identical twin pairs (PV^I).

3) In theory, the set of 100 unrelated men contains 100 different genotypes while the 100-person set of 50 identical twin pairs contains only 50 separate genotypes (the genotypes of identical twins are the same). So in this case $GV^U > GV^I$. Because the two sets of 100 men were selected from the same population, and because all members were raised separately, we can roughly assume that the EV for both samples is equal, and so infer that any difference between PV^I and PV^U can be attributed to differences between GV^I and GV^U.

In summary, the measurement of h^2 involves comparing trait levels between populations, each of which contains an identifiably different level of genotypic variation. The decimal h^2 measures the contribution of the population's GV to the population's variance in the trait level. It is a population statistic, so it does not measure the degree to which heredity contributes to a trait level in any individual. Returning to the infidelity example, rather than stating ". . .around 40 percent of the influence on the number of sexual partners and infidelity were (sic) due to genetic factors," it would have been closer to the research conclusions to state ". . .around 40 percent of the variance in number of sexual partners and infidelity within the sample was due to the genetic variance within the sample population." It is difficult to construe these two sentences as expressing the same idea.

It is worthy to note that in standard behavioral genetic methods only GV is measured directly (e.g., a set of 50 identical male twin *pairs* [100 men] has half the GV as a set of 100 unrelated men). The measures of EV appear as remainders from the subtraction of GV from 1.0. There is no accepted framework within behavioral genetics for independently measuring the EV of a population (Turkheimer, 2000), though intuitive assumptions are made (e.g., that the EV for twins raised apart is greater than for twins raised together). A different approach to disentangling nature and nurture that uses norms of reaction employs direct measures of the environment.

Heritability and the Possibility of Individual Change: An Example

What is the relevance of a trait's h^2 in a population to the prospects of modifying the trait in an individual in the population. Let's begin with an example from parenting. Kate and Steve have a 9-year-old son Jim. Both parents are outgoing and socially at ease. Both have successful careers and are convinced that their extroverted personalities have been important assets to them in both their personal and professional lives. Jim's personality, on the other hand, is on the introverted end of the spectrum as compared with his age peers and his parents. Kate and Steve worry about its effects upon his childhood social life and upon his later career prospects. They wonder about the prospects of altering Jim's environment in ways that would result in Jim becoming more extroverted. What relevance to these prospects is the h^2 factor of .49? If the h^2 factor were .29 or .79 would the prospects of changing Jim's introversion be any more or less difficult? One thesis of this article is that the h^2 of a trait in a population is causally unrelated to the degree of difficulty of altering that trait in any individual person. More specifically, some traits with high heritability will be simple to modify and others difficult. The very same is true of traits with low h^2. The evidence necessary to establish ease or difficulty of modifiability is independent of the evidence that establishes heritability.

The Belief That Heritability Affects Modifiability

On the surface, the thesis that levels of h^2 are irrelevant to modifiability seems counterintuitive, perhaps because heritability is associated with being "wired for" something and the latter seems formidably permanent. We could identify three distinct groups that discuss heritability numbers: (1) popular writers, including the press, (2) researchers outside of behavioral genetics who seek to employ heritability numbers in their own work, and (3) behavioral geneticists themselves. Of these three it is the first two who are eager to focus on applications of the heritability research, who are less careful about the limitations of h^2 numbers, and who are more likely to assume that higher h^2 numbers imply greater difficulty of modifiability in any individual. It is easy enough, therefore, to verify that Cook-Deegan (2001) is correct that "There is a persistent conceptual virus that conflates heritability with immutability." Below are five examples. They are taken largely from the psychological literature on "happiness" or "subjective well being" (SWB). I employ these examples because this is an interesting literature with promise for practical results, the citations are relatively recent, and it is a break from the "Heritability and IQ" literature that has dominated such discussions.

Example 1

Two of the most important researchers in this area are Lykken and Tellegen. They provide a very good example of researchers inferring difficulty of modifying traits in individuals from the results of heritability studies. In their widely quoted

paper "Happiness is a Stochastic Process" (1996) Lykken and Tellegen argue that the level of happiness at some specific time has an h^2 of about .50, but the average level (the "set point" or "baseline" above and below which momentary levels vary) is about .80 heritable. They conclude,

> If the transitory variations of well-being are largely due to fortune's favors, whereas the midpoint of these variations is determined by the great genetic lottery that occurs at conception, then we are led to conclude that individual differences in human happiness—how one feels at the moment and also how one feels on average over time—are primarily a matter of chance. (p. 189)

On this pessimistic note they go on to state that the high heritability of happiness set points implies that trying to be consistently happier ". . .is as futile as trying to be taller. . ." (p. 189), a remark that is often quoted and has been influential in the field (see Diener, 1999; Lyubomirsky et. al., 2005, below). There may, in fact, be good evidence for the fixity of mean individual happiness levels over time. The argument here will be that the level of heritability of mean happiness is not the type of evidence necessary to make this point.

Example 2

Lyubomirsky and colleagues (2005) accept Lykken's tallness analogy as a source of "pessimism" that happiness levels can be increased. They write,

> Thus, although there may be substantial variation around this baseline level in the short term, in the long term people perhaps cannot help but return to their set point, or to the middle of their set range: "What goes up must come down." (p. 113)

Again, the idea is that (as a general rule) environmentally-based changes in SWB are easier to modify than the genetically-based set point, and that the latter, as measured by h^2, is in fact very difficult to alter.

Lykken, however, regretted the tallness analogy almost immediately, though not for the correct reasons. He stated one year later,

> . . .if the happiness set-point is largely determined by genetic factors, does this mean that there is nothing we can do to increase our own or our child's hedonic level? In the paper in which Tellegen and I reported these findings, I said that it may be that trying to be happier is as futile as trying to be taller but I regretted that remark as soon as it appeared in print. I should explain first that if our happiness set-points were indeed biologically fixed, that would not really be as bad as it sounds because the vast majority of humans seem to be relatively happy most of the time. (Lykken, 1997, p. 6)

It is the view argued here that what Lykken should have regretted is something else, *to wit*, the assumption that high h^2 *per se*, without further evidence that goes beyond h^2 levels, implies difficulty of modification. He should have also

regretted the implication that the causes of a trait in any individual in a population can be inferred from the h^2 for that trait in a population.

Example 3

Martin Seligman has done interesting research on the factors that promote human happiness, and he presents a guardedly optimistic view that it might be enhanced in individuals (2001; Seligman & Csikzentmihalyi, 2002). Steven Pinker (2002) is most recently known for his arguments that important genetic factors shape human life. In a conversation between the two in *Slate* magazine (Pinker & Seligman, 2002) Seligman notes that there are learnable skills that can enhance "the pleasant life," but that

> Such "positive affectivity" is highly constrained genetically. It is roughly 50 percent heritable, with identical twins much more similar for it than fraternal twins. Like any heritable characteristic (e.g., body weight), the best we can achieve by dint of will and of tuition is to live in the best part of our set range of smiley good cheer. (p. 3)

The image here is of the causation of human traits and behaviors divided between the fixedly heritable and the modifiably environmental. The reasoning is, I suppose, that if the environment caused it then alterations of that environment can "uncause" it, and if the genes caused it then no modification of the environment can "uncause" it. Put starkly like this, however, the idea seems suspicious.

Pinker, however, likes the model and thinks of the environmental slice of life's causality—though not the genetic—as providing "wiggle room" or "elbow room" for individual change (2002). He states,

> Both of us have explored the elbow room left by our genetic endowment and evolutionary history. Traditionally this is attributed to some vague entity called "the environment" or "nurture," variously identified with some parenting, culture, schools, conditioning, statistical regularities in sensory input, and the mass media. . . (p. 8)

Example 4

The possibility of altering human happiness is of interest to economists as well as psychologists. After all, the economic concept of utility, designating the degree to which outcomes fulfill goals, grew out of the earlier utilitarian idea of happiness. British economist Richard Layard (2003) supplies reasons for the difficulty of boosting social happiness, then notes:

> All of which shows how difficult it is to produce a happier society. This is of course partly because of the strong role which our genes play in determining our temperament. Identical twins reared apart have happiness levels with a correlation as high as 0.5. But there is still plenty of room for nurture as well as

> nature—just as people are now inches taller than a century ago, despite the strong heritability of .9. (p. 9).

Here Layard is speaking of large-scale increases in the means of a heritable trait in a population while the variances remain the same. This is discussed below. Though he is not referring to individual change, we once again encounter the idea that genetic effects (specifically here as measured by h^2) enclose an immutable area of the causation that leaves only the environmental segment open to manipulation. This idea was reiterated by Layard in a lecture at the Washington, D.C. Brookings Institution (2005):

> I don't like heritability measures. . .but the heritability measures of cheerfulness is (sic) fairly high, like 40 percent or something. Not important, really, because another 60 percent becomes a fact. (pp. 23-24)

Example 5

Finally, Fujita and Diener (2005) argue that one of the explanations for the stability of SWB over time is the strong heritability of SWB, and a careful reading of David Armor's *Maximizing Intelligence* (2003) will reveal the same assumption that heritability translates into un-modifiability.

Arguments Against Inferences from h^2 to Individual Modifiability

Heritability Measures Cannot Refer to the Traits of an Individual

This article concerns the issue of *individual* trait change as opposed, for example, to changes in a population's variation for a trait. The measure h^2 is a population statistic only. It measures the portion of a trait's PV in a population that is explained by the GV of that population. An individual has no genotypic, phenotypic, or environmental variances for a trait. Thus it makes no sense to say of an introverted son, where introversion's h^2 is .49, "Well, at least we have 51% of the causation of the trait to work with." This logical fact has already been noted within the IQ and heritability literature. Block and Dworkin (1976), Kitcher (1985), and Stoltenberg (1997) have described examples of the error, yet it remains. In addition to examples in the previous section, Block (1995) quotes the error from *The Bell Curve* (Herrnstein & Murray, 1994), ". . .when we say 60% heritability, it's not 60% of the variation. It is 60% of the IQ in any given person." Lykken was mistaken in the quote cited above not only in that the high h^2 of SWB would imply un-modifiability, but that it has implications for "our own or our child's hedonic level" (1997, p. 5).

The h^2 of a Trait vs. the Mean Level of the Trait

Against the idea that the h^2 of a trait in a population implies nothing about the level or causation of the trait in an individual of the population, one might compare

h^2 to a straight mean. If we know that the mean height of males in a population is 5'9" and that heights are distributed normally, we could conclude at least that the most likely height of a random individual is 5'9" (though it might not be very likely). One might think that knowing the h^2 for a trait in a population might tell us at least what is most likely to be the level of genetic influence upon a random individual, but the analogy does not carry over. Although the h^2 for the male height of a population is .80, there is literally no corresponding feature of a random individual of that population that is .80. While it is perfectly possible for an individual to have the population's mean height of 5'9", he or she cannot have height's h^2 for that population. This distinguishes an h^2 number from a likelihood ratio that might be used in epidemiology or a concordance number resulting from a probandwise comparison study. In both these cases the numbers can be applied directly to individuals as probability estimates (Sternberg & Grigorenko, 1999). In general, then, considerations of meaning preclude inference from a population's variance for a trait to the trait's causation in any individuals in the population.

Heritability, Phenylketonuria, and Modifiability

A dramatic example of the failure of h^2 to predict ease or difficulty of modifying traits is the case of phenylketonuria (PKU). PKU is a single-gene trait that is inherited in a recessive fashion that inhibits the breakdown of phenylalanine in the blood and tissues. The results of PKU include mental retardation, microencephaly, and growth problems. Despite the fact that PV for these PKU *sequelae* in a population will be almost entirely explained by GV, the condition is modified easily (the phenotypic expression altered) by a low protein childhood diet. This widely cited case (see, for example, Rowe, 1994) is an example of very high h^2 for a trait that is very easily modified.

More of h^2's Failure to Predict Immutability: Height and IQ

Other examples are familiar to those who have spent time in the behavior genetic literature. They show that high h^2 for a trait in a population does not preclude large-scale *mean changes* for that trait in that population, as opposed to changes within any individual. Body height, despite its h^2 of .80, increased significantly in the last half of the twentieth century. In Europe the increase has been about 1 cm per decade in the twentieth century. These average increases are similar among European countries, though the differences in mean height between countries remain, the lowest being in Italy and the greatest in the Netherlands (Silventoinen et. al., 2003).

We have seen that the relatively high h^2 of happiness set points has led some researchers to question the feasibility of success in interventions. The relatively high h^2 of IQ has led others to question the value of interventions to raise IQ. Jensen (1969) is the most well-known figure on this issue. It is well established that the IQs of children adopted early in life correlate strongly with their biological mothers and weakly, if at all, with their adoptive mothers—but, interestingly, these data do not imply that adopted children's IQs are similar to their biological

mothers' IQs or that adoptive environments play no role in modifying adoptees' IQs. In fact, there seems to be an IQ "bonus" that the adoptive child receives, especially when the adoptive environment exceeds the socioeconomic status of its biological counterpart (Maccoby, 2002). In a French study of children adopted at a mean age of 5 years, those who entered families with low socioeconomic status gained an average of 8 IQ points while those who entered high socioeconomic status families gained an average of 19.5 IQ points (Maccoby, 2002). It is also the case that when children are adopted into a higher socioeconomic status their IQs will exceed those of their biological parents.

To illustrate how a lack of parent–child similarity can coexist with a parent–child correlation (and thus high h^2) Ariew (1996, p. 24) provided the following example which, while hypothetical, is not far from representing the facts of the issue. Consider the following table of IQ scores.

Birth Parent IQ	**Child IQ**	**Adoptive Parent IQ**
90	110	118
92	112	114
94	114	110
96	116	120
98	118	112
100	120	116
Population Mean	**Child IQ**	**Adoptive Parent IQ**
95	115	115

In this case the variation in IQ for children is accounted for by variation in IQ of biological parents since the IQ rank order of the children correlates perfectly with the biological parent and not at all with the adopting parent. However, the results also show that IQ is highly sensitive to environmental changes in that the children gained 20 IQ points over their biological parents. High h^2 in this case is coupled with high environmental sensitivity and so does not preclude environmental effects.

Against the idea that high h^2 does not imply low environmental effects one might make the following argument, for which I am grateful to an anonymous referee:

> Suppose over a very wide range of environments. . .h^2 is close to 1. Fixing genotype greatly reduces phenotypic variance. One might suppose this gives us some inductive evidence that further variations in the environment will not significantly increase phenotypic variance.

In this argument, under some conditions high h^2 might warrant predictions of continued low environmental influence under differing environmental conditions. Specifically, the argument is that as we enumerate more instances of new environmental variations coupled with fixed GV resulting in stable PV, the probability rises that the future will yield the same results. There are nits to pick with this, but let's say it is so. The argument does not affect the point being made

in this article. It is not an example in which we conclude from a trait's h^2 for a population anything about the modifiability of the trait in an individual of the population or about the modifiability of the mean trait level in that population. Rather, it is a case in which an inference about a future variance is drawn from data about a set of previous variances.

Finally, a substantial h^2 level for a trait in a population does not imply that the population mean of that trait cannot be modified by changing environments. The reported h^2 of IQ ranges between .51 and .80, yet the phenomenon known as "The Flynn Effect" indicates that mean IQs have significantly increased. Flynn refers to "massive shifts" in the last 40 to 50 years, as much as 3 points per decade (Flynn 1987; Flynn & Dickens, 2001). This is the same phenomenon found in data from body height (Silventoinen et. al., 2003). Both are cases in which high heritability coexists peacefully with environmentally-induced change in population means for a trait.

Heritability and Degrees of Innateness

It may still be thought that there must be some causal connection between the h^2 of a trait in a population and the innateness of the trait in the individuals of that population. One might try to argue that although the h^2 of .49 of introversion cannot be specifically applied to individuals *as an h^2 number*, the .49 does imply something about the degree to which an individual's introversion in that population is influenced by genes as opposed to the environment. Perhaps it implies that the genetic influence or level of innateness within individuals in the population is greater than if the h^2 had been found to be .19. As sensible as this might seem, the idea that there is a correlation between h^2 levels for a trait in a population and the degree of innateness of that trait in the population's members is a blind alley.

Meanings of Innateness

Before providing arguments for this let's look at the idea of innateness. Ariew (1996) provides a good example:

> . . .female mallards raised to reproductive age by pintail ducks show no sexual affinity for the pintail drakes. But upon seeing a male mallard for the first time, the female immediately engages in the sexual courtship behavior particular to its species. (p. 21)

This example appeals to one's linguistic intuitions as a clear instance of "innate," and we could no doubt think of many equally good examples. It remains difficult, however, to provide a general definition of innateness (Samuels, 2004). Explications of innateness as unlearned or present at birth are subject to numerous objections. The idea of "determined by the genes" as equivalent to innate is tempting, but it fails because of ambiguities about the word "determined." For example, if "determined" implies sufficient conditions, then no trait is so

determined, and if determined implies merely necessary conditions, then all traits are determined.

Samuels (2004) argues, correctly I believe, that there can be no definitional account of innateness that satisfies all uses of the term. This does not preclude, however, some clarifying points. The female mallard's innate behavior is certainly not independent of environmental influence. Every trait requires a myriad of environmental conditions to stimulate and maintain it. The innateness of the mallard's behavior can be thought of as the degree to which its genotype (1) restricts the set of expressions of the mating behavior that develops (e.g., "yes" to mallards and "no" to a myriad of others) and (2) extends the range of environments in which this highly prescribed behavior is expressed (e.g., even to environments that contain no modeling behaviors). The more highly innate a trait, the narrower its set of expressions is and the wider the environmental range in which those expressions take place (this is close to Ariew's analysis of innateness as canalization [1996]). Put in this way, the thesis is that h^2 measures of the contribution in a population of GV to trait variance are not predictive of levels of innateness. Arguments for this thesis are as follows:

1) Consider a hypothetical example of a population of Swedish adult males in which all are cloned from the same source. The h^2 of body height in such a population would be (GV ÷ PV = 0), since the numerator of the fraction is 0 (this is suggested by a different example from Block & Dworkin, 1976). It is certainly false that an individual's height in this population reflects zero genetic influence.

2) Consider a sample (S) of unrelated Swedish adult males (S^U) raised apart and an equally large sample of Swedish male identical twin pairs (S^I) raised apart. The heritability of height for S^U is:

$$h^2S^U = GVS^U \div [GVS^U + EVS^U]$$

The heritability of height for S^I, where GVS^I is $GVS^U/2$, is:

$$h^2S^I = GVS^U/2 \div [GVS^U/2 + EVS^I]$$

Assuming that $EVS^U = EVS^I$, then

$$h^2S^U > h^2S^I .$$

This conclusion makes sense for the population statistic "heritability." When the GV in a population decreases while the EV is constant, a greater portion of a trait's variance in the population will be accounted for by EV—but the differences in h^2 numbers between the two populations will not correspond in any way to degrees of innateness. There is no reason to think that by reducing the GV for a

trait within a population one would in any way change the role of genes for that trait in any individual in that population.

In the example above h^2 was decreased by decreasing GV. One could also decrease h^2 by increasing EV. We could easily reduce the role of the GV for height among Swedish adult males by varying the diet of a randomly selected half of the population. Since $h^2 = GV/PV$ where $PV = GV + EV$, h^2 is inversely related to EV, so such an increase in EV would decrease h^2. This decrease in h^2 does not in any way predict or correlate with a decrease in the comparative influence of genes versus the environment for any individual member of the population. These cases show that the h^2 of a trait in a population fails as a predictor of the comparative roles of the genes or the environment for that trait in any individual of the population, and so does not correlate with levels of innateness.

There might yet be a nagging thought that genotype or environment could only explain individual *differences* for some trait (T) in some population (P) if the genotype or the environment was a causal factor of levels of T in the individuals of P. Of course, it's trivially true that both are causal factors in all traits of individuals, but is it true that if h^2 is high for T in P then hereditary influences must be high for T in the individuals of P? The answer is no. Fred and Ted are identical twins with IQs of 120 and 75, respectively. Ted was brain damaged by a forceps delivery, a procedure that Fred did not have. Any measure of the degree to which their respective IQs were influenced by genotype would put the influence lower for Ted than for Fred, Ted having been victim of such a dramatic environmental factor. In fact, although the specific brain damaging event would account for most of the difference in their IQs, it accounts for none of Fred's IQ. More generally, the degree to which *population differences* for a trait are genetic or environmental cannot be used to predict the degree to which the trait in an individual is genetic or environmental. The conclusion is, then, that h^2 cannot predict innateness.

Innateness and Modifiability

High h^2 does not predict innateness. Nor does high h^2 predict difficulty of modification. Nor does innateness predict difficulty of modification, as the PKU example suggests. It might well be that sexual orientation is innate and that it is not environmentally modifiable, but the latter does not follow from the former. Innateness was explicated previously by the narrowness of the trait's expressions and the width of the environments in which these expressions occur; however, neither of these implies difficulty of modifiability. The difficulty of modifying a trait is related not to the number of environments in which the trait is expressed but to the access to the environmental manipulations that will alter the trait. The gene for PKU guarantees its expression in almost all possible environments, yet the one—or one of the few—environmental manipulation that prevents its expression (i.e., a low-protein childhood diet) is quite easily accessible. Similarly, while the phenotype "having two eyes" fits very well the two conditions of a high level of innateness, the manipulation necessary to reduce the number of eyes in a person, as gruesome as the thought of plucking out an eye might be, is quite simple to

achieve. Think of other examples in which highly genetic-based conditions are rather easily modified—cleft palate, crossed eyes, port wine birthmarks, and freckles. These examples need not involve genetic alteration. Even behavioral traits such a human warmth and agreeableness can be altered easily by traumatic or persistently negative influences. On the other hand, shyness, skin color, and sexual orientation seem far more difficult to alter. The point is not to argue that genetic-based traits are as modifiable as environment-based traits, as if long lists of each have been tested. The point is that knowing only the level of a trait's innate basis is not a predictor of it being easy or difficult to modify.

Innateness and Norms of Reaction

This conclusion is reinforced by focusing upon norms of reaction, a different method of disentangling nature and nurture than heritability analysis. Picture a line graph in which the y axis represents levels of some environmental variable (e.g., saturated fat in a human diet) and the x axis represents a trait level (e.g., blood low-density lipoprotein [LDL]). The plotted lines ("norms of reaction") for separate people will be very different. More to the point, we should not expect that trait levels will be linearly related to environmental levels. There will be lines with positive slopes, negative slopes, positive slopes that flatten at different thresholds, and flat lines that become positive or negative at different thresholds. There will be lines for different people that begin at different places at low levels of saturated fat and converge at higher levels—and of course we might expect similarly diverse norms of reaction (lines) for the complex human environments and traits such as the following:

Environmental Level	***Trait Level***
Academic enrichment	Academic achievement
Cultural enrichment	IQ
Parental affection	Personal agreeableness
Competitive sports	Personal competitiveness
Physical discipline	Aggressiveness

Each of these traits will have separate norms of reaction for each of its vast number of causally relevant[3] environmental variables. IQ, for instance, will have a differently shaped line for dietary protein than for cultural enrichment, and think of how distinct will be the dietary protein and IQ lines for PKU versus non-PKU children. The diversity and nonlinearity of norms of reaction have important implications (first discussed by Lewontin, 1974). It is very unlikely for lines to be linear for complex, interesting variables. One implication of this for the discussion of modifiability is that individual human traits typically result from patterns of gene and environmental effects. One does not inherit a stable phenotype, an IQ of

[3] One must say "causally relevant" here since every expression will be flat with respect to an infinite number of variables. Cherise's LDL will be flat with respect to growing levels of clutter in Maynard's office.

110, or a score on a measure of agreeableness for all environments. What one inherits for any trait is more like a norm of reaction for every variable that is causally relevant to the trait.

Focusing upon norms of reaction and upon their diversity and non-linearity puts the environmental modifiability of traits into the foreground. It is not surprising that the impression left by the behavioral genetic analysis of variance has been the opposite. Measures of PV for a trait in a population are aggregations of all the diverse, idiosyncratic norms of reactions. The method of measuring h^2 necessarily suppresses the individual sensitivities to diverse environments. Everyone who has observed children closely knows that each child responds differently, for example, to discipline, that each child has a different threshold beyond which discipline is counterproductive, and that some children thrive in the home with very little discipline and others only with a great deal. Heritability measures neither contradict nor elucidate these differences. The h^2 of .60 for authoritarianism reveals none of this response variety and, as such, it is not helpful to the parent who wonders about the ease or difficulty of modifying his or her child's behavior. In the case of Jim's introversion, the h^2 of .49 gives us no information about Jim's particular norm of reaction for the variable of social interaction, what environmental variables might alter his comfort level in social situations, or even how innate the trait is. Nor would any of this be different if the h^2 for the trait measured .19 or .79.

Even further, the degree of innateness of some trait relative to one environmental variable (e.g., as evidenced by relatively flat norms of reaction under diverse environments) does not predict the line for another variable. A wide range of variables for which the line is relatively flat, providing evidence for a high degree of innateness, does not rule out an easily accessible variable that will drastically alter the typical norm of reaction.

Finally, easily constructed examples can show us that traits attributed to genetics are not inherently more difficult to modify than traits attributed to the environment (i.e., the fact that a trait A is more genetically based than trait B supplies no evidence that A will be more difficult to modify). Suppose that 9-year-old Henry is very shy, as was his father when a youngster, and has been that way since his earliest days. Suppose that 9-year-old William was extroverted until age 7 years, when he was severely abused and is now very shy. Knowing that Henry's shyness is "innate" while William's is "environmental" gives us no strong reason to think either that Henry's or William's shyness would be more readily modified through some program of behavior therapy.

Conclusions

Some human traits in some individuals are easily modifiable by accessible (including legal and ethical) means and some are not. The factors that determine modifiability are extremely varied and need to be identified empirically. The first conclusion of this article is that measures of heritability as employed in behavioral genetics do not predict how difficult or easy it will be to modify any particular trait

for any particular individual. The second conclusion is that measures of heritability do not predict how deeply innate a trait might be for some individual. The third conclusion is that levels of innateness of traits of individuals do not predict the ease or difficulty of altering those traits through environmental manipulation. These three conclusions suggest that the disentangling of nature and nurture does not bear upon the ease or difficulty of individual human change.

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