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HAPPINESS: THE POTENTIAL POWER OF ENVIRONMENT

ABSTRACT: Happiness and Genes

Many scientists have argued that they can determine to what extent human happiness levels are controlled by genes by comparing the average happiness levels of identical twins raised apart. If we discover that identical twins raised apart tend to be more hedonically similar than fraternal twins raised apart, this is interpreted as evidence for the thesis that genes have a strong influence on our happiness levels. If identical twins are hedonically dissimilar, as dissimilar as fraternal twins raised apart, this has been taken as evidence for the thesis that happiness levels are determined in large part by the environment. I shall show that that these interpretations of these studies rely on a set of false assumptions. There is no good evidence our genes determine how happy we can be.

1. INTRODUCTION: HEDONIC POSSIBILITIES AND HERITABILITY STATISTICS

Many scientists have argued that they can determine to what extent human happiness levels are controlled by genes by comparing the average happiness levels of identical twins raised apart. If we discover that identical twins raised apart tend to be more hedonically similar

than fraternal twins raised apart, this is interpreted as evidence for the thesis that genes have a strong influence on our happiness levels. If identical twins are hedonically dissimilar, as dissimilar as fraternal twins raised apart, this has been taken as evidence for the thesis that happiness levels are determined in large part by the environment. I shall show that that these interpretations of these studies rely on a set of false assumptions. There is no good evidence our genes determine how happy we can be.

2. HERITABILITY, HAPPINESS AND TWIN STUDIES

Heritability is “the proportion of phenotypic variance attributable to genotypic variance in a population” (Downes 2009). The heritability of a particular trait is usually estimated by comparing the phenotypic traits of twins. If we needed to figure out the heritability of skin color, for example, we would compare the skin color of monozygotic twins to the skin color of dizygotic twins. If the skin color of pairs of monozygotic twins tends to be as dissimilar as pairs of dizygotic twins, the heritability of skin color is low. It is assumed that we see similar levels of variation in pairs of monozygotic twins and pairs of dizygotic twins because skin color is primarily determined by environmental factors. If the heritability of skin color is high, the skin color of pairs of monozygotic twins will tend to be more similar than the skin color of pairs of dizygotic twins. In this case, it is assumed that there is less variation in pairs of monozygotic twins than in pairs of dizygotic twins because of genetic factors.

Many psychologists think that twin studies can similarly be used to quantify the heritability of happiness, too. In the 2007 book, “What You Can Change and What You Can’t” Martin Seligman explains the quantification process:

Identical twins reared apart have the same genes, but they grow up in vastly different environments. If they are similar for some personality trait, it must be heritable and not learned. The study of identical twins reared apart is the best way to untangle the effects of child rearing from the effects of genetics. Indeed, if you want to attach a number for degree of heritability, simply take the correlation for

identical twins reared apart: When the correlation is 1.00, the trait is completely determined genetically; when it is lower, say .50, this means that the trait is half genetic and half nongenetic in origin (p. 76).

Seligman is assuming that we can determine how much of an influence genes have over our happiness levels by comparing the phenotypic traits of identical twins raised together to the phenotypic traits of identical twins raised apart. Seligman, and many other psychologists, think that the “degree of heritability” of average happiness levels is an excellent way to determine how much control we can have over our happiness.

It should be pointed out that in the work on heritability, happiness and associated states are usually referred to as ‘positive affect’ which is often discussed in terms of positive emotionality (PE), well-being (WB) or subjective well-being (SWB). ‘Positive affect’ refers to positive feelings, moods and attitudes. These feelings, moods and attitudes are considered positive because they are normally considered pleasant by those who experience them. Examples of positive affective states include feeling happy, being in a good mood and being proud or optimistic. Over time people experience a range of pleasant emotions, moods and attitudes and they express these emotions, moods and attitudes in a variety of ways, thus, the intensity and frequency of positive affect can be measured in variety of ways. One popular instrument is the Well-Being scale of the Multidimensional Personality Questionnaire, a self-rating questionnaire that measures the disposition to feel good (Tellegen 1982; Tellegen & Walker 1994). Positive and negative affect (unpleasant emotions, moods and attitudes are referred to as negative affect) can co-occur, thus, it’s important that studies take into account negative affect (Larsen et al. 2001). The Stress Reaction scale does just that. In the twin studies I discuss below, the researchers “estimated SWB simply as the difference between the two scores (i.e., $SWB = WB - SR$)” (Lykken & Tellegen 1996, p. 187). According to those that conducted the studies that utilize these fairly uncontroversial methods for measuring affect, genes have an insurmountable influence on how much happiness we are capable of experiencing.

Twin studies have shown that identical twins’ average levels of positive affect tend to be closer to each other than fraternal twins’ aver-

age levels of positive affect, which are about as close to each other as strangers, regardless of whether the twins were raised together or apart (Lykken & Tellegen 1996). In one such study, Lykken and Tellegen asked 128 pairs of identical and fraternal twins around the age of 20, born in Minnesota from 1936 to 1955 how happy they were, on average, compared to other people the same age, then asked them the same question 10 years later. When the twins were retested they correlated one twin’s happiness score with the other twin’s score 10 years later. For the 48 pairs of dizygotic twins, this cross-twin, cross-time correlation was .08; for the 79 monozygotic pairs of twins, it equaled .40 (Lykken & Tellegen 1996, p. 188). This study built on an earlier study of 217 monozygotic and 114 dizygotic pairs of middle-aged Minnesota Registry twins, plus 44 monozygotic and 27 dizygotic pairs of twins who were separated in infancy and reared apart. According to that study, the heritability of levels of positive affect was .48. When 26 pairs of the middle-aged twins reared apart were retested on the MPQ by mail about 4 1/2 years after their first testing they discovered that the heritability of the stable component of well-being was about .80. Tellegen believes that “unshared environmental effects would then account for the remaining 20% of the variance in the stable component of happiness” (Lykken & Tellegen 1996, p. 188). In another study, Lykken and Tellegen discovered that the happiness correlation between 647 pairs of monozygotic twins reared together turned out to be .44. The correlation between 733 dizygotic twins reared together was .08. The correlation between 75 pairs of monozygotic twins reared apart was .52. The correlation between 36 dizygotic twins reared apart turned out to be -.02. Lykken and Tellegen argue that the consistent similarities between monozygotic twins in all of these studies, “suggests that the stable component of well-being (i.e., trait happiness) is largely determined genetically.” Lykken and Tellegen come to the conclusion that:

Because the [dizygotic twins raised together] values are so small, and the monozygotic twins raised together] correlations are not larger than the [monozygotic twins raised apart] values, we can conclude that the effect of shared home environment on SWB were negligible after the twins reached middle age. This conclusion means that the vari-

ance in adult happiness is determined about equally by genetic factors and by the effects of experiences unique to each individual (1996, p. 188–9).

The common interpretation of these influential studies is that since the chances that sets of identical twins (raised together or apart) will have similar levels of positive affect is much greater than the chances that sets of fraternal twins (raised together or apart) will have similar levels of positive affect, genetic factors must have a profound influence on levels of positive affect.¹ The evidence, it is assumed, suggests that we have limited control over our happiness levels. In the words of Lykken and Tellegen, “trying to be happier is as futile as trying to be taller and therefore counterproductive” (pg. 189).

This interpretation of the data is still extremely popular. For instance, in her 2008 book, “The How of Happiness”, Sonja Lyubomirsky claims that “your genetically determined predisposition for happiness (or unhappiness) accounts for 50 percent of the differences between you and everyone else” (2008, p. 53). Lyubomirsky derived this number from heritability studies:

no matter which we look at it, the empirical data from the Happiness Twins study led to the conclusion that the genetic basis for happiness is strong—very strong. It appears that each of us is born with a happiness set point, a characteristic potential for happiness throughout our lives. The magnitude of that set point may originate from the sunny maternal side of our family or our depressive paternal side, or roughly equally from both; we’ll never know. The essential point is that even if major life changes, like a new relationship or a car accident, might push our happiness level up or down, we tend to revert to the genetically determined set point (2008, p. 57).

Lyubomirsky recognizes that levels of positive affect are flexible, but she believes that the limits of our flexibility are revealed by heritability studies. By her lights, “some of us simply possess higher set points for happiness, more cheerful dispositions, and higher potentials for well-being” (2008, p. 61).

3. THE POWER OF THE ENVIRONMENT

These studies depend, in part, on the assumption that identical twins raised apart are raised in different environments. However, I think that a closer examination of the studies reveals that we don’t really know whether average levels of positive affect are governed by genetic factors. This is because monozygotic twins are similar to each other and different from others in a variety of ways. The ways identical twins are similar to each other and dissimilar from others might consistently elicit positive, happiness inducing reactions in different environments. The correlation between genetic variation and average positive affect levels might be caused by environmental factors.² It isn’t clear that identical twins tend to have similar levels of positive affect because of a happiness gene or happiness genes rather than the countless other things they have in common, things which aren’t controlled for in heritability studies, things which might create environmental similarities between identical twins and consistent affect differences between pairs of identical and pairs of fraternal twins. We can’t determine the extent to which heritability statistics derived from twin studies are indicative of the presence or absence of a special happiness gene or genes that directly cause happiness levels, or other genes that increase happiness indirectly, via the environment.

The psychologists I have mentioned here use these studies to determine what we can change and what we can’t. Even if it turns out that happiness is highly heritable this doesn’t imply your happiness levels can’t be changed. If you change the environment, you can change happiness levels (and, in certain cases heritability might even remain high or become higher). It has been shown that many traits which are clearly plastic—academic performance, occupational status and toy number, to name a few—turn out to be highly heritable according to heritability studies (Plomin 1990). Presumably, football (of the American variety) playing is highly heritable (in the states, at least) even though this behavior isn’t determined by our genes. Males are socialized to play football thus, in this society, there is a correlation between having a Y chromosome and the tendency to play football. This trait would exhibit zero heritability in a society which didn’t encourage those with a Y chromosome to play football (or a country without American footballs). When football playing differences are caused by

chromosomal differences it's via extrasomatic pathways: males are encouraged to play football because they are males and, of course, males have a chromosome. Even though football playing differences are extremely heritable in certain circumstances, they aren't genetically determined in any interesting sense. Still, certain highly heritable traits, traits that are more or less 'directly' caused by genes via internal biochemical processes relatively unaffected by the environment are difficult (or impossible) to change, but we don't learn of this inflexibility by doing heritability studies.

Let's pretend that we actually discovered a gene or set of genes that guides brain development in a way that predisposes certain groups to be happier than other groups in certain environments. Does this mean we do not have control over our happiness levels? Absolutely not. Insofar as we have control of our environments, we have control over our happiness levels. We are just beginning to learn of the hedonic effects of various sources of environmental variation. Socioeconomic factors can have an effect on the happiness of a particular group. If we took a look at the heritability of average affect levels of African Americans thirty five years ago, we would have noticed that variation in average affect levels correlated with different allele frequencies: we would have seen that African Americans were less happy on average than other Americans. If we had reasoned then as many prominent positive psychologists do now, we would have made the assumption that the average affect differences we observed between African Americans and other Americans were because of the genetic differences between African Americans and other Americans rather than differences in the environment (this is especially worrisome given the recent fascination with differences in national happiness levels). If we subscribed to the same assumptions positive psychologists do now, we would have assumed that there was a happiness gene or genes and we would have been surprised to see that the average affect levels of these groups have changed. The average levels of positive affect among African Americans have increased over the past thirty five years (Stevenson & Wolfers 2010). Presumably this is because the rights of African Americans have been better respected in society. In any case, since the 70's, the average affect levels of this group have shifted. Environmental factors provide the best explanation of this shift, and these are factors that haven't

been controlled for in heritability studies.

4. HOW TO INVESTIGATE HEDONIC POSSIBILITIES PROPERLY: NORMS OF REACTION

The heritability of a trait doesn't reveal how much control we have over our happiness. However, certain genes have some sort of influence on our happiness in certain sorts of environments. How could we figure this out? We could start, at least, by constructing a norm of reaction chart—a graph predicting what phenotypic traits we should expect when a genotype is subjected to specific environmental factors (Lewontin 1974). Reaction norms reveal the plasticity of the phenotype. In most cases, a norm of reaction chart provides a less misleading picture of the relations between genes, environment and phenotypic traits.³ Consider the case of phenylketonuria:

PKU stems from a mutation in a single gene on chromosome 12 and, without treatment, leads to brain damage, resulting in mental retardation and premature death. PKU is said to have a heritability of 1, because it is entirely genetically determined. But this doesn't mean that an infant born with the gene that causes PKU is doomed to its lethal effects. If the parents ensure that the infant's diet is free of an amino acid called phenylalanine, which is found in such common foods as eggs, milk, bananas, and NutraSweet, the brain damage may be entirely prevented. It's important to note that the infant's genetic endowment doesn't change—she will always carry the mutant gene—but the expression of her genetic endowment can change (Lyubomirsky 2008, p. 58).

Lyubomirsky thinks that we have control over whether an individual manifests the symptoms of PKU. We don't have control over whether we inherit the allele which causes PKU. However, she argues that the heritability of PKU is 1. This depends on environmental factors. The heritability of PKU will be low in environments where babies are regularly tested for PKU and treated. It seems that Lyubomirsky is confusing heritability with the inheritance of the gene or genes that cause a trait. Genotypes are inherited, phenotypic traits aren't.

This mistake is worth correcting because many influential psychologists go on to use twin studies to quantify how much control we have over our happiness levels and this information is relayed to the public via the media. This may lead people to believe they are helpless to a certain extent, that they have less control over their lives than they think they have and less control over their lives than they might actually have. In order to determine just how much control we have over our happiness levels, we need to continue to carry out further experiments. We need to focus on manipulating phenotypic traits by tampering with non-genetic factors, seeing what environmental changes lead to higher average levels of affect. In other words, we should try to figure out, scientifically, how happy we can be.

Notes

¹It should be noted that these results have been replicated recently (Nes et al., 2006). However, Johnson, McGue and Krueger argue that only 38% of the stable variance in well-being is heritable (2005). I shall argue this discrepancy isn't surprising: it should be expected given the nature of heritability statistics.

²I shall set aside the fact that identical twins share a prenatal environment for the time being.

³Though it is less misleading than a heritability estimate, it is often oversimplified, especially when we take into account the combined effects of pleiotropy and epistasis.

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