Are DSM Psychiatric Disorders "Heritable"?

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A key concept in psychiatry and behavioral genetics is *heritability*. The concept was developed in the 1940s as a tool to help predict the results of selective breeding programs of farm animals, such as milk production in cows. Since the 1960s, it has been extended by behavioral researchers and others as an indicator of the strength or magnitude of genetic influences on various psychiatric disorders, and behavioral characteristics such as IQ and personality.[1]

A heritability estimate is presented as a percentage figure between 0% and 100%, or as a number between 0.0 and 1.0. Here I will describe the main issues surrounding heritability, while recognizing that for most people this "extraordinarily misunderstood" concept is difficult to comprehend and to articulate.[2]

"Heritability," according to psychiatry's psychiatric genetics subfield, "is usually expressed as the proportion of trait variance attributable to additive genetic factors."[3] The key word here is "variance" (variation), which in this context refers to how psychiatric disorders and behavioral characteristics are distributed in a population. In human genetic research, heritability is said to measure the extent to which variation among people is explained (accounted for) by genetic

influences. Leading behavioral geneticist Robert Plomin and colleagues believe that assessing the causes of variation allows researchers to estimate "how much genetics contributes to a trait," and allows them to quantify the "relative importance" of genetic and environmental influences.[4]

Heritability estimates are derived from correlations among relatives, which include mainly twins, but also adoptees, siblings, and other types of family relationships. Heritability estimates are designed to assess the causes of variation in a population. They do not address developmental processes that cause individuals to develop various characteristics and abilities.[5]

Leaders of psychiatric genetics believe that heritability estimates measure "the degree to which the vulnerability to develop a disorder is influenced by genes."[6] They believe that 0% heritability indicates that the vulnerability to develop a psychiatric disorder is "due entirely to environmental influences," whereas 100% heritability "indicates that the liability can be explained entirely by genes."[7] Estimates usually fall between these two extremes, and researchers argue that as heritability rises, so does the strength of the genetic influence. Psychiatric disorders in the 20-40% range are seen as having "moderate heritability," in the 40-60% range as having "moderately high heritability," in the 60-80% range as having "high heritability," and in the 90-100% range as having "very high heritability."[8]

Heritability estimates in psychiatry are often calculated from twin study data by doubling the reared-together MZ (monozygotic, identical) versus DZ (dizygotic, fraternal) correlation or concordance rate difference. (Twin pairs are *concordant* for a disorder when both members of the pair are diagnosed with the same disorder. Twin pairs are *discordant* when only one member is diagnosed with the disorder.) Using basic twin method reared-together twin data, if a sample of MZ pairs shows 50% concordance for schizophrenia, and the DZ sample shows 10% concordance, psychiatric geneticists would double the difference and estimate the "heritability of schizophrenia" in the sample as 80%.

Researchers frequently use "biometrical model fitting" statistical analyses to calculate heritability, which attempt to test the "fit" between a model of genetic and environmental relatedness against the observed data. All heritability estimates based on twin method MZ-DZ comparisons depend on the validity of the method's MZ-DZ "equal environment assumption," which critics have convincingly argued is not supported by the evidence.[9] Model fitting analyses are based on an additional set of very questionable assumptions about people, society, and genetics.

The authors of mainstream psychiatric and psychiatric genetic publications believe that the major psychiatric disorders are "moderately to highly heritable." Examples of pooled

heritability estimates for various disorders include schizophrenia 84%, bipolar disorder 84%, ADHD 75%, borderline personality disorder 69%, panic disorder 43%, PTSD 38%, major depression 37%, and social phobia 25%.[10] The remaining portion (100% minus heritability) is attributed mainly to environmental influences, usually divided into "shared" and "non-shared" components.

Criticism

As developmental psychologist David Moore wrote in 2013, the usefulness and validity of heritability estimates has "been the subject of unrelenting criticism from philosophers, biologists, and psychologists for nearly four decades."[11] The main points critics usually make include:

- Heritability estimates *do not* measure the strength of genetic influences on psychiatric disorders and behavioral characteristics, nor do they assess the relative importance of genetic and environmental influences, and are misleading and potentially harmful when they are presented this way.

- Heritability estimates are based on research methods that (to varying degrees) are unable to disentangle the potential influences of genes and environment on behavior, such as family, twin, and adoption studies.

- Although heritability estimates are based on the assumption that genetic and environmental factors are separate (additive) and do not interact, they clearly do interact.

- Even when heritability is high, or even when it is 100%, a simple environmental change or intervention can have an important preventative or curative impact. In most cases, therefore, heritability estimates tell us nothing about the potential effectiveness or non-effectiveness of an environmental intervention, or to what extent a psychological characteristic is or is not changeable.

- Heritability is the property of a population, not of the characteristic or disorder itself.

- Because it is a population statistic, heritability does not describe the importance of genetic factors as they relate to an individual.[12]

- Heritability estimates apply only to a specific population, at a specific time, and in a specific environment. Estimates can change substantially under different environmental conditions.

- High heritability *within* different populations does not mean that genetic differences exist *between* different populations (such as different ethnic groups).

- Recent findings that gene expression switches on and off "epigenetically" in response to environmental events and challenges provide additional important evidence that genetic and environmental influences are not additive, but are instead interactive.[13]

- The ongoing decades-old failure to discover genes that play a role in causing psychiatric disorders and behavioral characteristics (as opposed to merely being "associated" with them) provides additional evidence against the "heritability" of these disorders and characteristics.

Gene-Environment Interaction

A leading group of psychiatric genetic investigators wrote that heritability estimates "measure the degree to which genetic factors influence variability in the manifestation of the phenotype [disorder]," and that a disorder's variability "is presumed to arise from two independent factors: genetic variability … and environmental variability."[14] According to behavioral genetic twin researcher Nancy Segal, model fitting analyses that produce heritability estimates assume that "genetic and environmental effects are independent from each other, and [that] genetic and environmental effects combine additively."[15]

Many critics, however, have argued that genetic and environmental factors *are not* independent from each other,

and that gene–environment interaction reduces or even invalidates heritability estimates.[16] As the population geneticist Richard Lewontin wrote, “If these causes ‘interact’ in any generally accepted meaning of the word, it becomes conceptually impossible to assign quantitative values to the causes of that individual event. Only if the causes are utterly independent could we do so.”[17]

According to developmental researcher Michael Meaney, “research in biology reveals that the genome cannot possibly operate independent of its environmental context.”[18] Simple examples of potential gene–environment interactions include physically attractive people who experience much different responses from their social environments than do physically unattractive people, and children with musical talent who are sent to an expensive music academy to maximize their talent.

Flamingos provide an example from nature of the fallacy of partitioning genetic and environmental factors into separate additive influences. Flamingos become pink by ingesting a diet of shrimp and other foods rich in alpha and beta carotenoid pigments. Those whose diet does not include carotenoid pigments do not become pink. Flamingos are therefore born with a genetic potential to have pink feathers, but require a specific diet to achieve this potential.

Psychiatrist Michael Rutter cited flamingos as an example of gene-environment interaction. He noted that both genes and

environment play a crucial role in the ability of flamingos to turn pink, and that "you could feed seagulls forever on the same diet and they would never turn pink." He concluded that "it would make no sense to say that flamingos' color was 50 percent due to genes and 50 percent due to diet. It is 100 percent due to genes (which *have* to be present) and 100 percent due to the environmental diet (which *has* to be present)."[19]

Even if, for the sake of argument only, we allow that DSM psychiatric categories are valid discrete disorders with an underlying genetic component, like flamingo color it "makes no sense" to say, for example, that schizophrenia is 84% genetic and 16% environmental, that panic disorder is 43% genetic and 57% environmental, or that major depression is 37% genetic and 63% environmental. In such cases both genetic and environmental factors are essential interacting influences whose contributions jointly, as opposed to additively, combine to produce the observed behavioral characteristic (phenotype).

Heritability ≠ Inherited

Some writers have noted the common confusion between two different uses of the word "heritability." We have seen that the technical meaning of heritability refers to the proportion of individual differences in a population that can be attributed to genetic factors. In contrast, people commonly yet mistakenly

use the word “heritable” to mean “inherited,” or “hereditary.”[20]

According to the critical behavioral genetic researcher Jerry Hirsch, “heritability” and “heredity” are “two entirely different concepts that have been hopelessly conflated.” Because they sound alike, he wrote, “when we hear one of the two words, automatically we think the other.”[21] As Hirsch repeatedly pointed out, a heritability estimate is not a “nature-nurture ratio” of the relative contributions of genes and environment. The author of *The Mirage of a Space between Nature and Nurture*, Evelyn Fox Keller, found it unfortunate that “authors and readers alike routinely slide from one meaning [of heritability] to the other, wreaking havoc on the ways in which legitimate scientific measurements are interpreted.”[22]

Variation ≠ Cause

Lewontin showed long ago that a “trait can have a heritability of 1.0 [100%] in a population at some time, yet could be completely altered in the future by a simple environmental change.”[23] An example is phenylketonuria (PKU), a genetic disorder of metabolism that causes intellectual disability. Although PKU is a “highly heritable” single gene disorder, the administration of a low phenylalanine diet to the at-risk infant during a critical developmental period prevents PKU from causing intellectual disability.

The example of favism (Glucose-6-phosphate dehydrogenase deficiency) illustrates the fallacy of using heritability estimates to assess "how much" genes influence behavior ("IQ," "personality," psychiatric conditions, and so on). Favism is caused by an inherited deficiency of glucose-6-phosphate. The predisposing gene is located on the X chromosome. When the carrier eats fava (broad) beans or inhales fava bean pollen, favism appears. The disease is marked by the development of hemolytic anemia. In other words, "beans and genes" are both necessary for favism to appear.

Let's imagine "Country A," where all citizens (100%) carry the favism gene. In Country A, 15% of the citizens, all of whom of course carry the gene, are exposed to fava beans and subsequently develop favism. In Country A, because all citizens carry the gene, but only some were exposed to fava beans, all favism ***variation*** is caused by environmental factors (fava bean exposure or non-exposure). The "heritability of favism" in Country A, therefore, is 0% (0.0).

At the same time, it would be very mistaken to conclude that genes play no role in developing favism in Country A, or that the genetic influence is weak or irrelevant. A genetic predisposition is, in fact, a prerequisite for developing favism.

Now imagine "Country B," where all citizens (100%) eat a diet containing fava beans. In Country B, 15% of the citizens, all of whom of course eat a diet containing fava beans, carry the

favism gene and subsequently develop favism. In “Country B,” because all citizens are exposed to fava beans but only some carry the gene, all favism ***variation*** is caused by genetic factors (carrying or not carrying the gene). The “heritability of favism” in Country B, therefore, is 100% (1.0).

At the same time, it would be very mistaken to conclude that environmental factors play no role in developing favism in Country B, or that such factors are weak or irrelevant. Fava bean exposure is, in fact, a prerequisite for developing favism.

In the above examples, the “heritability of favism” simultaneously is 0% in Country A, and 100% in Country B, even though the causes of favism are the same in both countries. As we see, “heritability estimates” assess variation as opposed to cause, and do not indicate the strength or weakness of the genetic influence—or by implication the strength or weakness of the environmental influence.

As Moore concluded, “Because heritability statistics are about accounting for variation and not about causation, they do not actually reflect the strength of influence of genes on the development of a trait, even if it seems like they do.”[24]

Heritability and Psychiatric Disorders

Establishing the reliability and validity of a psychiatric disorder is a prerequisite for any study attempting to estimate

heritability. Reliability in psychiatry refers to the ability of psychiatrists to consistently agree on a diagnosis. A disorder must be valid in addition to being reliable. Validity refers to whether the concept actually exists as a true disorder. Many critics of psychiatry have argued that psychiatric disorders are not reliable or valid discrete illnesses, but rather describe people's varying psychological responses to having experienced adverse events and environments, or are socially disapproved behaviors that psychiatry labels as mental disorders.

In their 2013 work *Mad Science: Psychiatric Coercion, Diagnosis, and Drugs,* Stuart Kirk, Tomy Gomory, and David Cohen showed that there are serious reliability and validity problems in psychiatry, suggesting that research is impaired when it relies on diagnostic systems such as American psychiatry's Diagnostic and Statistical Manual of Mental Disorders (DSM) to diagnose people with similar problems.[25] A lack of reliability and validity has important implications for psychiatric genetic family, twin, and adoption studies—and accompanying heritability estimates—because many people diagnosed in these studies may not actually "have" the condition at all.[26]

The irrelevance and misleading nature of heritability estimates in psychiatry is seen in the example of autism. For many years, based mainly on the results of a few small twin studies, leading researchers variously described autism as showing "strong genetic determination,"[27] and as being "under a high degree of

genetic control."[28] Although the evidence in favor of genetics is surprisingly weak,[29] reviewers commonly estimate autism heritability at roughly 90% (0.9), based on Anthony Bailey and colleagues' 1995 twin study and review.[30]

Suppose a team of researchers conclusively proves that all children who eventually develop autism had eaten "Baby Delight Apricot Baby Food" between the ages of four and six months, and that further investigation had shown that the ingestion of a rare chemical found only in this brand of apricot baby food by genetically predisposed children, during this sensitive developmental period, caused autism. The government immediately removes Baby Delight Apricot Baby Food from the market, confiscates existing inventories of the product, and issues warnings to parents. What would happen to the rate of new autism diagnoses a few years later? The answer is that it would be reduced to virtually zero. Like PKU, presumed genetic factors appear to be "under a high degree of genetic control" (and difficult to change) only in the absence of (or denial of) identified environmental causes and triggers.

An example in the twin research literature illustrating this important point is found in Segal's description of a British reared-apart MZ pair who suffered from headaches and irritability, due to their shared allergy to foods containing gluten. Although medical researchers believe that gluten sensitivity is "strongly heritable,"[31] and that "genetic predisposition plays a key role,"[32] according to Segal one twin's

"health and spirit improved dramatically" upon "eliminating wheat from his diet," and his twin brother "agreed to make the same dietary changes when he returned home."[33]

As this example shows, an environmental intervention cured a "strongly heritable" condition in the same way as it would have cured a "weakly heritable" condition. This pair's story again shows that a heritability estimate does not indicate the strength or "relative importance" of the presumed genetic component, or the potential effectiveness or ineffectiveness of an environmental intervention.

It is not the task of critics to establish the "true heritability" of various psychiatric disorders and behavioral characteristics, or to show that heritability is zero. Instead, they must explain why the "heritability of psychiatric disorders and behavioral characteristics" concept itself is not valid, other than its use as a breeding predictor. Behavioral geneticist Douglas Wahlsten pointed out that "the only practical application of a heritability coefficient" is its original purpose of "predict[ing] the results of a program of selective breeding."[34] In human terms, this is called eugenics.

Why This Matters

The heritability statistic was developed in the mid-20th century as a means of predicting the results of selective breeding programs in agriculture. It *is not* an indicator of the degree of

genetic influence on psychiatric disorders. This holds true for all other areas of human behavior as well. As Moore and a colleague wrote, "The term 'heritability,' as it is used today in human behavioral genetics, is one of the most misleading in the history of science."[35]

"Heritability" and "inherited" have very different meanings, and heritability estimates are highly misleading because they do not indicate the relative magnitude of environmental and presumed genetic influences on behavior. Although researchers will continue to claim that various behavioral characteristics are influenced by genetic factors, assigning numerical heritability estimates to such claims in psychiatry and other behavioral science fields should be discontinued.

Notes:

[1] Bouchard, T. J., Jr., (2009), Genetic Influence on Human Intelligence (Spearman's g): How Much?, *Annals of Human Biology, 36,* 527-544, p. 527; Lush, J. L., (1949), Heritability of Quantitative Characteristics in Farm Animals, *Hereditas* (Suppl.), G. Bonnier & R. Larsson (Eds.), 356-375.

[2] Moore, D. S., (2008), Espousing Interactions and Fielding Reactions: Addressing Laypeople's Beliefs about Genetic Determinism, *Philosophical Psychology, 21,* 331-348, p. 338.

[3] Owen, M., J., & Williams, N. M., (2021), Explaining the Missing Heritability of Psychiatric Disorders, *World Psychiatry, 20,* 294-295, p. 294.

[4] Plomin et al., (2013), *Behavioral Genetics* (6th ed.), New York: Worth Publishers, pp. 86-87.

[5] For more on this point, see Gottlieb, G., (2003), On Making Behavioral Genetics Truly Developmental, *Human Development, 46,* 337-355; Tabery, J., & Griffiths, P. E., (2010), "Historical and Philosophical Perspectives on Behavioral Genetics and Developmental Science," in K. Hood et al., (Eds.), *Handbook of Developmental Science, Behavior, and Genetics* (pp. 41-60), Malden, MA: Wiley-Blackwell.

[6] Faraone, S. V., Tsuang, M. T., & Tsuang, D. W., (1999), *Genetics of Mental Disorders,* New York: Guilford, p. 32.

[7] Faraone et al., 1999, p. 32.

[8] Kendler & Prescott, 2006, pp. 44-45.

[9] Joseph, J., (2015), *The Trouble with Twin Studies: A Reassessment of Twin Research in the Social and Behavioral Sciences*, New York: Routledge.

[10] Glatt, S. J., Faraone, S. V., & Tsuang, M. T., (2008), "Psychiatric Genetics: A Primer," in J. Smoller, B. Sheidley, & M. Tsuang (Eds.), *Psychiatric Genetics: Applications in Clinical Practice* (pp. 3-26), Washington, DC: American Psychiatric Publishing.

[11] Moore, D. S., (2013b), "Current Thinking about Nature and Nurture," in K. Kampourakis (Ed.), *The Philosophy of Biology: A Companion for Educators* (pp. 629-652), Dordrecht: Springer, p. 636.

[12] Chaufan, C., (2008), Unpacking the Heritability of Diabetes: The Problem of Attempting to Quantify the Relative Contributions of Nature and Nurture, *DataCrítica: International Journal of Critical Statistics, 2,* 23-38.

[13] Charney, E., (2012), Behavior Genetics and Postgenomics, *Behavioral and Brain Sciences, 35,* 331-358; Moore, D. S., (2013a), "Behavioral Genetics, Genetics, and Epigenetics," in P. D. Zelazo (Ed.), *Oxford Handbook of Developmental Psychology* (pp. 91 - 128), New York: Oxford University Press; Meaney, M. J., (2010), Epigenetics and the Biological Definition of Gene x Environment Interactions, *Child Development, 81,* 41-79, p. 42.

[14] Glatt et al., 2008, p. 9.

[15] Segal, N. L., (2012), *Born Together—Reared Apart: The Landmark Minnesota Twin Study,* Cambridge, MA: Harvard University Press, p. 63.

[16] For example, see Chaufan, 2008; Goldberger, A. S., (1979), Heritability, *Economica, 46,* 327-347; Joseph, J., (2004), *The Gene Illusion: Genetic Research in Psychiatry and Psychology under the Microscope*, New York: Algora; Layzer, D., (1974), Heritability Analysis of IQ scores: Science or numerology?, *Science, 183,* 1259-1266; Lewontin, R. C., (1974), The Analysis of Variance and the Analysis of Causes, *American Journal of Human Genetics, 26,* 400–411; McGuire, T. R., & Hirsch, J., (1977), "General Intelligence (g) and Heritability (H^2, h^2)," in I. Uzgiris & F. Weitzmann (Eds.), *The Structuring of Experience* (pp. 25-72), New York: Plenum Press; Taylor, H. F., (1980), *The IQ Game: A Methodological Inquiry into the Heredity-Environment Controversy*, New Brunswick, NJ: Rutgers University Press; Wahlsten, D., (1990), Insensitivity of the Analysis of Variance to Heredity-Environment Interaction, *Behavioral and Brain Sciences, 13,* 109-120; Zuk et al., (2012), The Mystery of Missing Heritability: Genetic Interactions Create Phantom Heritability, *PNAS, 109,* 1193-1198.

[17] Lewontin, 1974, p. 402.

[18] Meaney, 2010, p. 42.

[19] Rutter, M., (2006), Genes and Behavior: Nature-Nurture Interplay Explained, Malden, MA: Blackwell, p. 24, italics in original.

[20] Hirsch, J., (1997), Some History of Heredity-vs-Environment, Genetic Inferiority at Harvard (?), and The (Incredible) Bell Curve, *Genetica, 99,* 207-224; Stoltenberg, S. F., (1997), Coming to Terms with Heritability, *Genetica, 99,* 89-96.

[21] Hirsch, 1997, p. 220.

[22] Keller, E. F., (2010), *The Mirage of a Space Between Nature and Nurture*, Durham, NC: Duke University Press, p. 59.

[23] Lewontin, 1974, p. 400.

[24] Moore, 2013b, p. 636.

[25] Kirk, S. A., Gomory, T., & Cohen, D., (2013), *Mad Science: Psychiatric Coercion, Diagnosis, and Drugs,* New Brunswick, NJ: Transaction.

[26] I placed the word "have" in quotation marks because, lacking a proven biological basis, someone cannot "have" a psychiatric disorder in the same way as someone has a real biologically based medical condition.

[27] Folstein, S., & Rutter, M., (1977), Genetic Influences on Infantile Autism, *Nature, 265,* 726-728, p. 728.

[28] Bailey et al., (1995), Autism as a Strongly Genetic Disorder: Evidence from a British Twin Study, *Psychological Medicine, 25,* 63-77, p. 63.

[29] For a critical review of autism genetic research, see Joseph, J., (2006), *The Missing Gene: Psychiatry, Heredity, and the Fruitless Search for Genes*, New York: Algora, Chapter 7.

[30] Bailey et al., 1995.

[31] Hadjivassiliou et al., (2010), Gluten Sensitivity: From Gut to Brain, *Lancet Neurology*, *9*, 318-330, p. 320.

[32] Sapone et al., (2012), Spectrum of Gluten-Related Disorders: Consensus on New Nomenclature and Classification, *BMC Medicine*, *10,* (1), 13, p. 4, doi:10.1186/1741-7015-10-13.

[33] Segal, 2012, p. 227.

[34] Wahlsten, 1990, p. 119.

[35] Moore, D. S., & Shenk, D., (2016), The Heritability Fallacy, *WIREs Cognitive Science*, doi: 10.1002/wcs.1400