The debate over twin studies: an overview

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> It is a thing of no great difficulty to raise objections against another man's oration,—nay, it is a very easy matter; but to produce a better in its place is a work extremely troublesome.
>
> -Plutarch

Introduction

Although many twin studies have been conducted (which is quite an understatement; there are almost 9,000 hits for "twin study" on PubMed!), there have long been critics who argue that they are scientifically worthless. Obviously, the behavior geneticists who conduct these studies with the aim of separating the influences of genes from that of environment are none too happy about people calling one of their favorite research designs fatally flawed. So how do they respond, and are their responses more compelling than the original criticisms? I will dig into these in this article (which will be long, so be warned).

First, I should define what a twin study is in this context. When the phrase "twin study" is used, it is almost always used to refer to a study using the so-called "classical twin design" (abbreviated CTD). So for the rest of this paper, I will use the phrase "twin study" to refer only to studies using the CTD. This type of study involves comparing monozygotic (MZ) twins and dizygotic (DZ) twins with the purpose of estimating how much of the variation in a given trait (i.e. %) is due to genes, a.k.a. "heritability".

The way this is done in the CTD is by calculating concordance (the degree of similarity between each twin in a pair; referred to as correlation if a disease is being studied rather than a normal trait) on the trait in question for the monozygotic twins and dizygotic twins separately, and comparing the two concordances. (Important note: monozygotic twins-aka "identical twins"-are assumed to share 100% of their genes, while dizygotic twins-aka "fraternal twins"-only share 50% of theirs. More on the first of these assumptions later.) These concordances are then converted into an estimate of heritability (in this case, narrow-sense heritability, h^2, which only includes additive genetic effects) using this formula (known as Falconer's formula):

$$h^2 = 2(r_{MZ} - r_{DZ})$$

Where r_{MZ} and r_{DZ} are, respectively, the phenotypic correlation (for traits) or concordance (for diseases) between MZ twins, and that between DZ twins, on the trait/disease of interest.(Mayhew & Meyre, 2017)

Thus, when this formula is used (which it typically is in twin studies), if the concordance is higher among monozygotic twins than among dizygotic twins, this is taken to be due to genetic differences between the two sets of twins, and will yield a nonzero heritability estimate. In contrast, if the concordance rates were the same between MZ and DZ twins on the trait, it would indicate an estimated narrow-sense heritability

of zero for the trait, under the CTD model(Guo, 2001). This is obvious from looking at the formula above, where r_{MZ} and r_{DZ} being equal makes the value of h^2 equal to 0. Note that the extent to which $h^2 > 0$ is often conflated with the extent to which a trait is under "genetic influence" in the twin study literature.

Final note: there are two other components to this model: shared and nonshared environment. The three components of the model, to recap, are: additive genetic variance (A), shared/common environment (C), and nonshared environment (E). Thus it is often called the "ACE model".(Maes, 2005) The basic idea behind this model is that total phenotypic variance is exactly equal to the sum of each of these three components: A + C + E.

But there have long been many criticisms of the specific way that twin studies are conducted, and critics claim that such studies can't really separate genetic and environmental contributions to *any* trait. But Beatty et al. (2002) inform us that such critics are mistaken: "..., satisfactory responses to critics (see for example, Bouchard, 1993, 1994; Goldsmith, 1983; Lykken, 1995; Martin, Boomsma, & Machin, 1997; Scarr & Carter-Saltzman, 1979; Segal, 1997, 1999) have led contemporary behavior geneticists to describe the twins design as "the perfect natural experiment" (Martin et al., 1997. p. 387)."(Beatty et al., 2002), (p. 2)

Oh, so that's good to know, apparently criticisms of twin studies have already been conclusively refuted. So I'll go back to these sources later in this article to see if they actually conclusively show that critics of the twin method are wrong about its purported uselessness.

Criticism 1: The equal environments assumption

For decades, critics of twin studies have argued that they don't really separate the influence of genes and environment on a specific trait because the validity of their heritability estimates depends on the so-called "equal environments assumption" (abbreviated EEA). This is the assumption that the within-twin similarity of environmental exposures that are relevant for the causation of the trait being studied are equal for MZ and DZ twins. In other words, it assumes that MZ twins experience equally similar environments as do DZ twins, at least with regard to the environmental factors that can cause the trait being studied. If this assumption is wrong, it will lead to heritability estimates that are also wrong: if MZ twins experience more similar environments than DZ twins, this can produce higher concordance rates without any genetic influence on the trait whatsoever.(Guo, 2001)

As you may have guessed, there is a lot of evidence that MZ twins experience more similar environments than do DZ twins (for a summary, see (Simons et al., 2012); see also Joseph 2002, Horwitz et al. 2003, Rende et al. 2005). In fact, even an article by BGists aiming to *defend* the validity of twin studies acknowledged that "There is overwhelming evidence that MZ twins are treated more similarly than their DZ counterparts" (Evans & Martin 2000). But to be fair, that's not necessarily the same as the kind of fundamental invalidating flaw critics often portray violations of the EEA to be. Typically, behavior geneticists respond to this evidence by making one or more of the following arguments (hereafter referred to as "argument #", where "#" is the number listed corresponding to each argument immediately below):

1. **Greater environmental similarity between twins in a pair is not significantly associated with greater phenotypic similarity.** This argument is based on the assumption that the EEA does not have to be entirely valid for all environmental factors, but instead just for those that are "trait-relevant", i.e. that have a causal effect on whatever trait is being studied. The practice of defining the EEA as applying only to "trait-relevant" environmental factors is common in behavior genetic research (e.g. (Mitchell et al., 2007), (Bouchard & McGue, 2002),(Kendler et al., 1993)). This

argument further claims that the hypothesis that the EEA is invalid for trait-relevant environmental factors must be tested rather than assumed to always be true, after which studies are cited and/or performed that supposedly test this definition of the EEA. These "EEA-test" studies come in many forms: a) "perceived zygosity" or "misclassified twin" studies, a specific type of twin study designed to test the EEA. Such studies take advantage of the fact that some MZ twins are perceived as being DZ and vice versa. The idea is that if greater genetic similarity between MZ twins is what makes them more similar, then actual zygosity should have a greater impact on twin similarity, i.e. MZ twins should be more phenotypically similar to each other than DZ twins even if the MZ twins are incorrectly perceived as DZ. By contrast, if greater environmental similarity (such as is, presumably, experienced by twins *perceived* to be MZ, whether they are actually MZ or not) explains the greater phenotypic similarity of MZ twins relative to DZ twins, then MZ twins perceived as DZ should be no more phenotypically similar to each other than are DZ twins correctly perceived as DZ. Indeed, actual zygosity has been found to be more strongly associated with twin concordance in multiple such studies.(Conley et al., 2013) b) studies aimed at correlating physical similarity and phenotypic similarity between twins in a pair, and if no such (statistically significant) correlation is found, then it is concluded that the EEA is "valid" in that it doesn't significantly affect heritability estimates for whatever trait is being studied.(Kendler et al., 1993) c) studies including perceived zygosity as well as other measures of environmental similarity, controlling for such measures, and then comparing the "corrected" MZ and DZ correlations to see if the difference between the two correlations remains significant. Two such studies have found that it does.(LaBuda et al., 1997)(CRONK et al., 2002)

2. **Greater environmental similarity between members of an MZ twin pair than a DZ twin pair, rather than causing the greater phenotypic similarity between MZ than DZ twins, is actually due to the MZ twins' greater genetic similarity.** This argument posits that because MZ twins are more genetically similar than DZ twins, they are treated more similarly and have more similar behavior, which in turn leads to the environments of MZ twins being more similar than those of DZ twins. Many twin researchers have made this argument since at least the 1950s. It has been widely criticized as circular because it assumes that the greater genetic similarity between MZ than DZ twins causes their greater phenotypic similarity, which is also what it purports to demonstrate.(Joseph, 2012)
3. **Non-CTD studies that do not rely on the EEA generate similar heritability estimates to CTD studies for the same trait.** Ostensibly, the similar results generated by these non-CTD methods "validate" the EEA on which CTD studies depend. The word "validate" or derivatives thereof is often used in this literature (for two recent examples, see (Kendler et al., 2014) and (BARNES et al., 2014)). The methodologies of these studies include twins reared apart, adoption studies, and full- vs. half-sibling studies.(BARNES et al., 2014)

Response by critics of behavior genetics

Criticism 1: Response to Argument 1

Joseph commonly rebuts this argument by pointing out that it reveals a different standard of logical inference that twin researchers use in assessing twin studies versus family studies. Specifically, according to twin researchers, the greater similarity of environments among MZ compared to DZ twins does *not* necessarily invalidate the classical twin method as a means of separating genetic and environmental influences, but the greater similarity of environments shared by members of the same family relative to members of the general population *does* necessarily invalidate the ability of family studies to separate such influences. For this reason, Joseph argues that twin researchers are guilty of special pleading by arguing that standards that apply to family studies should not apply to classical twin studies. As an example, he wrote in 2002 that:

> "genetic researchers acknowledge that family studies do not prove the existence of genetic factors—since the clustering of a condition among family members could be caused by purely

> environmental factors. However, no one to my knowledge has argued for a "trait-relevant EEA" for family studies; that is, the claim that family studies prove the existence of genetic influences unless specific environmental factors shared by family members are demonstrated to have a causal relationship to the condition in question. Quite the contrary; family studies are acknowledged to be confounded by the simple fact that family members share a common environment. In the same way, the twin method is confounded by the greater environmental similarity of MZ twins, regardless of whether the specific trait-relevant environmental factors are known."(Joseph, 2002), (p. 76)

Joseph also notes that by using this argument and defining the EEA as needing to be "trait-relevant", twin researchers attempt to falsely claim that the burden lies on critics of the classical twin method to prove that MZ environments are more similar than those of DZ twins specifically with regard to those environmental factors that affect a given trait. He also points out some other logical problems with this argument in some of his papers.(Joseph, 2013)(Joseph, 2002)

In addition, Joseph and other researchers have been highly critical of the EEA-test studies cited as part of this argument.(Joseph, 2013; Joseph, 2012)(Richardson & Norgate, 2005)(Beckwith & Morris, 2008)

Criticism 1: Response to Argument 2

Joseph has noted the following about this argument: "...this is a circular argument, because twin researchers' conclusion that MZ-DZ differences are explained by genetics is based on assuming the very same thing."(Joseph, 2013), (p. 5) Furthermore, he points out some other logical issues with it, such as that it portrays "...children (twins) as behaving according to a genetic behavioral blueprint, yet somehow parents and other adults have themselves tossed aside the blueprint and are able to flexibly change their behavior and treatment of others on the basis of the twins' behavior and personalities," which he wittily sums up with the phrase "Genetically-programmed human children, meet your ever-so-flexible human parents." He has also pointed out another problem with this argument, namely that "...even if twins do indeed create more similar environments for themselves because of their greater genetic similarity, MZ pairs could still show much higher concordance for psychiatric disorders than DZ pairs for purely environmental reasons."(Joseph, 2012)

Examples of the above arguments

Argument 1: No significant relationship between environmental/physical similarity and phenotypic similarity in twins

"Measures of the degree of similarity in parental treatment turn out to not be correlated with similarity in IQ or other personality measures."(Cesarini et al., 2009), (p. 828)

"...mothers' and fathers' approach to raising twins had no significant influence on twin resemblance for the four examined psychiatric disorders [namely, major depression, generalized anxiety disorder, phobia, and alcoholism]. These results suggest that the differential treatment of MZ and DZ twins by their parents is unlikely to represent a significant bias in twin studies of these major psychiatric disorders."(Kendler et al., 1994)

"If the greater genetic relatedness of MZ twins was irrelevant to their observed greater ideological similarity, then MZ and DZ twins should display substantially reduced correlations when environmental similarity or contact is reduced. In addition, the correlations for MZ and DZ pairs should be approximately equal across levels of environmental similarity and contact...this is not the case."(Smith et al., 2011)

"...in the relatively rare cases where parents miscategorize their twins as MZ instead of DZ (or the converse), differences in correlations of cognitive ability and personality persist."(CESARINI et al., 2010), page 1742

"Controlling for environmental similarity reduced heritability significantly for only one out of the 32 outcomes examined: neuroticism."(Felson, 2014)

"The EEA was investigated with a linear regression model, examining if the twins contact frequency predicted within-pair differences in PA [physical activity], and further by a simulation study. We found no support for violation of the EEA."(Eriksson et al., 2006)

"...the validity of the equal environments assumption has been evaluated using mislabeled twins (twins labeled DZ when they are in fact MZ) or and MZ twins who are in fact treated differently. Both methods rely on the idea that MZ twins who are treated more individually should show more differences than those who are treated more similarly. Studies using both methods provide evidence for the validity of the assumption."(Kohler et al., 2011)

"...studies that examined twin physical similarity or parental behavior towards twins as well as the effects of perceived zygosity (where DZ twins were identified incorrectly as MZ)...have concluded largely that previous assertions of violations of EEA may have been overstated."(Agrawal & Lynskey, 2008)

"...similarity of friends or parental treatment is not associated with twin similarity in personality, interests, or abilities."(Bouchard & McGue, 2002)

Argument 2: Greater genetic similarity of MZ than DZ twins causes their greater environmental similarity

For a list of examples of this argument, see (Joseph, 2012).

Argument 3:

"...findings garnered from studies using the classical twin design, the adoption design, and the twins reared apart design largely converge"(BARNES et al., 2014)

"...the results of adoption studies and studies of twins reared apart are generally consistent with the results of classical twin studies."(Felson, 2014)

Criticism 2: But they aren't identical!

Twin studies assume that MZ twins are 100% genetically identical, which is not true. For one, they have significant differences in their epigenetic profiles.(*DNA not necessarily your destiny? The role of epigenetics in pharmacy*, n.d.)(Fraga et al., 2005)(Haque et al., 2009)(Petronis et al., 2003) Yet another classical twin study assumption bites the dust. Or does it? Twin researchers can reply to this claim in a number of ways, but their preferred approach is to say that there are no better ways to control for genetic factors than by comparing MZ and DZ twins, because they are so close to being 100% and 50% identical, respectively.

For instance, Verhulst & Hatemi (2013) argue that "... there is no better control of gene expression than identical twins, and there are no better controls for both family environments and genotype than identical twins. Indeed, the best models of genetic expression for complex human traits involve identical twins."(Verhulst & Hatemi, 2013) Similarly, Segal et al. (2011), in their response to a *Slate* article slamming classical twin studies, describe genetic and other biological differences between each twin in a pair of MZ twins. They then argue that "Despite these biological differences, MZ twins (whether reared apart or together) are more alike than any other pair of individuals. Moreover, **to the extent that MZ twins are not genetically identical, the true importance of genes is actually underestimated by the MZ-DZ comparison** (my emphasis)."(Segal, 2011)

This argument (that identical twins not being identical makes heritability estimates from twin studies too low, not too high) is also a common one among twin study defenders, ranging from Segal et al. to Tabarrok's and Collins' responses to the same 2011 *Slate* article.(*Twin Studies and Beyond - Marginal REVOLUTION*, n.d.)(*Underestimating heritability*, n.d.) It has received support from a recent study in *Behavior Genetics*, which found that if identical twins aren't quite identical, this leads to heritability estimates being higher than, and only slightly different from, what they should be, based on mathematical simulations.(Liu et al., 2017) Of course, whether these simulations accurately represent actual biological or epigenetic processes is another matter entirely.

Criticism 3: What about epigenetics and gene-environment interactions?

A recent paper by Richard Lerner and Willis Overton, both longtime and outspoken critics of behavior genetics, argues that "epigenetics invalidates all models involving genetic reduction". They argue that this is the case because, contrary to genetic reductionist models such as that of the classical twin study, "Genes do not determine behavior" (Lerner & Overton 2017). Note that such models are supposed to include behavior genetic models, which they are accusing of being "genetic reductionist" because they (supposedly) grossly oversimplify the ways that genes affect human traits. Another recent paper reviewed the literature on the genetic contribution to aging traits and concluded: "...it is likely that epigenetics contributes to heritability because MZ twins (particularly dichorionic who have split earlier in development) have less DNA methylation difference than DZ twins."(Steves et al., 2012)

And then there is the issue of gene-environment interactions (often abbreviated GxE). As a 2004 *Monitor on Psychology* article noted, "Some researchers think that interactions between genes and environment, rather than genes and environment separately, may influence many traits. A recent study from *Science* (Vol. 297, No. 5582) by Avshalom Caspi, PhD, of King's College London, for example, suggests that a gene might moderate propensity for violence, particularly in people who are severely maltreated as children. Many twin study designs don't take this type of complication into account" (Winerman 2004).

But wait, you haven't heard behavior geneticists defend their beloved twin studies against *these* accusations yet. So what are their rebuttals?

You could take the approach of Barnes et al. (2014), who simply write that "...much of the GxE literature is under heavy criticism and epigenetics is in its infancy". They also use the argument that many (perhaps 90%!) of the findings of reported GxE interactions are false positives to cast doubt on whether such findings exist to a significant extent.

Alternatively, rather than questioning whether GxE effects occur, you could acknowledge that they do, but then say that behavior geneticists are already well aware of them and incorporate them into their models regularly. You could also argue that, sure, the CTD is seriously flawed because it ignores GxE interactions, but argue that behavior geneticists have other types of models they can use that do take into account such interactions. Consider the following quote from a response by two "genopolitics" researchers to a critique of the CTD:

> "GE interplay is clearly ingrained within the theory and statistical model of modern twin analyses... the fact that the CTD does not estimate GE interplay is not a failure of the model. If GE interplay is suspected, then extensions of the CTD are available and should be utilized" (Verhulst & Hatemi 2013).

After citing this paper, Hatemi et al. (2014) argue that "Behavior genetic approaches have also inspired

philosophical objections due to the firm belief that behavioral differences are entirely socialized. Several publications in the political science literature have begun to erode this view and explicate the theoretical and empirical justification for inclusion of genetic influences and biological mechanisms in general, including gene-environment interplay"(Hatemi et al., 2014).

So what about epigenetics? Do BGists have any decent responses to the "epigenetics refutes the CTD" argument? Well, they could say this: "Most researchers consider political traits to be influenced by thousands of genetic markers both indirectly and through interactions with numerous environmental stimuli and other genes in complex genomic, epigenetic, and neural pathways. By contrast, many criticisms are developed as if responding to the view that political traits are simple Mendelian traits, governed by a single gene or a small set of genes."

Another time-honored (but questionable) method is to do a simulation study estimating the effect of (be warned, this is a mouthful) differences in genetic similarity between MZ and/or DZ co-twins from assumed values on heritability estimates. In classical twin studies, genetic similarity between MZ and DZ co-twins is assumed to be 100% and 50%, respectively, and a recent simulation modeling study estimated that when the actual similarity is different from the assumed values, it leads to wrong estimates, but not that wrong: "Although estimates of genetic and nonshared environmental influences from the standard biometric model were found to deviate from "true" values, the bias was usually smaller than 10% points indicating that the interpretations of findings from previous twin studies are mostly correct."(Liu et al., 2017)

In short, behavior geneticists argue that the evidence of GxE interactions and epigenetic effects is too preliminary and/or weak and/or inconsistent to cast much doubt on the results of twin studies. Unless they don't, in which case they say, "Uh, we already know about this shit, so you don't need to tell us, and we already take these phenomena into account", and "Don't attack a straw man, we don't think political beliefs are only determined by a single gene, of course other factors are important!"

Criticism 4: These studies assume random mating

Barnes et al. (2014) note that classical twin studies assume random (or assortative) mating. They state further that "...violation of the random mating assumption leads to inflated estimates of the shared environment effect and deflated estimates of heritability." Similarly, Verhulst & Hatemi (2013) write that "...CTD models assume random mating and due to the very large correlation between spouses (r [?] 0.6) for attitudes, the CTD actually underestimates the magnitude of genetic influences".

Some researchers have used the "nuclear twin family model" to account for this assumption, and the assumption that dominant genetic effects and shared environment aren't present together, in the CTD. This has yielded somewhat lower estimates of heritability.(Burt & Klump, 2012)

Criticism 5: These studies assume MZ twins, DZ twins, and singletons all have the same prevalence for the trait under study

The CTD assumes that the trait being studied is equally prevalent in MZ twins, DZ twins, and singletons (this last word refers to people who aren't any kind of twin).(Thomsen, 2014) BGists respond by simply saying that this assumption is true, e.g.: "Twins are indeed representative of the general population for a wide range of traits and diseases..."(Evans & Martin, 2000) And in many other studies by BGists, the same claim has been made, namely that twin samples are representative of the general population:

- "...twins do not systematically differ from the general population of non-twins on many measures of behavior and development"(Barnes & Boutwell, 2013)

- "The TEDS [Twins' Early Development Study] sample has been shown to be reasonably representative of the general population in terms of parental education, ethnicity, and employment status". (Haworth et al., 2008)
- "... results from twin samples generalize to singletons."(Munn-Chernoff et al., 2013)
- "... twins are representative of the general population of MS [Multiple Sclerosis] patients and are probably influenced by the same susceptibility genes and environment as nontwin cases."(Willer et al., 2003)
- "It would appear that, with respect to personality, twins are not systematically different from other people."(Johnson et al., 2002)
- "... volunteer twins were not found to differ from age-matched singleton women in distribution or prevalence of: bone mineral density, osteoarthritis, blood pressure, hypertensive drug use, height, history of hysterectomy and ovariectomy, menopausal status and current alcohol and overall tobacco consumption."(Andrew et al., 2001)

But other researchers are more cautious, for example: "Twins are representative of the general population for some but not all measures of ocular biometry. Consequently, care should be taken when extrapolating twin data for these traits in heritability and other genetic studies."(Sanfilippo et al., 2011)

Criticism 6: Like, what even *is* heritability, anyway?

Critics of BG have long noted that the concept of "heritability" (at least in the context of human BG) is highly misleading because it seems to mean "genetic determination", but it really does not mean that at all. Instead, it just means the % of variation in a population in a given trait that is due to genes. "Genetic determination" is an *individual-level* characteristic, not a *population-level* one like heritability, so heritability says nothing at all about how much of any trait YOU have is due to YOUR genes.(Sauce & Matzel, 2018) As Ned Block noted more than two decades ago, it is therefore meaningless to ask, for example, "What's the heritability of my IQ?"(Block, 1995) Also, heritability analyses, which are based on the analysis of variance (ANOVA) statistical technique, are not very good at detecting gene-environment interactions if they are present.(Wahlsten, 1990) Lastly, since the concept of heritability was invented in the 1940s for predicting the success of selective breeding in farm animals,(Wahlsten, 2003) it is very unlikely that all, or even most, of the assumptions on which heritability estimates for the purpose of selective breeding depend will be met in normal human populations. As Stoltenberg (1997, p. 91) noted, "... human populations are much more unlikely [than farm animals] to satisfy the host of assumptions that undergird partitioning behavioral variance into the P = G + E model. Such assumptions include no dominance deviation, no epistasis, no assortative mating, no genotype × environment (G x E) interaction, and no GE covariance".(Stoltenberg, 1997)

But there are even more problems: heritability refers only to the % of variation of genetic origin *in a given population* and *in a given environment*, so changes in the environment can *always* affect how heritable something is.(Rose, 2006) Not only that, but heritability also says nothing about the relative influence of genes or environment on a trait, and since genes definitely have to do with almost all human traits, knowing the percent "heritability" of any such trait is almost always useless.(Moore & Shenk, 2016) Clearly, the common view that high heritability is indicative of current low environmental influence for any trait, or a lack of susceptibility of that trait to environmental changes, is entirely wrong.(Tucker-Drob et al., 2013)(Hopper, 2017)

Validated?

One of the key arguments used by BGists to defend the CTD doesn't really fit neatly into any of the categories above. Instead, it is used as though it by itself could answer all the criticisms above, and any other criticisms that could be made of the methodology. This is the argument that the results of CTD studies can be "**validated**"by producing similar estimates of heritability using different methodologies.

Thus, if this kind of replication of results is found when using non-CTD methods, it is cited by BGists as slam-dunk evidence that the CTD is valid for estimating heritability. One of the main reasons for this argument's strength (apparent or real) is that CTD studies are based on different assumptions than other genetic study designs (specifically, reared-apart twins, family, and adoption studies). One of the main reasons for this argument's strength (apparent or real) is that CTD studies are based on different assumptions than other genetic study designs (specifically, reared-apart twins, family, and adoption studies).

So here are some examples of BGists making this argument (all emphases are mine):

- "Heritability estimates for CB (criminal behavior) from full- and half-siblings closely approximated those found from twins in the same population, **validating the twin method**."(Kendler et al., 2015)
- "For the three externalizing syndromes examined, **concerns that heritability estimates from twin studies are upwardly biased... were not supported**... The heritability estimates for CB, AUD (alcohol use disorder) and DA (drug abuse) that we obtained from our sibling trios were very similar to those obtained from MZ and DZ twins from the same population using the same diagnostic methods."(Kendler et al., 2016)
- "Moving beyond the twin-only design leads to the conclusion that for most political and social attitudes, genetic influences account for an even greater proportion of individual differences than reported by studies using more limited data and more elementary estimation techniques."(Hatemi et al., 2010)
- " The aim of the current study is to characterize more reliably than previously possible ECG [electrocardiogram] trait h^2 using GREML estimation, and to compare these outcomes to those of the classical twin model... **We found no evidence that the classical twin model leads to inflated h^2 estimates**."(Nolte et al., 2017)
- Not too long ago, I sent an email to prominent twin researcher Dorret Boomsma, asking her about how she would respond to classical twin studies' critics. In her reply (which I received July 17, 2018), Boomsma said that she would respond to them by "... pointing to the empirical evidence from extended twin-family designs and molecular studies, which all confirm findings from the classical twin design, as well as findings from twins reared apart studies."
- In a 2016 article in *the Guardian* written as a response to Oliver James, Marcus Munafo argues, "Twin, family and adoption studies... have been criticized... However, two lines of evidence give us confidence in the overall conclusion. First, many of the assumptions have been directly tested and shown to hold, at least sufficiently to mean that the method is valid. Second, and more importantly, **each method relies on a *different* set of assumptions, and yet they all give very consistent results. It's highly unlikely that invalid assumptions across all of these methods would distort the results in exactly the same way."** (My emphasis.)(*Genetic denialism is unhelpful - genes play a role in who we are*, n.d.)
- "Although the twin and adoption designs have separately been criticized, these two designs generally converge on the same conclusion, despite making very different assumptions, which adds strength to these conclusions."(Plomin et al., 2016)

So this argument is supposed to show that twin studies are a valid research design for estimating heritabilities because other methods (even if they have also been criticized for having flaws) yield similar results.

As you can imagine, critics of BG have responded to this response already. For instance, Joseph et al. (2015) argued, "there are major problems with previous criminal and ASB [antisocial behavior] adoption studies, many of which were discussed by Burt and Simons, and reared-apart twin studies are greatly flawed on

several critical dimensions."(Joseph, 2015)

The tactic of persecution

BGists love to construct a persecution complex that they supposedly are hindered by in their research, bravely challenging the orthodox view that all social behaviors are entirely determined by the environment. For instance, Hatemi et al. (2014) write that "The notion of a genetically informed model of attitudes and ideology, understood as a psychological disposition that guides behavior, independent of, and interacting with, social forces presented a fundamental challenge to the dominant rational choice and behaviorist social science paradigms." Thus they portray themselves as bravely going wherever the evidence leads, though they give their ideological genetic-determinist agenda away later in the same paper, when they cite criticism that BG studies of political behavior as "limiting the integration of biological factors into mainstream conceptualizations of political ideology."(Hatemi et al., 2014) Certainly, such a statement seems to indicate that whoever would write or say it would view biological factors as inherently important in political ideology, as an *a priori* belief, no matter what the evidence really says, no?

I am not the first to note this weird *ad hominem* tactic of BGists responding to scientific criticisms by accusing their critics of being political ideologues set to deny the potential for biological influences on human behavior. Panofsky even came up with a name for it: the "hitting-them-over-the-head style" (*Misbehaving Science*, page 142). Like global warming deniers, they will paint themselves as being persecuted for their "heretical" or "politically incorrect" beliefs that challenge the "dogma" of the scientific establishment. But while global warming deniers claim to be questioning the dogma of mainstream global warming science, BGists portray themselves as bravely fighting against the dogmatic beliefs of environmental determinism and the assumption of a total lack of biological or genetic influence on human behavior.

I will close this section by citing more examples of the "hitting-them-over-the head" approach used by BGists dismissing critics as ideologically motivated to deny the role of genes in human behavior completely. Enjoy! (Note: all emphases below are mine.)

1. Ä disconnect has developed between criticisms that focus on improving existing models and those that **seek to abolish or eliminate the entire research agenda, oftentimes for ideological reasons, such as wholesale objections to biological work because of fear of past abuse, or threats to current dominant models.** As a result of such largely unspoken existential divides, it has proven difficult for life and social scientists to enter an honest discussion about the limitations inherent in genetic work and still employ the methods in a progressive and useful manner."(Hatemi & McDermott, 2012)
2. SSince its inception, [behavior genetics] has been a lightning rod of criticism, especially by scholars who are inalterably opposed to linking biology with behavior."(BARNES et al., 2014)
3. "Beginning in the 1970s, politically motivated critics of behavioral genetics launched an all-out crusade against such methods, the findings emanating from them, and even on the researchers themselves (Segerstrale, 2000). These critics called for an end to the idea that biology had anything to do with behavior, noting that sociobiology was a "dangerous idea." "(BARNES et al., 2014)

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