

and chemistry. Ellestad leaps to the conclusion that the explanation must reside in our genes because he can't fathom an environmental one and then suggests a just-so story: that "there seems little doubt that this was a survival mechanism when the family groups were the only defense against other primitive men." There are, however, myriad variables that cause one person to murder another or one group to commit genocide, and the ultimate explanation will not be so convenient or easy.

Regardless, the logic of Ellestad's argument is flawed. Ellestad, and evolutionary psychologists for that matter, need to understand that just because

some human behaviors seem to be universal, doesn't mean that they are, nor does it mean that such behaviors have a genetic origin. To begin with, genocide is not universal; it is not occurring in the U.S., Europe, Canada, Australia, South America, etc. Are we missing the necessary genes? Where it does occur, it is largely religious (and sometimes ethnic). Would Ellestad suggest that there are genes for religious differences? Even behavior genetics researchers understand that behaviors that are exhibited by many people can result from shared environments as well as shared genes. In fact, the vast majority of human beings have learned "not to murder

those they don't agree with."

So, after all this wrangling about nature and nurture, we're still left trying to understand how genes and learning interact to produce the behavior of individuals, either alone or in groups. As I asserted in my article, principles of learning science, and in the particular instance of group behavior, decades of good research by social psychologists, can provide a more parsimonious explanation than vague ad lib evolutionary speculations and the correlational research methods of behavior geneticists.

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Comments about Frank Miele's Article "The Revival of Human Nature ≠ the Denial of Human Nurture"

The Gene Illusion: A Critique of Frank Miele

Of the many problems with Miele's article, I would like to focus my comments on his reliance on behavior genetic research, since a major portion of my recent book, *The Gene Illusion: Genetic Research in Psychiatry and Psychology Under the Microscope*, is dedicated to a critical analysis of this body of research.¹

Like Steven Pinker in *The Blank Slate* and Matt Ridley in *The Agile Gene*, Miele's argument rests mainly on the results of reared-together twin studies, and studies of reared-apart twins.² However, twin research is plagued by bias, methodological problems, and a reliance on untenable theoretical assumptions. The main tool of behavior genetics and psychiatric genetics is the "classical twin method," more commonly known as "the twin method." The twin method compares concordance rates or correlations of reared-together identical (also known as monozygotic or MZ) twins, who share 100% genetic similarity, versus the same measures of reared-together same-sex fraternal (also known as dizygotic or DZ) twins, who average a 50% genetic similarity. Based on the assumption that both types of twins experience equal childhood and adult environments, known as the "equal environment assumption" or "EEA," twin researchers attribute a significantly higher resemblance among identical versus same-sex fraternal to

genetic factors.

However, as I documented in *The Gene Illusion*, there is overwhelming evidence that identical twins experience much more similar environments than fraternal, and, perhaps more important, identicals experience a stronger psychological bond and more often experience identity confusion.³ Twin researchers often concede these points, yet continue to uphold the validity of the twin method on the basis of two claims. The first is that, although identical and fraternal twin environments are different, critics must identify the "trait-relevant" environmental factors for which identical and fraternal twins experience dissimilar environments. However, because a basic tenet of science holds that the burden of proof falls on the claimant, not on critics,⁴ twin researchers themselves bear the burden of proof for showing that identical and fraternal twins are not differentially exposed to potentially relevant environmental factors. Moreover, although faced with a similar problem, twin researchers and behavior geneticists do not make the "trait-relevant" argument when discussing potential environmental confounds in family studies. In this case they are willing to concede that, because family members share a common environment ("trait-relevant" or not), one cannot draw valid conclusions in favor of genetics on the basis of the family resemblance of a trait.

The second claim in defense of the

twin method is that identical twins "create" more similar environments for themselves by virtue of their greater genetically-caused similarity of behavior.⁵ Therefore the twin method's validity, according to twin researchers, is based on determining why—not whether—identical twins experience more similar environments than fraternal. Twin researchers and popularizers of their work, however, fail to understand that the reason identical twins experience more similar environments than fraternal twins, be it environmental or genetic, is completely irrelevant in assessing the validity of the EEA. The only relevant question is whether—not why—identical twins experience more similar environments. Moreover, the "twins create their environment" argument is circular because the evidence that twin behavioral similarity is caused by genetics is implicitly derived from previous twin studies.

Therefore, like family studies, the twin method is unable to disentangle the possible role of genes and environment on psychological trait variation or psychiatric disorders. There is, in fact, little reason to accept that the twin method measures anything other than the more similar treatment, greater environmental similarity, and closer psychological association experienced by identical versus fraternal twins. This brings us to the frequently cited "twins reared-apart" (TRA) studies, which I analyze in

Chapter 4 of *The Gene Illusion*.

Problems in this area include (1) the dubious "separation" of twins, who in many cases grew up together and had quite a bit of contact over much of their lives; (2) the similarity bias of the samples; (3) researchers' failure to publish or share raw data and life history information for the twins under study (Thomas J. Bouchard, Jr.'s Minnesota study), and (4) the impact that the researchers' bias in favor of genetic explanations had on the interpretation of their results.⁶

The main problem with TRA studies such as Bouchard's, however, is that the investigators mistakenly compared reared-apart identical twins (monozygotic twins reared-apart, or MZAs) to reared-together identicals—thereby failing to control for the fact that both sets share several important environmental similarities. These include common age (birth cohort), common sex, similar appearance, and similar political, socioeconomic, and cultural environments. (In addition to MZAs, the Minnesota study used reared-apart fraternal twins as a comparison group, but they also share several of the environmental influences experienced by MZAs. The investigators also attempted to correct MZA correlations for age and sex effects, but their adjustments were inadequate and unclear.) Thus, behavior geneticists and TRA researchers such as Bouchard and his colleagues used the wrong control group, leading to their erroneous conclusions in favor of genetics. A scientifically acceptable study would compare the resemblance of a group consisting of MZAs reared apart from birth and unknown to each other, versus a control group consisting not of reared-together identical twins, but of biologically unrelated pairs of strangers sharing all of the following characteristics: they should be the same age, they should be the same sex, they should be the same ethnicity, the correlation of their rearing environment socioeconomic status should be similar to that of the MZA group, they should be similar in appearance and attractiveness, and the degree of similarity of their cultural backgrounds should be equal to that of the MZA pairs. Moreover, they should have no contact with each other until after they are eval-

uated and tested. After concluding such a study, we might find that the biologically-unrelated pairs correlate similarly to MZAs, which would suggest that MZA correlations are the result of environmental influences. Because no study of this type has ever been attempted, and because of the major flaws and biases in the studies that have been undertaken, we can draw no valid conclusions in support of genetic influences on psychological trait variation from reared-apart twin studies published to date.

Moving on to adoption studies, Miele claims that "adoptees become less like their adoptive parents or siblings and more like their biological parents." However, due to the restricted range of adoptive family environments, we would not expect large psychological trait correlations between adoptees and their adoptive parents,⁷ just as the correlation between boxers' weights, and their win/lose records is greatly reduced by the creation of weight divisions.⁸ Thus, boxing fans could erroneously conclude that the lack of a correlation between boxers' win/lose records and their weights means that increased weight would not improve a boxer's chance of winning, even if weight divisions were abolished. Turning to the resemblance of adoptees to their biological parents, a 1998 study coming out of the Colorado Adoption Project found that the mean personality scale correlation between birthparents and their adopted-away biological offspring (N = 245)—a relationship the investigators considered the most powerful adoption design for estimating genetic influence—was zero (.01 to be precise).⁹ The results of this longitudinal adoption study, performed by leading behavior geneticists, are rarely cited in the behavior genetic literature or in popular works.

Finally, Miele argues that autism is "highly heritable," citing a 2001 article claiming that regions on chromosomes 2 and 7 "were found to contain genes involved in the disorder." In fact, like schizophrenia, bipolar disorder, ADHD and the other major psychiatric disorders, autism molecular genetic research is characterized by the striking failure to identify genes, many years after researchers expected to make such discoveries.¹⁰ Several subsequently retract-

ed or non-replicated claims of "autism gene findings" have been made since at least 1997,¹¹ and similar unsubstantiated claims for other psychiatric disorders go back more than 20 years. A September, 2004, autism molecular genetics review article by genetic investigators Veenstra-VanderWeele and Cook made no mention of any findings on chromosomes 2 or 7. While they were optimistic that researchers will soon discover autism genes, Veenstra-VanderWeele and Cook acknowledged that "the detection of genetic variants responsible for the disease has thus far been elusive," and that "No gene variant has been identified yet as contributing to autism susceptibility in the majority of patients with ASD [autism spectrum disorders]."¹² Other genetically-oriented reviewers include Volkmar and Pauls, who wrote in 2003 of the "current lack of success in finding genes for autism..." and Pericak-Vance in 2003, who, although considering areas on chromosomes 2, 3, 7, and 15 to be "regions of interest," claimed no autism gene discoveries.¹³ At least ten complete autism genome scans have been published to date, yet they have yielded inconsistent and inconclusive results.¹⁴ (Another example of Miele's reliance on outdated information is his claim that humans have about 100,000 genes. This estimate was reduced to 30,000 to 35,000 at the time of the human genome sequencing in 2001, and by 2004, leading genetic researchers had reduced the estimate to 20,000 to 25,000. Thus, humans "have about the same number of genes as a small flowering plant or a tiny worm."¹⁵)

Moreover, despite the near-unanimous opinion that autism has an important genetic component, the evidence in support of this position is weak. It consists mainly of family studies, which cannot disentangle possible genetic and environmental influences, and four small methodologically flawed twin studies whose results, like all studies using the twin method, are explainable on environmental grounds. (In this case, since non-genetic biological factors might contribute to the condition, the more similar prenatal environments experienced by identical twins might be a major factor in their higher concordance

for autism.) But even if autism were caused by faulty genes, it would do little to strengthen Miele's argument. Clearly, the fact that there are many true genetic disorders, such as Huntington's Disease, does not mean that human psychological trait variation in general has a genetic component, just as the fact that brain tumors exist does not mean that abnormal behavior in general is caused by various brain diseases. But it just so happens that there is little scientifically acceptable evidence in support of autism as a genetic disorder.

One can only welcome an ongoing exchange on nature-nurture issues in future editions of *SKÉPTIC*. A major focus, in my view, would be a critical assessment of the validity and usefulness of key behavior genetic concepts such as twin research, adoption research, and "heritability," in addition to behavior geneticists' use of concepts such as "IQ" and "personality."

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Nature and Nurture: Putting All the Pieces Together—Miele Replies to Joseph and Schlinger

I welcome this opportunity to respond to Jay Joseph and Henry Schlinger, and to tell *SKÉPTIC* readers why our viewpoints are so very different and why I believe the preponderance of evidence supports mine.

Reply to Jay Joseph

Joseph rejects heritability estimates based on twin and adoption studies by calling into question a number of assumptions that are part of the standard behavior genetic models.

• *The Equal Environment*

Assumption. We know that monozygotic (MZ or identical) twins, who share all of their genes in common, are often treated more alike, for example, being dressed similarly or sharing the same friends, than are dizygotic (DZ) twins. So environmental factors, not genes, could account for their greater similarity. The critical question, however, is not whether MZ twins are in some ways

treated more similarly than DZs, but whether such treatment has an effect on the trait that is being measured, such as IQ or personality.

Loehlin and Nichols found that the greater similarity of identical twins in terms of being dressed alike, playing together as children, spending time together, having the same teachers in school, and sleeping in the same bedroom could not "plausibly account for more than a very small fraction of their greater observed similarity on the personality and ability variables" they studied.¹ Numerous other studies have confirmed these findings.²

Particularly interesting are the cases where parents have misclassified their twins thinking that MZs were really DZs and vice versa. Loehlin and Nichols studied the correlation between parental misclassifications and similarity in parental treatment. The correlations were negligible and agreed with Scar's

earlier study.³ Even parental opinion about zygosity is not as critical in producing the observed similarity between twins or the fact that correlations for MZs are higher than those for DZs.

• *The Prenatal Environment.* It has also been argued, not unreasonably, that the prenatal environment is critical, since MZ twins usually share the same placenta, while DZs twins never do. Could the common prenatal environment account for the greater similarity of MZ twins?

A recent study with a large and representative sample indicates that the prenatal environment is far less powerful than the genes. The correlations in IQ were: .83 for 173 pairs of MZ twins who shared the same placenta (technically, the same chorion), .82 for 95 MZ pairs who did not, and .44 for 181 pairs of DZ twins.⁴ Twin researcher Nancy Segal summarized the research on prenatal effects on twin similarity:

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