

**The Genetics of Political Attitudes and Behavior:**  
**Claims and Refutations**

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### **Abstract**

Some political scientists have argued in recent years that twin research shows that genetic factors play an important role in shaping political attitudes, ideologies, and behavior. Moreover, some researchers claim to have identified genes for political traits at the molecular level. The author argues that the main theoretical assumption of the twin method, which holds that monozygotic and dizygotic twin pairs experience equal environments, is untenable. Therefore, the results of twin studies can be completely explained by non-genetic factors. The author also argues that recent gene discovery claims in political science are unlikely to be replicated. He concludes that because genetic interpretations of twin study results are confounded by environmental factors, political scientists have no reason to revise previous socialization theories of political traits.

**KEYWORDS:** Behavioral genetics, equal environment assumption, genetics, political science, twin study, molecular genetics, voting.

### List of Abbreviations

CPI	California Psychological Inventory
CTD	Classical Twin Design (the twin method)
DZ	Dizygotic Twin Pairs (reared together)
DZA	Dizygotic Twin Pairs (reared apart)
DZSS	Same-Sex Dizygotic Twin Pairs (reared together)
DZOS	Opposite-Sex Dizygotic Twin Pairs (reared together)
EEA	Equal Environment Assumption
GU	Genetically Unrelated
MISTRA	Minnesota Study of Twins Reared Apart
MZ or MZT	Monozygotic Twin Pairs (reared together)
MZA	Monozygotic Twin Pairs (reared apart)
TRA	Twins Reared Apart

### **Politics, Genetics, and the Twin Method**

Since at least 2005, political scientists Alford, Funk, and Hibbing (2005) and others have argued that differences in political orientation and behavior have an important genetic basis (others making such claims include Alford, Funk, & Hibbing, 2008a, 2008b; Bell, Shermer, & Vernon, 2009; Fowler & Dawes, 2008; Fowler, Baker, & Dawes, 2008; Hatemi, Medland, Morely, Heath, & Martin, 2007; Hatemi, Alford, Hibbing, Martin, & Eaves, 2009; Hatemi, Medland, & Eaves, 2009; Medland & Hatemi, 2009; Hatemi et al., 2010). Intuitively we might reject such an idea out of hand, yet the past few years have seen claims that the link between genes and political behavior and attitudes has been established by twin research. This has led to the creation of the nascent field of “genopolitics,” and to the claim that there is a “developing consensus that genes play an important role in political behavior” (Settle, Dawes, & Fowler, 2009, p. 601).

It is my understanding that political scientists take quantitative empirical methodology very seriously. Thus, one can only welcome a thorough evaluation of twin research by this field. This does not usually occur in psychiatry and psychology, where journals regularly publish the results of behavioral genetic research with little or no critical analysis. Conversely, genetic theories and claims have sparked a debate in political science on the validity of twin research. (For criticism of twin research in political science, see Beckwith & Morris, 2008; Charney, 2008a, 2008b, 2010; Suhay, Kalmoe, & McDermott, 2007. For responses to these critics, see Alford et al. 2008a, 2008b; Hannagan & Hatemi, 2008).

What concerns us here is the possible role of genetic influences on individual differences in political attitudes and behavior, not the undisputed fact that human beings are the product of both their genes and their environments. Political scientists Hannagan and Hatemi (2008) stated

the obvious when they wrote, mistakenly implying that critics of genetic research disagree, “the scientific community recognizes that genes are very much a part of what it means to be human” (p. 332). We might as well say that the scientific community recognizes that Barack Obama won the 2008 United States presidential election. Human behavioral genetic researchers, however, are concerned with trait *variation* in the population, and usually conclude that heredity plays an important role in explaining this variation.

In their 2005 twin study, Alford and colleagues concluded that “genetics plays an important role in shaping political attitudes and ideologies . . .” (Alford et al., 2005, p. 153). They reached this conclusion on the basis of finding a significantly higher correlation of MZ (monozygotic, identical) versus same-sex DZ (dizygotic, fraternal) twin pairs on Wilson-Patterson Attitude Inventory scores. They used the “classical twin method” (hereafter the “twin method”; political science twin researchers sometimes refer to the twin method as the “classical twin design,” or “CTD”). Specifically, the twin method compares the trait resemblance of reared together MZ twin pairs, who share a 100% genetic similarity, versus the resemblance of reared together same-sex DZ twin pairs, who average a 50% genetic similarity. Based on the fact that MZ pairs share a greater genetic resemblance than DZ pairs, twin researchers believe that genetic factors explain a finding of significantly greater behavioral or attitude resemblance among MZ versus same-sex DZ twin pairs.

A critical theoretical assumption of the twin method, which allows researchers to conclude in favor of genetics, states that the childhood and adult environments of both types of twins are comparable. This is known as the *equal environment assumption* (or “EEA”).

As both twin researchers and their critics recognize, the validity of the equal environment assumption is central. Indeed, in their twin study publication Alford and colleagues wrote,

This assertion that the effect of genetics is measurably distinct for MZ and DZ twins, while the effect of the environment is either equivalent or at least randomly distributed around equivalence [EEA], *is crucial to everything that follows from twin research*. (Alford et al., 2005, p. 155) [italics added]

And the authors of a subsequent publication recognized that “no feature of” the twin method “has generated more attention and concern” than the EEA (Hatemi et al., 2010, p. 800).

Alford and colleagues cited evidence which they believed upheld the validity of the equal environment assumption. While critics Beckwith and Morris agreed that “the EEA is essential for conclusions drawn from twin studies concerning human behavioral traits,” they concluded “that the EEA has not been well tested nor validated,” thereby casting doubt on Alford and colleagues’ original conclusions in favor of genetics (Beckwith & Morris, 2008, p. 788). Political scientists Evan Charney (2008a, 2008b, 2010), and Elizabeth Suhay and colleagues (2007), have also questioned the validity of the equal environment assumption and the twin method. My purpose here is (1) to elaborate further on the untenable basis of the EEA, (2) to show that the twin method is no more able to disentangle potential genetic and environmental factors than is a family study, and (3) to suggest that it is very unlikely that “genes for” political attitudes and behavior have been found, or will ever be found.

Twin researchers and most of their critics agree that MZ twin pairs resemble each other more (i.e., correlate higher) than same-sex DZ pairs for most behavioral and psychological traits. The key question, however, is the determination of what factor or factors explain this finding. Behavioral geneticists argue that genetic factors provide the best explanation (e.g., Bouchard & McGue, 2003; Plomin, DeFries, McClearn, & McGuffin, 2008; Rutter 2006), whereas critics frequently argue that environmental factors play a large or exclusive role.

On the basis of the ubiquitous finding that MZ pairs resemble each other more than DZ pairs for behavioral traits, it should have come as no surprise to either genetic researchers, or to

proponents of a purely social theory of political attitudes and behavior, that Alford et al. found MZ pairs to resemble each other more than DZ pairs for political attitudes as well. Political scientists influenced by behavioral genetics argue that this finding provides solid evidence that genetic factors play an important role (see for example Carmen 2007; Fowler & Schreiber, 2008). Thus, political behavior joins a long list of other intuitively non-genetic behaviors now claimed for genetics on the basis of twin research. Among these are included anorexia (Bulik et al., 2006; Wade, Bulik, Neale, & Kendler, 2000), breakfast eating patterns (Keski-Rahkonen, Viken, Kaprio, Rissanen, & Rose, 2004), frequency of orgasm in women (Dawood, Kirk, Bailey, Andrews, & Martin, 2005), loneliness (Boomsma, Willemsen, Dolan, Hawkey, & Cacioppo, 2005), perfectionism (Tozzi et al., 2004), and religiousness (Koenig, McGue, Krueger, & Bouchard, 2005).

There are two main conclusions that we can reach on the basis of such research:

- 1) **Twin Researchers' Conclusion**: The greater resemblance of MZ vs. same-sex DZ twin pairs provides solid evidence that a sizable portion of the population variance of these and other traits can be explained by genetic factors, **or**
- 2) **Twin Method Critics' Conclusion**: The twin method is a faulty instrument for assessing the role of genetics, given the likelihood that MZ vs. same-sex DZ comparisons measure environmental rather than genetic influences. Therefore, all previous interpretations of the twin method's results in support of genetics are not supported by the evidence.

Here, I argue that Conclusion #2 is the correct one, and that it is unlikely that the twin method measures anything other than the more similar treatment, socialization resemblance, environment, and emotional bond experienced by MZ vs. DZ twin pairs.

**The two main definitions of the equal environment assumption.** As most people intuitively understand, MZ twin pairs experience much more similar environments than do DZ pairs. This has been recognized not only by some early twin researchers and commentators (see

Joseph, 2004, chapter 2), but by leading contemporary behavioral geneticists as well. Some examples from the latter group are as follows:

- Sandra Scarr and Louise Carter Saltzman in 1979: “The evidence of greater environmental similarity for MZ than DZ twins is overwhelming” (Scarr & Carter-Saltzman, 1979, p. 528).
- David Rowe in 1994: “The question is not whether MZ twins receive more similar treatments (they do, and to claim otherwise would be foolish), but whether these treatments influence a particular trait” (Rowe, 1994, p. 45).
- Stephan Faraone and colleagues in 1999: “Several studies have found that the social environments of MZ twins are more similar than those of DZ twins. For example, habits, activities, personal preferences, parental treatment, and self-image tend to be more similar between MZ twins. Moreover, MZ twins are more likely to be dressed alike and are more likely to be confused for one another in childhood” (Faraone, Tsuang, & Tsuang, 1999. p. 38).
- Thomas Bouchard, Jr. and Matt McGue in 2003: “MZ twins are more likely than DZ twins to share friends and parental treatment in adolescence” (Bouchard & McGue, 2003, p. 9).
- Kenneth Kendler and Carol Prescott in 2006: “Consistent with other studies, we found evidence that some aspects of the environment of members of MZ pairs are, on average, more similar than those of members of DZ pairs” (Kendler & Prescott, 2006, p. 124).

The EEA has been defined in two main ways since the development of the twin method in the 1920s. The first is the *traditional definition*, which was the only definition twin researchers used until the mid-1960s, and states simply that MZ and same-sex DZ twin pairs experience roughly equal childhood and adult environments. Most twin researchers now recognize that this definition of the EEA *is false* (for example, see Kendler 1983; Rowe 1994). Thus, in the 1960s they changed the definition of the EEA to the currently used *trait-relevant definition* (see Gottesman & Shields, 1966). For example, psychiatric genetic twin researcher Kenneth Kendler and his colleagues defined the EEA as follows:

The traditional twin method, as well as more recent biometrical models for twin analysis, are predicated on the equal-environment assumption (EEA)—that monozygotic (MZ) and

dizygotic (DZ) twins are equally correlated for their exposure to environmental influences *that are of etiologic relevance to the trait under study* [italics added]. (Kendler, Neale, Kessler, Heath, & Eaves, 1993, p. 21)

After subtly redefining the equal environment assumption by adding the *ad hoc* trait-relevant condition, some twin researchers then implied or stated that *critics* bear the responsibility of identifying trait-relevant environmental factors for which MZ and DZ twin pairs differ. An example of twin researchers reversing the burden of proof from themselves onto critics is seen in Alford and colleagues' response to Evan Charney's (2008a) critique of their study. After acknowledging that MZ pairs do experience more similar environments to some extent, they argued that the "central question" is whether these factors influence political beliefs, and that "no evidence has yet been presented that it does" (Alford et al., 2008a, p. 322). Other twin researchers attempting to place the burden of proof onto critics include Faraone and Biederman (2000), and Lyons et al. (Lyons, Kendler, Provet, & Tsuang, 1991).

However, as psychologist Scott Lilienfeld and his colleagues noted, "a basic tenet of science is that the burden of proof always falls squarely on the claimant, not the critic . . . . Consequently, it is up to the proponents of these techniques to demonstrate that they work, not up to the critics of these techniques to demonstrate the converse" (Lilienfeld, Lynn, & Lohr, 2003, p. 3).

Most twin researchers in political science use the trait-relevant definition of the equal environment assumption (Bell et al., 2009, did not discuss the EEA). According to Medland and Hatemi, one of the two "central questions of the equal environment assumption (EEA)" is "whether these differences [between MZs and DZs] influence the specific trait under analysis . . . ." (Medland & Hatemi, 2009, pp. 198-199). Like previous twin researchers, political scientists focus narrowly on factors such as dressing alike, or having common playmates as children, while

downplaying factors relating to the greater psychological and emotional bond experienced by MZ pairs, and their greater propensity to experience “ego fusion” and mutual association when compared with DZ pairs (Jackson, 1960; Kringlen, 1967).

**Do twins create their own environments?** A major argument that contemporary twin researchers put forward in defense of the equal environment assumption is that MZ pairs “create” or “illicit” more similar environments for themselves because they are more similar genetically than DZ pairs. In 1983, Kendler upheld the validity of the twin method mainly on the basis of this position, arguing that “the available evidence suggests that the similarity of the social environment of monozygotic twins is the result of the behavioral similarity of the twins” (Kendler, 1983, p. 1416), and that “the similar phenotypes in monozygotic twins are caused by their genetic similarity” (p. 1414).

Political science twin researchers also utilize this “twins create their own environment” argument. According to political scientist James Fowler and his colleagues, “although MZ twins are sometimes in more frequent contact with each other than DZ twins, it appears that twin [genetic] similarity (e.g., in attitudes and personality) may cause greater contact rather than vice versa” (Fowler et al., 2008, p. 235). For Medland and Hatemi, the second “central question” of the EEA is whether MZ-DZ “environmental differences are manifestations of the genetic similarity of MZ twins” (Medland & Hatemi, 2009, pp. 198-199). Finally, Sturgis and colleagues concluded that “MZ environments are more similar than DZ environments . . . *because of the initial difference in genetic predispositions*” (Sturgis et al., 2010, p. 222, italics in original).

However, to invalidate the EEA and the twin method it is necessary only to show *that* MZ pairs experience more similar environments. For example, suppose for genetic reasons that MZ pairs are more likely than DZ pairs to enjoy spending time at the beach together. Although

researchers might find higher skin cancer correlations among MZ versus DZ pairs, this does not mean that skin cancer is a genetically-based disease. This is similar to Charney's example of the genetic trait of skin color leading to enslavement as an indication of the "absolutely fallacious nature of the assumption that the effects of behavior which is a response to a 'genetic trait' should itself be counted as genetic." As Charney noted:

Are we to assume then, that the effects upon blacks of their enslavement by European whites were *genetic*, because slavery was "caused" or "elicited" or "created" by the genetic trait of black skin color? (Charney, 2008b, 337, italics in original)

Moreover, the "twins create their own environment" argument is based on circular reasoning in that twin researchers' contention that twins' resemblance for behavior, temperament, and personality is due to heredity is based implicitly on genetic interpretations of previous twin studies. The argument, therefore, assumes the very thing that needs to be demonstrated. Thus, modern twin researchers (including twin researchers in the political science field) circularly *assume* that twins' behavioral resemblance is caused by genetics, in order to *conclude* that twins' behavioral resemblance is caused by genetics (Joseph, in press).

Twin researchers have made the twin method difficult to falsify as they argue that the EEA is valid (a) if MZ and DZ pairs experience equal environments, **or** (b) if MZ and DZ pairs experience far different environments. As the critical psychiatrist R. D. Laing wrote long ago in response to a similar argument made by schizophrenia twin researcher Franz J. Kallmann, "With this two-headed penny it is not clear how Kallmann can lose" (Laing, 1981, p. 143)

**Twin studies and family studies.** Before performing twin studies, behavioral geneticists frequently use *family studies* to assess whether a trait is familial. Family studies attempt to determine whether the biological relatives of persons manifesting a given trait exhibit the trait more often than do members of the general population or a control group. Although the results

often show that a trait is *familial*, this does not mean that it is *genetic*. Most researchers in the behavioral sciences agree that family studies are unable to disentangle the potential roles of genetic and environmental factors (for example, see Bouchard & McGue, 2003; Faraone et al., 1999; Plomin et al., 2008). As behavioral geneticist Robert Plomin and his colleagues recognized, “Many behaviors ‘run in families,’ but family resemblance can be due to either nature or nurture” (Plomin et al., 2008, p. 70). And Merikangas and Risch wrote, “While family studies indicate the degree to which diseases aggregate in families, they alone cannot address the question of genetic versus environmental factors as the source of such aggregation” (Merikangas & Risch, 2003, p. 625).

Thus behavioral geneticists recognize that, because family members share a common environment as well as common genes, a trait “running in the family” can be completely explained on genetic *or* environmental grounds. In other words they recognize, correctly, that potential genetic and environmental influences cannot be disentangled in a family study.

Behavioral geneticists, however, imply or argue that there is a qualitative difference between family studies and twin studies. Although most recognize that MZs experience more similar environments than DZs, they maintain that the twin method is *not* confounded by environmental factors and therefore provides unequivocal evidence in favor of genetics.

This is a puzzling and contradictory position. If differing environments automatically invalidate genetic interpretations of family study data, then the differing environments of MZ vs. DZ twin pairs must automatically invalidate genetic interpretations of twin method data as well.

Family members experience a more similar environment and a greater psychological association with other family members than do randomly selected members of the general population. In the same way, MZ twin pairs experience a more similar environment and a greater

psychological association than DZ pairs. However, although the experimental (index) and control groups in both family studies and twin studies experience far different environments, behavioral geneticists and popularizers of their work approach family studies and twin studies as if they were completely different phenomena.

From the standpoint of environmental confounds, however, family studies and the twin method have precisely the *same* problem. This means that the twin method is merely a variation on the family study design, and is no more able than a family study to disentangle possible genetic and environmental influences on a trait.

**Testing the validity of the equal environment assumption.** Although most proponents of genetic theories in political science recognize that MZ twin pairs do indeed experience more similar environments than DZ pairs (e.g., Alford et al., 2005; Alford et al., 2008a; Hatemi, Alford, et al., 2009; Medland & Hatemi, 2009), they place great emphasis on a body of research in which the investigators tested, and in most cases claimed to have upheld, the validity of the EEA.

Ironically, the authors of most “EEA-test” studies confirmed that MZ pairs experience much more similar environments than DZ pairs (for example, Borkenau, Riemann, Angleitner, & Spinath, 2002; Kaprio, Koskenvuo, & Rose, 1990; Kendler & Gardner, 1998; LaBuda, Svikis, & Pickens, 1997; Lytton, 1977; Morris-Yates, Andrews, Howie, & Henderson, 1990; Scarr, 1968; Scarr & Carter-Saltzman, 1979). However, most researchers concluded that their findings supported the EEA on the basis of, broadly speaking, (1) their claim that greater environmental similarity or more frequent contact, or greater physical resemblance, is not associated with greater twin resemblance for psychological traits, and/or (2) their claim that MZ pairs create

more similar environments for themselves on the basis of their greater genetic similarity (the “twins create their own environment” argument).

One of the few research teams to test the EEA whose members lacked professional or philosophical allegiances to behavioral genetics and twin research was that of Alan Horwitz and his colleagues (Horwitz, Videon, Schmitz, & Davis, 2003). Horwitz et al. analyzed data from 414 twin pairs (230 MZ, 187 DZ) and assessed the relationship between several environmental variables. They concluded that “measures of the social environment sometimes reduce or eliminate apparent genetic affects,” suggesting that “past twin studies could overstate the effect of genetic influences because some similarities in behavior among monozygotic compared to dizygotic twins stem from social influences” (p. 111). Indeed, most schizophrenia twin researchers believed that MZ-DZ concordance rate differences are caused in part by non-genetic factors (see Joseph, 2004, pp. 171-175; concordance rates are used extensively in psychiatric twin research, and denote a finding that both members of a twin pair are diagnosed with the same disorder).

The EEA-test literature, then, is a body of research carried out by twin researchers focusing narrowly on areas that they claim support the EEA and the twin method. In doing so, they choose to de-emphasize the fact that their tests usually show that MZ pairs experience much more similar environments than DZs.

Going further, one could argue that the very idea that the EEA can be “tested” is faulty, and that we can evaluate the twin method’s validity only by looking at the larger picture of how MZ and DZ twin pairs exist in, and interact with, the social, political, and familial environments in which they live—in *precisely the same way that behavioral geneticists and others currently evaluate family studies*. Behavioral geneticists do not require critics to identify the “trait

relevant” features that lead to behavioral resemblance in family studies, nor do they argue that the results of family studies point to genetics because family members “create their own environments,” nor do they perform “family study test” research. It is time that they apply these standards to the twin method as well.

Twin researchers also frequently overlook empirical findings clearly inconsistent with the validity of the EEA. One of many such examples we find in the 85-year history of the twin method is that, when studied, *same-sex* DZ pairs (DZSS) usually correlate higher than *opposite-sex* DZ pairs (DZOS) for psychiatric disorders and behavioral traits. For example, the pooled DZSS concordance rate across all schizophrenia twin studies is 11.3% (59/523), but is only 4.7% (20/422) for DZOS pairs (Joseph, 2004). Because both types of twins share the same genetic—but not environmental or interpersonal—relationship with each other, on genetic grounds there should be no correlational differences between these two types of twin pairs for traits failing to show sex differences in the general population (Jackson, 1960). But in fact, there usually are.

Looking at the brief history of twin studies in political science, we find similar results: DZSS pairs correlate significantly higher than DZOS pairs on some politically-related traits (see Hatemi et al., 2007, Table 3; Hatemi, Alford, et al., 2009, Table 3. Alford et al., 2005, Fowler et al., 2008, and Bell et al., 2009, did not report DZOS correlations). In Hatemi and colleagues’ 2007 Australian twin study of voting behavior, the authors found “some substantial differences in the correlations of opposite sex [DZ] pairs compared to those for the same sex DZ pairs” (Hatemi et al. 2007, p. 442).

The problem this presents for the twin method is that higher DZSS vs. DZOS correlations are difficult to explain on genetic grounds (I am not aware of any genetic researcher attempting to do so since the inception of the twin method in the 1920s), but are consistent with the

argument that the twin method measures nothing more than differing levels of environmental influence and mutual association among various types of twin pairs. Thus, as psychiatrist Don Jackson (1960) argued long ago, environmental interpretations of twin method findings predict that, according to the degree of environmental similarity experienced by siblings and twins, we would expect greater behavioral resemblance, without concern for genetic relationship.

**The Martin et al. social attitudes twin study.** Alford and colleagues relied heavily on the 1986 Martin et al. twin study of social attitudes in support of their contention that co-twin contact, which they recognize is “higher for MZ than for DZ twins,” does “not predict the extent of [twins’] political similarity” (Alford et al., 2008b, p. 794). But in fact, the larger picture of Martin and colleagues’ results strongly suggests that greater contact *does* lead to greater correlation for social attitudes.

Predictably, Martin and colleagues found that MZ twin pairs correlate significantly higher than DZ pairs for various social attitudes. Although, as Alford and colleagues point out, these investigators reported little correlation between twins’ “frequency of contact” and “absolute intrapair difference in conservatism” (Martin et al., 1986, p. 4367), from a broader perspective their results are completely explainable on environmental grounds. The logic I present below, though plausible, is dismissed by behavioral geneticists both in and out of political science:

- 1) Environmental factors play a role in shaping social attitudes (twin method critics, Martin et al., Alford et al., and most behavioral geneticists would agree with this statement).
- 2) MZ twin pairs as a population experience more similar environments, and have more “frequency of contact,” compared with DZ twin pairs as a population (twin method critics, Alford et al., and most behavioral geneticists would agree with this statement).

- 3) MZ twin pairs resemble each other more (correlate higher) than DZ pairs for most social attitudes (some twin method critics, Alford et al., Martin et al., and most behavioral geneticists see this as a replicated scientific finding).
- 4) **CONCLUSION**: A plausible interpretation of the *entire* Martin et al. study is that it proves nothing about genetics, but does suggest that environmental similarity, and frequency of contact, lead to greater twin pair resemblance for social attitudes.

Taking this a step further, *one could argue that the entire body of twin method data (going back to the 1920s) provides no evidence in support of genetics, but does suggest strongly that the more similar environment experienced by MZ versus same-sex DZ twin pairs—which includes their closer emotional bond, more similar treatment by parents and others, greater frequency of contact, and more similar socialization—is the sole cause of MZ pairs’ greater resemblance for behavioral and psychological traits, and of their greater resemblance for psychiatric disorders.*

Thus Alford and colleagues and the EEA-test researchers, similar to the old story of the blind men and the elephant, emphasize the correlations they believe support the EEA, while overlooking numerous other correlations that point to the falseness of the EEA.

In the same way, given that twin studies are hopelessly confounded by environmental factors (just as family studies are hopelessly confounded by environmental factors), the results of Alford and colleagues’ 2005 twin study of political attitudes are completely explainable on environmental grounds. As David Rosenthal, a leading psychiatric genetic researcher of the 1960s and 1970s, concluded toward the end of his career, both family studies and twin studies are “confounded,” and “one can draw conclusions about them only at considerable risk” (Rosenthal, 1979, p. 25).

**The “mistaken zygosity” studies.** Alford and colleagues (2005, 2008a) and other twin researchers (e.g., Fowler et al., 2008) cite the results of the “mistaken zygosity” EEA-test studies in support of the twin method. These studies assess twins who misidentify, or whose parents misidentify, their zygosity status (whether a pair is MZ or DZ). Researchers then compare the resemblance of these misidentified pairs to correctly identified pairs in order to test the effects of “true” versus “perceived” zygosity on twin psychological trait resemblance. Although Alford and colleagues (2005) claimed that “the degree of correspondence between MZ twins surpasses DZ twins even in the large subpopulation of twins thought by their parents to be MZ twins” (p. 155), an examination of the data shows that some of the comparisons fall in the direction predicted by environmental theories (see Joseph, 2006, chapter 9).

Behavioral geneticist Sandra Scarr, the most well known researcher of mistaken zygosity among twins, ended her frequently cited study as follows:

The critical assumption of equal environmental variance for MZ and DZ twins is tenable. Although MZ twins generally experience more similar environments, this fact seems to result from their genetic similarities and not to be a cause of exaggerated phenotypic resemblance. (Scarr & Carter-Saltzman, 1979, p. 541)

Thus, Scarr’s entire argument rested on the claim that the twin method is valid because MZ pairs create more similar environments for themselves on the basis of their greater genetic similarity. We have seen, however, that this circular “twins create their own environment” argument lends no support to the EEA. Thus, we see that another pillar of Alford and colleagues’ defense of the twin method rests on shaky ground indeed.

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In conclusion, the widely recognized greater environmental similarity of MZ versus DZ twins *invalidates the twin method on its face*, meaning that the twin method is confounded by environmental factors regardless of what EEA-test researchers claim. What they actually must

demonstrate—without qualification—is that MZ and same-sex DZ twin pairs experience roughly equal environments. The validity of twin method can be determined only by assessing *whether*—not *why*—MZ twins experience more similar environments than same-sex DZs (see Joseph 2004, 2006, in press).

### **Twins Reared Apart Studies**

According to Alford and colleagues, “the most powerful refutation” of criticism of the twin method “comes in recent studies utilizing MZ and DZ twin raised apart. These studies uniformly validate MZ and DZ differences found in earlier studies of twins raised together” (Alford et al., 2005, p. 155). Other political science twin researchers have also cited twins reared apart (known as “TRA”) studies in defense of the EEA and the twin method, such as Fowler and colleagues writing, “studies of twins raised together have been validated by studies of twin reared apart” (Fowler et al., 2008, 235; see also Settle et al., 2009). The most well known TRA investigation, the Minnesota Study of Twin Reared Apart (known as MISTRA), was carried out by Thomas J. Bouchard, Jr. and colleagues in the 1980s and 90s (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990).

Twins reared apart studies compare the psychological trait resemblance (e.g., personality, IQ) of purportedly reared apart MZ pairs (“MZAs”) to that of reared together MZs (“MZTs”). Although some studies have included reared apart DZ pairs (“DZAs”), claims in favor of genetics are usually made on the basis of MZA-MZT comparisons. TRA (twins reared apart) researchers usually conclude that, because MZA correlations for psychological traits are far greater than zero, and are comparable to MZT correlations, their study supports an important role for genetic factors and supports only a small role for shared family influences.

Critics, however, have pointed to several key methodological problems with TRA studies (TRA study critics include Farber, 1981; Joseph, 2001, 2004, in press; Kamin, 1974; Kamin & Goldberger, 2002; Lewontin, Rose, & Kamin, 1984; Taylor, 1980). These problems include (1) the doubtful “separation” of twins, who frequently grew up together and had contact over much of their lives; (2) similarity bias in the methods of MZA identification and recruitment; (3) the questionable status of “intelligence” and “personality” as valid and quantifiable constructs; (4) the failure of the MISTRA researchers to publish or share raw data and life history information for the twins under study, and (5) the impact that the researchers’ bias in favor of genetic interpretations may have had on their results and conclusions.

While these and other issues are important, the main problem with TRA studies such as Bouchard’s MISTRA is clear: The investigators used the wrong control group (MZTs). By using MZTs as controls, they failed to control for several key environmental factors shared by both MZA *and* MZT pairs (see Joseph, 2004; Rose, 1982). Environmental influences shared by both MZAs and MZTs include, but are not limited to:

- They are exactly the same age (birth cohort)
- They are always the same sex
- They are almost always the same ethnicity
- Their appearance is strikingly similar (which will elicit more similar treatment from the social environment)
- They usually are raised in the same socioeconomic class
- They usually are raised in the same culture
- They shared the same prenatal environment
- Most studied pairs spent a certain amount of time together in the same family environment, were aware of each other’s existence when studied, and often had regular contact over long periods of time (Farber, 1981; Kamin, 1974).

Although the Minnesota TRA studies adjusted correlations to take into account age and sex effects (McGue & Bouchard, 1984), these adjustments were inadequate and at best account for

only two environmental variables shared by MZAs. We should therefore expect, on purely environmental grounds, that MZAs reared apart from birth (and very few were; see Farber, 1981) would correlate well above zero for psychological and behavioral traits.

A major reason is that MZA correlations are heavily influenced by *cohort effects*, which account for similarities in people's behaviors and preferences that arise from the characteristics of the historical periods and cultural milieu (including the political milieu) in which they experience stages of life at the same time. In other words, we would expect two genetically-unrelated adults of the same gender, who are born at the same time, to resemble each other more for psychological traits, behaviors, tastes, political behavior and attitudes, etc. than would two randomly selected members of the population, spanning the entire adult age range.

Thus, for reasons having nothing to do with heredity, we should expect to find a much higher "video game playing behavior" correlation in the United States among pairs of randomly selected 15-year-old boys than we would expect to find among randomly selected pairs drawn from the entire 15-100-year-old male and female population of the United States (Joseph, in press). This example illustrates one of the central fallacies of twins reared apart studies.

As evidence of the effects of common age alone, a 1981 study (Martin, Blair, Dannenmaier, Jones, & Asako, 1981) looked at the relationship between age and personality among genetically-unrelated people (non-twins). The researchers found an average correlation between age, and personality scale scores on the California Psychological Inventory (CPI), of .28 across all 18 scales, with 10 scales showing a correlation of .35 or higher. If these findings reflect age effects in the general population, the influence of common age, which represents only one of many environmental variables shared by MZAs (see above), accounts for more than half of the reported MZA personality correlations. (The CPI was used in the MISTRA studies.)

In order to control for the influence of cohort effects, a scientifically valid TRA study would compare the resemblance of MZA pairs reared apart from birth and unknown to each other, versus a control group consisting not of MZT pairs, but rather of *genetically-unrelated pairs of strangers* who (1) are the same age, (2) are the same sex, (3) share the same ethnicity and culture, (4) share a similar socioeconomic status, and (5) are similar in appearance and attractiveness (Joseph, 2004, in press). Moreover, both the MZA and genetically-unrelated (GU) pairs should have no contact with each other until after they are evaluated and tested. After concluding such a study, we might find that GU pair and MZA pair correlations are similar, which would suggest that MZA correlations are the result of non-genetic influences. I am unaware of any attempt to compare MZA and GU pairs as I have described above, and for this and other reasons we can draw no valid conclusions in support of genetics—or in support of the validity of the twin method—on the basis of twins reared apart (TRA) studies published to date (Joseph, 2004, in press).

Although Alford, Funk, and Hibbing (2005, p. 155) implied that the MISTRA researchers' main comparison was between MZAs and DZAs, we have seen that the main comparison in TRA research is between MZA and MZT pairs (for example, see Bouchard et al., 1990). The MISTRA, however, was the first twins reared apart study to collect a sizable sample of DZAs. Due to MZAs' identical genetic makeup, of course, from the genetic standpoint they *must* correlate significantly higher than DZAs. However, the MISTRA results, though selectively published, failed to demonstrate that same-sex DZAs correlate much differently than MZAs on personality measures and IQ tests (see Joseph, 2004, pp. 130-33; Kamin & Goldberger, 2002).

Finally, we could conduct a thought experiment on political behavior and attitude formation among reared apart MZ twin pairs who, although genetically identical, grow up in

*truly* uncorrelated environments in different eras. Suppose one male MZA twin is placed at birth in an aristocratic Japanese family in 1802. The other male MZA co-twin is placed at birth in a poor peasant family living in the highlands of El Salvador in 1960. Unlike previous TRA studies, in which the investigators calculated correlations among *partially* reared apart twins sharing *many* cultural influences (Farber 1981), in our thought experiment we eliminate cultural influences such as family (which most MZAs share to some extent), and mutual association and influence (which most MZAs also share to some extent). We also reduce or eliminate national, regional, political, ethnic, religious, economic class, and birth cohort influences. (Of course, they would still be the same sex.)

I conclude this thought experiment by posing the following question: Would we expect a study of genetically identical pairs of this type to find sizable correlations for political behavior and social attitudes?

### **Genes for Political Behavior?**

In 2008, Alford and colleagues attempted to bolster their argument further by claiming that the “preliminary results” of efforts to identify genes associated with political behavior at the molecular level, using genome-wide scans and allelic association tests, “are promising” (Alford et al., 2008b, p. 795). However, we have seen similar subsequently non-replicated claims in psychiatry and psychology since the 1970s. (As an early example, manic-depression genetic researchers Mendlewicz & Rainer, 1977, p. 327, wrote that a “genetic vulnerability to manic-depressive disorder has been demonstrated by family, twin, and [molecular genetic] linkage studies.”)

As I write this, the fact remains that, although there have been literally thousands of false positive claims (see below), gene finding efforts in psychiatry and psychology have *failed* to

discover any genes shown by consistent replication of association, and by evidence that the association is causal, to underlie psychiatric disorders (Akil et al., 2010). This is true for schizophrenia (Bergen et al., 2010; Need et al., 2009), bipolar disorder (Craddock & Sklar, 2009), autism (Burmeister, McInnis, & Zöllner, 2008), attention-deficit/hyperactivity disorder (Faraone, Doyle, et al., 2008), and variation in psychological traits (e.g., IQ, personality; Plomin et al., 2008; Smoller, Sheidley, & Tsuang, 2008).

In the words of a leading group of psychiatric geneticists, writing in 2008, “It is no secret that our field has published thousands of candidate gene association studies but few replicated findings.” (Faraone, Smoller, Pato, Sullivan & Tsuang, 2008, p. 1). To locate these “thousands” of false positive findings, one can simply scan the past 20 or so years of online abstracts for journals such as *Molecular Psychiatry*, *The American Journal of Psychiatry*, *Archives of General Psychiatry*, *The American Journal of Human Genetics*, *The American Journal of Medical Genetics (Part B)*, *Psychiatric Genetics*, and *Behavior Genetics* (a 2006 edition featured an article describing the discovery of a gene for “loneliness”; Boomsma, Cacioppo, Slagboom, & Postuma, 2006). As molecular genetic researcher Neil Risch and his colleagues concluded in a 2009 article in *The Journal of the American Medical Association*, “few if any” genes have been identified:

Despite progress in risk gene identification for several complex diseases, few disorders have proven as resistant to robust gene finding as psychiatric illnesses. The slow rate of progress in psychiatry and behavioral sciences partly reflects a still-evolving classification system, absence of valid pathognomonic diagnostic markers, and lack of well-defined etiologic pathways. Although these disorders have long been assumed to result from some combination of genetic vulnerability and environmental exposure, direct evidence from a specific example has not been forthcoming. Few if any of the genes identified in candidate gene association studies of psychiatric disorders have withstood the test of replication. (Risch et al., 2009, p. 2363)

Turning to the search for the genes believed to underlie general cognitive ability (IQ), in 2008 Plomin and colleagues recognized that, after the initial failures of the mid-1990s, “Dozens of studies have subsequently explored other candidate gene associations with g [general cognitive ability] but none have shown consistent results” (Plomin et al., 2008, p. 170). Efforts to identify genes for personality have experienced a similar fate. According to Plomin et al., “replication of [personality] associations has been difficult” (p. 263).

Writing as if a generation of false positive (yet highly publicized) “gene discoveries” did not happen, Alford et al. claimed that “Scholars . . . have uncovered genes involved with reading disorders, depression, autism, risk-taking, and attention deficit hyperactivity disorder” (Alford et al. 2008a, p. 324). And a pair of political science genetic researchers made similar claims with regard to alleged “discoveries of genes that influence depression, autism, obesity, and host of important social and medical traits” (Medland & Hatemi, 2009, p. 211). Again, despite countless subsequently unsubstantiated gene finding *claims* in these areas, no verified discoveries have been made for psychiatric disorders or variation in psychological traits.

In their 2008 molecular genetic study, Fowler and Dawes found that individuals with a particular genetic variation “are significantly more likely to have voted in the 2004 presidential election.” They continued, “These are the first results ever to link specific genes to political behavior” (Fowler & Dawes, 2008, p. 579). However, as Risch and colleagues noted, virtually all such “results” in molecular genetic behavioral research turn out to be false positives (in addition to the fact that an “association” or “link” does not equal “cause”). This means that the claims put forward by Alford and colleagues, Fowler and Dawes, and others are potentially irresponsible given the inevitable reports in the media trumpeting yet another non-existent “gene discovery.”

The journalists filing such reports usually fail to mention the fact that subsequent replication attempts almost always fail to substantiate such findings.

It is clear that molecular genetic research in the behavioral sciences is massively plagued by false positive results, and that systematic error has been repeated year-after-year, and decade-after-decade (Joseph, 2006, in press; see also Ioannidis, 2005; Wacholder, Chanock, Garcia-Closas, El ghormli, & Rothman, 2004). Unfortunately, researchers usually fail to consider the possibility that genes for psychiatric disorders and variation in behavioral traits do not exist.

Thus, there is every reason to believe that molecular genetic researchers in political science have painted themselves into the very same corner as the behavioral genetic and psychiatric genetic researchers before them. They have landed in this corner due to their mistaken belief that the twin method produces unequivocal evidence that genetic factors contribute to the observed variation in political behavior and attitudes. This means that gene finding efforts in political science, despite recent claims, are unlikely to bear fruit.

### **Summary and Conclusions**

Recent claims in favor of important genetic influences on differences in political behavior, ideologies, and attitudes are based (1) on genetic interpretations of the results of studies using the twin method, and (2) on claims that genes associated with political behavior have been discovered at the molecular level. I have chosen to overlook many other methodological problem areas in twin research in order to focus on the Achilles Heel of the twin method, which is the clearly false theoretical assumption that MZ and DZ twin pairs experience equal environments.

I agree with Evan Charney (2008a) when he wrote that the twin method in general, and Alford and colleagues' 2005 study in particular, is "based upon a faulty paradigm" (p. 311), with Beckwith and Morris (2008), who concluded that the Alford et. al. twin study "is of dubious scientific value" (p. 785), and with Elizabeth Suhay and colleagues (2007), who wrote, "heritability claims based on twin studies generally cannot be trusted due to the confounding influence of the greater environmental similarity of MZ twins" (p. 16).

Because the twin method is no more able than a family study to disentangle potential genetic and environmental influences, twin studies of political attitudes and behavior provide no scientifically acceptable evidence in support of genetic factors. Moreover, recent gene finding claims use the same methodology that has led to 30 years of false-positive findings for behavioral traits and psychiatric disorders, and there is every reason to believe that these claims will also suffer the same fate.

Clearly, the unconvincing body of behavioral genetic research produced during the last six years has provided no reason for political scientists to revise previous theories emphasizing social and environmental explanations of concepts such as political attitude formation, party affiliation, voter preferences and turnout, the "left-right spectrum," the political "gender gap," "social trust," and so on.

Alford and colleagues (2008b) wrote that "concerns with twin studies have been raised and rebutted before" (p. 793), and Hatemi and colleagues (Hatemi, Alford, et. al., 2009) wrote, "the present [EEA] debate simply recapitulates that which was exhausted in psychology and psychiatry more than 20 years ago" (p. 586). In fact, twin researchers have never been able to refute the critics (Joseph, 2004). Instead, they have overlooked abundant evidence against the validity of the equal environment assumption while constructing questionable *ad hoc* hypotheses

in order to salvage the twin method. The twin method survives today not because the critics have been successfully “rebutted,” but rather for the same reason that critical behavioral geneticist Douglas Wahlsten gave for the endurance of heritability analysis, which he viewed as “the outcome of a power struggle, not the resolution of a debate among scientists” (Wahlsten, 1994, p. 254; for a critique of behavioral geneticists’ use of the heritability statistic, see Joseph, 2004, chapter 5).

Beckwith and Morris (2008) concluded by urging “political scientists to take a more critical look at the studies that supposedly provide the foundation for this field” (p. 788). At the outset of this article I welcomed political scientists’ detailed scrutiny of the assumptions of the twin method, and noted that there has not been enough such scrutiny in the fields of psychiatry and psychology. It may be that behavioral genetics has finally ventured into a field that will examine its claims with the utmost scrutiny, and that the twin method will not survive this scrutiny.

## REFERENCES

- Akil, H., Brenner, S., Kandel, E., Kendler, K. S., King, M., Scolnick, E., Watson, J. D., Zoghbi, H. Y. (2010). The future of psychiatric research: Genomes and neural circuits. *Science*, 327, 1580-1581.
- Alford, J. R., Funk, C. L., & Hibbing, J. R. (2005). Are political orientations genetically transmitted? *American Political Science Review*, 99, 153-167.
- Alford, J. R., Funk, C. L., & Hibbing, J. R. (2008a). Beyond liberals and conservatives to political genotypes and phenotypes. *Perspectives on Politics*, 6, 321-328.
- Alford, J. R., Funk, C. L., & Hibbing, J. R. (2008b). Twin studies, molecular genetics, politics, and tolerance: A response to Beckwith and Morris. *Perspectives on Politics*, 6, 793-797.
- Beckwith, J., & Morris, C. A. (2008). Twin Studies of Political Behavior: Untenable Assumptions? *Perspectives on Politics*, 6, 785-791.
- Bell, E., Shermer, J. A., & Vernon, P. A. (2009). The origins of political attitudes and behaviors: An analysis using twins. *Canadian Journal of Political Science*, 42, 855-879.
- Bergen, S. E., Fanous, A. H., Kuo, P-H., Wormley, B. K., O'Neill, F. A., Walsh, D., Riley, B. P., & Kendler, K. S. (2010). No association of dysbindin with symptom factors of schizophrenia in an Irish case-controls sample. *American Journal of Medical Genetics (Part B)*, 153B, 700-705.
- Boomsma, D. I., Cacioppo, J. T., Slagboom, P. E., & Posthuma, D. (2006). Genetic linkage and association analysis for loneliness in Dutch twin and sibling pairs points to a region on chromosome 12q23-24. *Behavior Genetics*, 36, 137-146.
- Boomsma, D. I., Willemsen, G., Dolan, C. V., Hawkey, L. C., & Cacioppo, J. T. (2005). Genetic and environmental contributions to loneliness in adults: The Netherlands Twin Register Study. *Behavior Genetics*, 35, 745-752.
- Borkenau, P., Riemann, R., Angleitner, A., & Spinath, F. M. (2002). Similarity of childhood experiences and personality resemblance in monozygotic and dizygotic twins: A test of the equal environments assumption. *Personality and Individual Differences*, 33, 261-269.
- Bouchard, T. J., Jr., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of human psychological differences: The Minnesota Study of Twins Reared Apart. *Science*, 250, 223-228.
- Bouchard, T. J., Jr., & McGue, M. (2003). Genetic and environmental influences on human psychological differences. *Journal of Neurobiology*, 54, 4-45.

Bulik, C. M., Sullivan, P. F., Tozzi, F., Furberg, H., Lichtenstein, P., & Pedersen, N. L. (2006). Prevalence, heritability, and prospective risk factors for anorexia nervosa. *Archives of General Psychiatry*, 63, 305-312.

Burmeister M., McInnis, M. G., & Zöllner S. (2008). Psychiatric genetics: progress amid controversy. *Nature Reviews Genetics*, 9 (7), 527-540.

Carmen, I. H. (2007). Genetic configurations of political phenomena: New theories, new methods. *Annals of the American Academy of Political and Social Science*, 614, 34-55.

Charney, E. (2008a). Genes and Ideologies. *Perspectives on Politics*, 6, 292-319.

Charney, E. (2008). Politics, genetics, and “greedy reductionism.” *Perspectives on Politics*, 6, 337-343.

Charney, E. (2010). Twin studies, post genomics, and behavioral plasticity. Retrieved July 10th, 2010, from <http://www.sanford.duke.edu/research/papers/SAN10-01.pdf>.

Craddock, N., & Sklar, P. (2009). Genetics of bipolar disorder: Successful start to a long journey. *Trends in Genetics*, 25, 99-105.

Dawood, K., Kirk, K. M., Bailey, J. M., Andrews, P. W., & Martin, N. G. (2005). Genetic and environmental influences on the frequency of orgasm in women. *Twin Research and Human Genetics*, 8, 27-33.

Faraone, S. V., & Biederman, J. (2000). Nature, nurture, and attention deficit hyperactivity disorder. *Developmental Review*, 20, 568-581.

Faraone, S. V., Doyle, A. E., Lasky-Su, J., Sklar, P. B., D'Angelo, E., Gonzalez-Heydrich, J., Kratochvil, C., Mick, E., Klein, K., Rezac, A. J., & Biederman, J. (2008). Linkage analysis of attention deficit hyperactivity disorder. *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics)*, 147B, 1387-1391.

Faraone, S. V., Smoller, J. W., Pato, C. N., Sullivan, P., & Tsuang, M. T. (2008). The new neuropsychiatric genetics. *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics)* 147B, 1-2.

Faraone, S. V., Tsuang, M. T., & Tsuang, D. W. (1999). *Genetics of mental disorders*. New York: The Guilford Press.

Farber, S. L. (1981). *Identical twins reared apart: A reanalysis*. New York: Basic Books.

Fowler, J. H., Baker, L. A., & Dawes, C. T. (2008). Genetic variation in political participation. *American Political Science Review*, 102, 233-248.

Fowler, J. H., & Dawes, C. T. (2008). Two genes predict voter turnout. *Journal of Politics*, 70, 579-594.

Fowler, J. H., & Schreiber, D. (2008). Biology, politics, and the emerging science of human nature. *Science*, 322, 912-914.

Gottesman, I. I., & Shields, J. (1966). Contributions of twin studies to perspectives on schizophrenia. In B. Maher (Ed.), *Progress in experimental personality research* (Vol. 3; pp. 1-84). New York: Academic Press.

Hannagan, R. J., & Hatemi, P. K. (2008). The threat of genes: A comment on Evan Charney's "Genes and Ideologies." *Perspectives on Politics*, 6, 329-334.

Hatemi, P. K., Alford, J. R., Hibbing, J. R., Martin, N. G., & Eaves, L. J. (2009). Is there a "party" in your genes? *Political Research Quarterly*, 62, 584-600.

Hatemi, P. K., Hibbing, J. R., Medland, S. E., Keller, M. C., Alford, J. R., Smith, K. B., Martin, N. G., & Eaves, L. J. (2010). Not by twins alone: Using the extended family design to investigate genetic influence on political beliefs. *American Journal of Political Science*, 54, 798-814.

Hatemi, P. K., Medland, S. E., & Eaves, L. J. (2009). Do genes contribute to the "gender gap"? *Journal of Politics*, 71, 262-276.

Hatemi, P. K., Medland, S. E., Morley, K. I., Heath, A. C., & Martin, N. G. (2007). The genetics of voting: An Australian twin study. *Behavior Genetics*, 37, 435-448.

Horwitz, A. V., Videon, T. M., Schmitz, M. E., & Davis, D. (2003). Rethinking twins and environments: Possible social sources for assumed genetic influences in twin research. *Journal of Health and Social Behavior*, 44, 111-129.

Ioannidis, J. P. A. (2005). Why most published research findings are false. *PLoS Medicine*, 2, 696-701.

Jackson, D. D. (1960). A critique of the literature on the genetics of schizophrenia. In D. Jackson (Ed.), *The etiology of schizophrenia* (pp. 37-87). New York: Basic Books.

Joseph, J. (2001). Separated twins and the genetics of personality differences: A critique. *American Journal of Psychology*, 114, 1-30.

Joseph, J. (2004). *The gene illusion: Genetic research in psychiatry and psychology under the microscope*. New York: Algora. (2003 United Kingdom Edition by PCCS Books)

Joseph, J. (2006). *The missing gene: Psychiatry, heredity, and the fruitless search for genes*. New York: Algora.

Joseph, J. (in press). Genetic research in psychiatry and psychology: A critical overview. In K. Hood, C. Tucker Halpern, G. Greenberg, & R. Lerner (Eds.), *Handbook of developmental science, behavior, and genetics*. Malden, MA: Wiley-Blackwell.

Kamin, L. J. (1974). *The science and politics of I.Q.* Potomac, MD: Lawrence Erlbaum Associates.

Kamin, L. J., & Goldberger, A. S. (2002). Twin studies in behavioral research: A skeptical view. *Theoretical Population Biology*, *61*, 83-95.

Kaprio, J., Koskenvuo, M., & Rose, R. J. (1990). Change in cohabitation and intrapair similarity of monozygotic (MZ) cotwins for alcohol use, extraversion, and neuroticism. *Behavior Genetics*, *20*, 265-276.

Kendler, K. S. (1983). Overview: A current perspective on twin studies of schizophrenia. *American Journal of Psychiatry*, *140*, 1413-1425.

Kendler, K. S., & Gardner, C. O. (1998). Twin studies of adult psychiatric and substance dependent disorders: Are they biased by differences in the environmental experiences of monozygotic and dizygotic twins in childhood and adolescence? *Psychological Medicine*, *28*, 625-633.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). A test of the equal-environment assumption in twin studies of psychiatric illness. *Behavior Genetics*, *23*, 21-27.

Kendler, K. S., & Prescott, C. A. (2006). *Genes, environment, and psychopathology*. New York: Guilford.

Keski-Rahkonen, A., Viken, R. J., Kaprio, J., Rissanen, A., & Rose, R. J. (2004). Genetic and environmental factors in breakfast eating patterns. *Behavior Genetics*, *34*, 503-514.

Koenig, L. B., McGue, M., Krueger, R. F., & Bouchard, T. J., Jr. (2005). Genetic and environmental influences on religiousness: Findings for retrospective and current religiousness ratings. *Journal of Personality*, *73*, 471-488.

Kringlen, E. (1967). *Heredity and environment in the functional psychoses: An epidemiological-clinical study*. Oslo: Universitetsforlaget.

LaBuda, M. C., Svikis, D. S., & Pickens, R. V. (1997). Twin closeness and co-twin risk for substance use disorders: Assessing the impact of the equal environment assumption. *Psychiatry Research*, *70*, 155-164.

Laing, R. D. (1981). A critique of Kallmann's and Slater's genetic theory of schizophrenia. In R. Evans, *Dialogue with R. D. Laing* (pp. 97-156). New York: Praeger.

- Lewontin, R. C., Rose, S., & Kamin, L. J. (1984). *Not in our genes*. New York: Pantheon.
- Lilienfeld, S. O., Lynn, S. J., & Lohr, J. M. (2003). Science and pseudoscience in clinical psychology: Initial thoughts, reflections, and considerations. In S. Lilienfeld, S. Lynn, & J. Lohr (Eds.), *Science and pseudoscience in clinical psychology* (pp. 1-14). New York: Guilford.
- Lyons, M. J., Kendler, K. S., Provet, A., & Tsuang, M. T. (1991). The genetics of schizophrenia. In M. Tsuang, K. Kendler, & M. Lyons (Eds.), *Genetic issues in psychosocial epidemiology* (pp. 119-152). New Brunswick, NJ: Rutgers University Press.
- Lytton, H. (1977). Do parents create, or respond to, differences in twins? *Developmental Psychology*, *13*, 456-459.
- Martin, J. D., Blair, G. E., Dannenmaier, W. D., Jones, P. C., & Asako, M. (1981). Relationship of scores on the California Psychological Inventory to age. *Psychological Reports*, *49*, 151-154.
- Martin, N. G., Eaves, L. J., Heath, A. C., Jardine, R., Feingold, L. M., & Eysenck, H. J. (1986). Transmission of social attitudes. *Proceedings of the National Academy of Science*, *83*, 4364-4368.
- McGue, M., & Bouchard, T. J., Jr. (1984). Adjustment of twin data for the effects of age and sex. *Behavior Genetics*, *14*, 325-343.
- Medland, S. E., & Hatemi, P. K. (2009). Political science, biometric theory, and twin studies: A methodological introduction. *Political Analysis*, *17*, 191-214.
- Mendlewicz, J., & Rainer, J. D. (1977). Adoption study supporting genetic transmission in manic-depressive illness. *Nature*, *268*, 327-329.
- Merikangas, K. R., & Risch, N. (2003). Will the genomics revolution revolutionize psychiatry? *American Journal of Psychiatry*, *160*, 625-635.
- Morris-Yates, A., Andrews, G., Howie, P., & Henderson, S. (1990). Twins: A test of the equal environments assumption. *Acta Psychiatrica Scandinavica*, *81*, 322-326.
- Need, A. C., Ge, D. et al. 2009. A genome-wide investigation of SNPs and CNVs in schizophrenia. *PLoS Genetics*, *5* (2), 1-19.
- Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008). *Behavioral genetics* (4th ed.). New York: Worth Publishers.
- Risch, N., Herrell, R., Lehner, T., Liang, K., Eaves, L., Hoh, J., Griem, A., Kovacs, M., Ott, J. & Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression. *Journal of the American Medical Association*, *301*, 2462-2471.

- Rose, R. J. (1982). Separated twins: Data and their limits. *Science*, 215, 959-960.
- Rosenthal, D. (1979). Genetic factors in behavioural disorders. In M. Roth & V. Cowie (Eds.), *Psychiatry, genetics and pathography: A tribute to Eliot Slater* (pp. 22-33). London: Oxford University Press.
- Rowe, D. C. (1994). *The limits of family influence: Genes, experience, and behavior*. New York: The Guilford Press.
- Rutter, M. (2006). *Genes and behavior: Nature-nurture interplay explained*. Malden, MA: Blackwell.
- Scarr, S. (1968). Environmental bias in twin studies. *Eugenics Quarterly*, 15, 34-40.
- Scarr, S., & Carter-Saltzman, L. (1979). Twin method: Defense of a critical assumption. *Behavior Genetics*, 9, 527-542.
- Settle, J. E., Dawes, C. T., & Fowler, J. H. (2009). The heritability of partisan attachment. *Political Research Quarterly*, 62, 601-613.
- Smoller, J. W., Sheidley, B. R., & Tsuang, M. T. (Eds.). (2008). *Psychiatric genetics: Applications in clinical practice*. Washington, DC: American Psychiatric Publishing.
- Sturgis, P., Read, S., Hatemi, P. K., Zhu, G., Trull, T., Wright, M. J., & Martin, N. G. (2010). A genetic basis for social trust? *Political Behavior*, 32, 205-230.
- Suhay, E., Kalmoe, N., & McDermott, C. (2007). Why twin studies are problematic for the study of political ideology: Rethinking "Are Political Orientations Genetically Transmitted?" Presented at the International Society of Political Psychology. Retrieved July 9th, 2010, from [http://sitemaker.umich.edu/suhay/files/critique\\_of\\_twin\\_studies\\_-\\_suhay\\_kalmoe\\_mcdermott\\_101007.pdf](http://sitemaker.umich.edu/suhay/files/critique_of_twin_studies_-_suhay_kalmoe_mcdermott_101007.pdf) )
- Taylor, H. F. (1980). *The IQ game: A methodological inquiry into the heredity-environment controversy*. New Brunswick, NJ: Rutgers University Press.
- Tozzi, F., Aggen, S. H., Neale, B. N., Anderson, C. B., Mazzeo, S. E., Neale, M. C., & Bulik, C. M. (2004). The structure of perfectionism: A twin study. *Behavior Genetics*, 34, 483-494.
- Wacholder, S., Chanock, S., Garcia-Closas, M., El ghormlı, L., & Rothman, N. (2004). Assessing the probability that a positive report is false: An approach for molecular epidemiology studies. *Journal of the National Cancer Institute*, 96, 434-442.
- Wade, T., Bulik, C. M., Neale, M., & Kendler, K.S. (2000). Anorexia nervosa and major depression: Shared genetic and environmental risk factors. *American Journal of Psychiatry*, 157, 469-471.

Wahlsten, D. (1994). The intelligence of heritability. *Canadian Psychology*, 35, 244-259.