



The Lost Study: A 1998 Adoption Study of Personality That Found No Genetic Relationship between Birthparents and Their 240 Adopted-Away Biological Offspring

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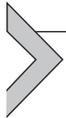
Abstract

In 1998, Robert Plomin and his Colorado Adoption Project (CAP) colleagues published the results of a longitudinal adoption study of personality. They found an average personality test score correlation of only 0.01 between birthparents and their 240 adopted-away 16-year-old biological offspring, suggesting no genetic influences on personality. However, the researchers interpreted their results in the context of previous twin studies, produced an average 14% heritability estimate, and concluded that nonadditive genetic factors underlie personality traits. The author challenges these conclusions and notes that the near-zero correlation stands in contrast to other types of behavioral genetic methods, such as twin studies, that are more vulnerable to environmental confounds and other biases. The author shows that authoritative psychology texts frequently fail to mention this 1998 CAP study. When it is mentioned, the original researchers' conclusions are usually accepted without critical analysis. The author also assesses the results in the context of the 20-year failure to discover the genes that behavioral geneticists believe underlie personality traits. He concludes that this 1998 investigation is a "lost study" in the sense that, although it is one of the most methodologically sound behavioral genetic studies ever performed, its results are largely unknown.

There are genetic studies in psychology and psychiatry whose results run counter to the common claim that hereditary factors play an important role in the development of psychiatric disorders and variation in psychological traits such as IQ and personality, but the implications of these studies are rarely discussed in authoritative textbooks and academic journals. I have identified some of these "lost studies" in previous publications (Joseph, 2004, 2006). One such study contains finding by Kringlen (1967) that reared together monozygotic twin pairs (MZ, identical; 100% genetic similarity) experience much more similar environments and have much higher levels of emotional closeness and "identity confusion" when compared with reared together dizygotic twin pairs (DZ, fraternal; average 50% genetic similarity; see also Joseph, *in press*). A second is a large register-based schizophrenia twin study that found an MZ concordance rate of only 11% (Koskenvuo, Langinvainio, Kaprio, Lönnqvist, & Tienari, 1984). A third is an autism twin study (Ritvo, Freeman, Mason-Brothers, Mo, & Ritvo, 1985) that found a 23.5% DZ concordance rate, which is several times larger than the rate among non-twin sibling pairs (who share the same genetic relationship as DZ twins), and is therefore difficult to explain on genetic grounds.

In cases such as Koskenvuo and colleagues' investigation, the study for the most part does not exist in the "genetics of schizophrenia" literature. In other cases, such as Kringlen's investigation, the study is frequently cited on the basis of other results, but the finding that MZ twin pairs experience much more similar environments and greater levels

of closeness and identity confusion is rarely mentioned. In cases such as the Ritvo and colleagues autism twin study, prominent researchers at times fail to mention it or cite its results (Bailey et al., 1995), whereas other researchers argue that it does not qualify as an “acceptable study of infantile autism” because of perceived selection bias and the inclusion of opposite-sex twin pairs (Steffenburg et al., 1989, pp. 405–406; see also Joseph, 2006, Chapter 7).



1. PLOMIN AND COLLEAGUES' 1998 PERSONALITY ADOPTION STUDY

Here, I will focus on perhaps the most important “lost study” in the literature—a study whose results bear directly on the question of whether genetic factors have an important influence on human behavioral differences. In 1998, Robert Plomin and his behavioral genetic colleagues Robin Corley, Avshalom Caspi, David Fulker, and John DeFries published an adoption study of personality (Plomin, Corley, Caspi, Fulker, & DeFries, 1998) based on their work in the longitudinal Colorado Adoption Project (CAP; Plomin & DeFries, 1985).

This adoption study of personality, which from this point forward I will call the “1998 CAP study,” has become lost in the sense that its most important result—that genes play little if any role in personality development—has all but disappeared in a tidal wave of claims by the authors of textbooks, popular works, scientific articles, and press reports that genetic differences play a major role in human behavioral and psychological development. We will see that when the study is cited, this result is frequently transformed into a finding in support of the importance of genetics, even as molecular genetic research has failed to discover the genes (gene variants) that researchers believe underlie variation in personality and other behavioral traits (see Section 7 below).

The *raison d'être* of an adoption study is its presumed ability to make a clean separation between genetic and environmental influences, because adopted children inherit the genes of their birthparents (biological parents), but are reared in the environment of another (adoptive) family with whom they share no genetic relationship. As Plomin and colleagues wrote in 1997,

The adoption design is powerful because it capitalizes on the intervention of adoption to disentangle genetic and environmental sources of resemblance between parents and offspring by comparing biological parents and their adopted-away offspring, who share genes but not environment, with adoptive parents and their adopted children, who share environment but not genes.

(Plomin, Fulker, Corley, & DeFries, 1997, p. 442)

Although most human genetic researchers believe that twin studies are also able to disentangle genetic and environmental influences, a leading group of psychiatric genetic researchers observed that, because of misgivings about twin research in some quarters, “For some tastes adoption studies provide a cleaner, crisper, separation between the effects of genes and those of environment” (McGuffin, Owen, O’Donovan, Thapar, & Gottesman, 1994, p. 35).

Indeed, critics have argued for decades that both family studies and twin studies are unable to disentangle the potential influences of genes and environment (Joseph, 2004). They have pointed out that the validity of the “equal environment assumption” (EEA) of the “classical twin method,” which holds that reared together MZ and DZ twin pairs experience similar environmental influences, is not supported by the evidence. These critics have argued that the widely recognized finding by Kringlen and others that MZ twin pairs experience more similar environments than those experienced by DZ pairs, a finding recognized by most contemporary twin researchers (Joseph, 2004, 2010a), confounds the results of the twin method. Therefore, the usual twin method finding that MZs correlate higher than DZs for behavioral traits can be *completely* explained by non-genetic factors. In addition, many previously accepted biological and genetic assumptions underlying twin research may not be true (Charney, 2012), which is “necessitating a rethinking of every one of the assumptions of the classical twin study methodology” (Charney & English, 2012, p. 1).

Most behavioral genetic researchers recognize that family studies are unable to disentangle genetic and environment influences, but continue to maintain that the twin method’s EEA “appears reasonable” and that MZ–DZ comparisons provide solid evidence that genes play an important role (Plomin, DeFries, McClearn, & McGuffin, 2008, pp. 75–80). They do this mainly by arguing that MZ twin pair environments are more similar than DZ environments because MZs “create” or “elicit” more similar environments for themselves because they are more similar genetically (for example, see Caspi & Shiner, 2006; Flint, Greenspan, & Kendler, 2010; Plomin et al., 2008; Rutter, 2006; Segal, 2012; for many more examples of leading twin researchers defending the validity of the twin method on the basis of this argument, see Joseph, 2012, pp. 70–72).

However, this “twins create their own environment” argument is a circular one because twin researchers simultaneously assume and conclude that the greater behavioral trait resemblance of MZ versus DZ twin pairs is caused by the former’s greater genetic similarity. Thus, modern twin

researchers' position that genetic factors explain that the greater behavioral resemblance of MZ twin pairs is, circularly, both a premise and a conclusion of the twin method (Joseph, 2010a, 2012, *in press*).

Some pioneers of personality twin research also had doubts about the validity of the EEA. For example, in his 1963 study, Irving Gottesman correctly observed that the twin method "assumes that the within-pair environmental variance is the same for the two types of twins." He concluded, however, "This is not necessarily true for the personality traits as measured by the tests, but one can proceed only on the assumption that such variance is not too different for the two types of twins" (Gottesman, 1963, p. 8).

Returning to adoption studies, there are many problem areas that have been addressed by both critics and researchers alike. These problems include the restricted range of adoptive families, selective placement, late separation, parent-child attachment disturbance, problems with the tests, and the reliability and validity of the trait under study (Bouchard & McGue, 2003; Faraone, Tsuang, & Tsuang, 1999; Horn & Loehlin, 2010; Joseph, 2004, 2006, 2010a; Kamin, 1974; Rutter, 2006; Stoolmiller, 1999). Selective placement in adoption research refers to adoption agencies' practice of placing adoptees into homes correlated with the socioeconomic and perceived genetic status of the birth (biological) parents. As the Texas Adoption Project (TAP) researchers observed, "Selective placement seems to be an integral part of every adoption agency's operating procedures. Most agencies try to find the right 'match' between adoptive child and adopting parents" (Horn, Loehlin, & Willerman, 1979, p. 178). The selective placement of adoptees increases birthparent/adopted-away biological offspring correlations for environmental (non-genetic) reasons. Thus, adoption studies' presumed ability to make a clean separation between genetic and environmental influences is questionable. Nevertheless, it is theoretically possible that a very well designed and executed adoption study, one that controlled for environmental confounds and other potential biases (including biases in the data collection and publication processes), could disentangle genetic and environmental factors and put the nature-nurture question to the test (Joseph & Ratner, 2013).

Behavioral genetic studies also assume that "personality traits are relatively enduring individual differences in behavior that are stable across time and across situations" (Plomin *et al.*, 2008, p. 238) and can be measured and quantified with psychometric tests. These positions are controversial, however, and constitute another questionable yet rarely discussed set of assumptions underlying behavioral genetic research. Moreover, behavioral genetic (and accompanying psychometric) "individual differences" approaches tend

to magnify and emphasize human differences and tend to de-emphasize the common behaviors, abilities, longings, and many other qualities that most human beings share (Fischer et al., 1996; Rose, 1997).

I have discussed these important potential problem areas of behavioral genetics and adoption research elsewhere (Joseph, 2004, 2006, 2010a), and the scope of the present review is therefore limited to (1) highlighting the results of the 1998 CAP study and showing how they contrast with other behavioral genetic studies, (2) a critical examination of how the researchers chose to interpret their results, and (3) a survey of the impact—or lack of impact—that this study has had on the fields of psychology and behavioral genetics.

The 1998 CAP study is noteworthy for four main reasons.

- This was one of the most carefully performed human behavioral genetic studies (Wilson, 1985) and was less vulnerable than previous studies to environmental confounds and other biases. The researchers saw their study as unique because it was the first adoption study to test for genetic influences on self-reported normal personality traits (Plomin et al., 1998, p. 211).
- The researchers found a presumably non-significant average 0.01 personality test score correlation between birthparents and their 240 adopted-away biological offspring, based on eight yearly test score correlations from ages 9 through 16 (Plomin et al., 1998, p. 214).¹ In other words, the results showed no genetic influence on personality.
- The results stand in striking contrast to those from personality studies of twins reared together (the twin method) and twins reared apart (TRA) studies such as the Minnesota Study of Twins Reared Apart (MISTRA; Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Segal, 2012). The MISTRA researchers' claims in support of important genetic influences on personality and IQ have been widely reported, whereas the 1998 CAP study zero correlation is largely unknown. Reared together MZ twin pairs correlate significantly higher than reared together DZ pairs on personality tests (Loehlin, 1992). Based on their acceptance of the EEA's validity, behavioral geneticists believe that this difference is caused by genetic factors.
- The results can be seen in the context of the ongoing lack of replicated molecular genetic gene findings for personality traits, after nearly two decades of searching (see Section 7 below).

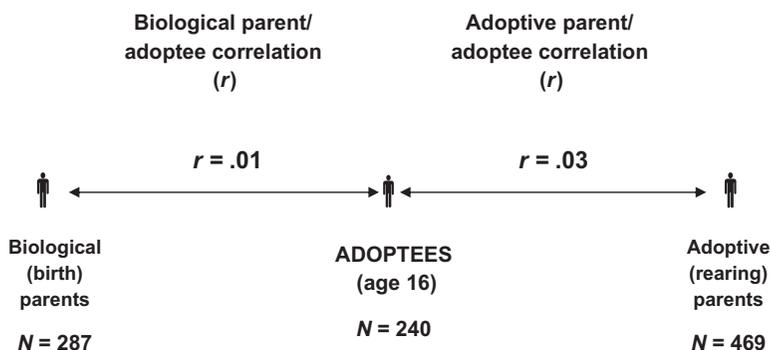
¹Although the researchers studied 245 adoptees, by age 16 only 240 were available for testing.



2. OVERVIEW OF THE STUDY

The CAP was initiated by Plomin and DeFries in the mid-1970s (Plomin & DeFries, 1985; Rhea, Bricker, Wadsworth & Corley, 2013). Because it was a longitudinal study, the researchers were able to track and assess the families since the birth of the children. As seen in Fig. 5.1, the sample consisted of 287 birthparents (238 biological mothers and 49 biological fathers), the 240 children (adoptees) they put up for adoption at birth, and the 469 adoptive (rearing) parents of these adoptees (238 adoptive mothers and 231 adoptive fathers). The researchers also established a control group consisting of 245 non-adopted children and their biological parents. Adoptees were placed with their adoptive parents at an average age of 29 days. More than 90% of the parents in each group were of European ancestry. Birthparents and adoptive parents had completed an EASI self-report personality questionnaire around the time of the children's birth. The EASI was designed to assess "emotionality," "activity," "sociability," and "impulsivity," which according to the investigators are "thought to be the most heritable personality traits" (Plomin et al., 1998, p. 212).

The children were assessed between the ages of 9 and 16 using the Colorado Childhood Temperament Inventory, a self-report measure that is an extension of the EASI questionnaire for children. At age 16, the children were administered the EASI. Because of its unique status as a longitudinal



CAP group sizes and correlations reported in Plomin et al., 1998.

EASI: Self-report personality questionnaire designed to assess "emotionality," "activity," "sociability," and "impulsivity."

Control group consisting of 245 non-adopted children and their biological parents is not shown. The EASI correlation between these control biological children and their parents was reported as 0.07 at age 16.

Figure 5.1 1998 CAP study design and EASI parent-child personality correlations.

adoption study following children from birth, at age 16, the children “completed the same EASI questionnaire that their parents had completed 16 years earlier” (Plomin et al., 1998, p. 212).

The average personality correlation between the birthparents and their 240 adopted-away biological offspring, which the researchers (p. 211) viewed as “the most powerful adoption design for estimating genetic influence,” was 0.01. The average correlation between adoptive parents and these children was 0.03 (Fig. 5.1).

The researchers’ preliminary conclusions were that these results suggest “little effect of nature or nurture” (p. 212), that “genetic factors correlated with parents’ self-reported personality have little effect” (p. 212), and that, “On the face of it,”

these results from CAP suggest that neither nature nor nurture contribute importantly to individual differences in self-reported personality. For example, a direct test of genetic influence comes from the resemblance between biological parents and their adopted-away offspring, who correlated only .01 averaged across the 8 years of assessment of the offspring and across the four EASI traits. (p. 215)

According to the researchers, the birthparent/adopted-away biological offspring correlation “directly indexes genetic influence, unlike the indirect comparisons between nonadoptive and adoptive relatives or between identical and fraternal twins” (p. 211).

The investigators could have stopped at this point and concluded that the study found no genetic influence on personality. However, they decided to interpret the results on the basis of (1) “model fitting” analysis, and (2) the results of the previous twin studies. As the researchers described it, model fitting calculations take potential environmental confounds such as assortative mating (resemblance between spouses) and selective placement “into account while estimating genetic and environmental sources of transmission from parent to child as well as the correlation between genetic and environmental sources (genotype–environment correlation)” (Plomin et al., 1998, p. 214). Using model fitting analysis, the researchers calculated an average personality trait heritability of 14%, which was not significantly different from previous adoption studies, although it was “lower than the average estimates from twin studies” (p. 215). They assumed that there was no selective placement in their sample (see the note in Plomin and colleagues’ Table 4.4, p. 215; see also Rhea et al., 2013).

Plomin and colleagues noted that the authors of previous twin studies of personality had estimated heritability at roughly 40%, and argued that the “most obvious implication of these results is that other family and

adoption studies are needed to triangulate with twin studies on the estimation of genetic influence for personality as assessed by self-reported personality questionnaires” (p. 215). They speculated that the discrepancy between the results of their adoption study and those of previous twin studies was because of adoption studies’ reduced ability to detect nonadditive genetic factors: “The most interesting hypothesis to explain lower heritability estimates from adoption studies as compared to twin studies is nonadditive genetic variance” (p. 217). They elaborated as follows:

Additive genetic effects occur when alleles (alternative forms of a gene) at a locus (place on a chromosome) and across loci add up to affect a trait. Nonadditive genetic effects are interactive effects in which the effects of alleles differ in the presence of other alleles. Nonadditive effects include dominance and epistasis. Dominance is a nonadditive genetic effect in which alleles at a locus interact rather than add up to affect a trait. When several genes affect a trait, the alleles at different loci can add up to affect the trait, or they can interact. This type of interaction between alleles at different loci is called epistasis.

(Plomin et al., 1998, p. 217, emphasis in original)

The researchers argued that studies based on first-degree relatives, such as their 1998 CAP study, “will not detect nonadditive genetic variance” (p. 217) and chose to conclude that their results can be explained by “non-additive genetic influence, which can be detected by twin studies but not by adoption studies” (p. 211).



3. ALTERNATIVE EXPLANATIONS OF THE RESULTS

3.1. The Abstract

According to the sixth edition of the American Psychological Association’s *Publication Manual* (American Psychological Association, 2010), an abstract provides “a brief, comprehensive summary of the contents of the article,” which “allows readers to survey the contents...and, like a title it enables persons interested in the document to retrieve it” (p. 25). In the 1998 CAP study, both the abstract and the title, “Adoption Results for Self-Reported Personality: Evidence for Nonadditive Genetic Effects?” gave the impression that the researchers found evidence in support of important genetic influences on personality. Here is the abstract in full:

Twin studies consistently indicate moderate genetic influence on individual differences in personality as assessed using self-report questionnaires, with heritability estimates typically about 40%. In this first analysis of self-report personality data from the longitudinal Colorado Adoption Project, little evidence is found for additive genetic

influence in parent-offspring and sibling adoption analyses based on a foundation sample of 245 adoptive families and 245 nonadoptive families with adopted and nonadopted children assessed yearly from 9 to 16 years. Although several factors might contribute to the discrepancy between twin and adoption results, we suggest that nonadditive genetic influence, which can be detected by twin studies but not by adoption studies, is a likely culprit. These findings have important implications for attempts to identify specific genes responsible for genetic influence on personality.

(Plomin et al., 1998, p. 211)

In a review of the CAP in its early stages, behavioral genetic investigator Ronald Wilson observed, “Any research project so large and complex involves countless decisions about data analysis” (Wilson, 1985, p. 1370). The 1998 CAP study abstract reflected the researchers’ decision to frame their results in the context of the supposed findings of twin studies and the supposed inability of adoption studies to detect nonadditive genetic influence. The study’s major finding of no significant average personality test score correlation between birthparents and their adopted-away biological offspring is nowhere to be found.

The impression given in the abstract is that this report is just another behavioral genetic study whose authors concluded in favor of genetic influences on psychological trait variation. (A cursory reading of the abstract might even lead readers to think that this was a twin study.) Although Plomin and colleagues decided to conclude that nonadditive genetic influence is “a likely culprit,” they could have decided (and as we will see in Section 4, *should* have decided) to conclude that the misinterpretation of previous twin studies in favor of genetics is a likely culprit.

3.2. Neither Nature nor Nurture?

A valid interpretation of the study is that there are no genetic influences on the self-reported personality traits measured by the EASI. However, although Plomin and colleagues’ preliminary conclusion was that “neither nature nor nurture contribute importantly to individual differences in self-reported personality,” we have seen that their final conclusion in favor of genetics was based on the results of previous twin studies of personality, on model fitting analysis that produced a heritability estimate of 14%, and on the claim that adoption studies are unable to record the effects of nonadditive genetic factors.

The researchers’ conclusion that a low adoptee/adoptive parent personality correlation suggests that nurture (parental) influences play little role in personality development, and that “non-shared” environmental influences play a much larger role (Plomin et al., 2008), illustrates a common

misunderstanding in behavioral genetic research. Indeed, two decades of research into these supposed “non-shared” environmental influences have yielded few results (Plomin, 2011; Turkheimer, 2011a).

As Hoffman (1991) and others have pointed out, developmental psychologists do not view psychological trait resemblance as the appropriate measure of parental influence, because “environmental influences do not produce clones of the parent” (Hoffman, 1991, p. 189). As Hoffman observed, an anxious child could be produced by a non-anxious parent that creates a frightening world for the child, and to “explain dependency in a child, one would not expect to find a dependent parent but rather an over-protective one” (p. 189). Non-depressed parents who regularly beat their children could certainly produce depressed children, yet behavioral genetic researchers would find no depression correlation between parents and children and might mistakenly conclude that parental behavior has little influence on childhood depression. Thus, Plomin and colleagues’ finding of a low personality correlation between adoptees and their adoptive parents does not mean that parental (nurture) influences do not play a major role in personality development.

Behavioral genetic accounts of the underlying causes of personality variation often overlook other major factors shaping personality and development, such as schools and neighborhoods (Theokas & Lerner, 2006), social class, religion, the effects of oppression, and culture (Winter, 1996). According to Gordon Allport, a pioneer of personality trait theory, “Everyone admits that culture is vastly important in shaping personality. ...The impact of culture is so indisputable that some writers regard it as the *all-important* factor” (Allport, 1961, p. 165, emphasis in original). Narrowly focusing on correlations between family members fails to capture the “indisputable” larger role of cultural, religious, social class, and other non-genetic influences on personality and behavioral development.

Let us now examine more closely the three reasons Plomin et al. gave for concluding that their study confirmed the genetic basis of personality traits.

3.3. The Converging Evidence Argument

According to psychiatric geneticists Faraone, Tsuang, and Tsuang, “We cannot rely on either a single study or class of studies to draw conclusions about the effects of genes and environment on mental illness. Instead, from an examination of many studies we seek a pattern of converging evidence that consistently confirms genetic and/or environmental hypotheses about the familial transmission of the disorder” (Faraone et al., 1999, p. 45).

However, Scott Lilienfeld and colleagues, who focused on ways to analyze dubious scientific claims, pointed out that proponents of “converging evidence” arguments in support of such claims “typically maintain that scientific claims can be evaluated only within the context of broader claims and therefore cannot be judged in isolation” (Lilienfeld, Lynn, & Lohr, 2003, p. 9). This enables such proponents to “readily avoid subjecting their claims to the risk of falsification” (p. 9).

Plomin and colleagues chose to “triangulate” the 1998 CAP study findings with the results of family and twin studies, and argued that the results should be interpreted in the context of twin studies’ supposed ability to detect nonadditive genetic influence.² Indeed, the opening paragraph of their publication is about twin studies and the 40% heritability estimate that researchers had derived from their results. In other words, they argued that their results should not be evaluated in isolation and should be viewed in the context of broader “converging evidence” claims about genetics. This claim is difficult to falsify.

3.4. Heritability and Model Fitting

The heritability concept is a controversial one. Proponents of the usefulness of heritability estimates (which range from 0.0 to 1.00 or 0–100%) claim that they are an indicator of the strength of genetic influences on trait variation in a population. However, the use of heritability estimates in the behavioral sciences has been criticized by many reviewers (Feldman & Lewontin, 1975; Joseph, 2004; McGuire & Hirsch, 1977; Rose, 1997; Wahlsten, 1994). Many critics of the concept have argued that heritability estimates are misleading, meaningless, or even harmful.

Leaving aside the question of the validity of heritability estimates in the behavioral sciences—and this is an important issue in and of itself—Plomin et al. (1998) used model fitting analysis to convert the failure to find a personality test score correlation between birthparents and their adopted-away biological offspring into a genetic finding: “At 16 years, the average model fitting estimate of heritability was 14% for the four [EASI] traits using data from the three types of parents and their adopted and nonadopted children” (p. 215). In other words, through statistical transformation, the researchers turned a finding that genes play no role in personality formation into a finding that genes *do* play a role in personality formation. They went on to

²The words “twin” or “twins” appeared no fewer than 70 times in the 8-page 1998 CAP study publication (roughly 3 pages consisted of references and tables).

write that this 14% heritability estimate was not significantly lower than the handful of previous adoption studies (p. 215), implying that the 1998 CAP study results were comparable with these previous studies. At an earlier stage of the investigation, when the children were 7-years-old, the researchers found a virtually identical lack of birthparent/adoptee correlation (0.0) and concluded that, in direct contrast to 1998, “This pattern of correlations provides little or no evidence for hereditary influence at these ages [1–7]—a conclusion supported by model fitting analyses of the CAP parent–offspring and sibling data” (Plomin, Chipuer, & Loehlin, 1990, p. 228).

Nevertheless, in their 1990 “Behavioral Genetics and Personality” chapter in Pervin’s *Handbook of Personality: Theory and Research*, Plomin, Chipuer, and Loehlin cautioned that model fitting “has the disadvantage of being complex and sometimes seems to be a black box from which parameter estimates magically appear” (Plomin, Chipuer, et al., 1990; Plomin, DeFries, & McClearn, 1990, p. 235). Furthermore, they wrote that

We should not stand too much in awe of model fitting or allow it to obfuscate the basic simplicity of most behavioral genetic designs. For example, the twin design estimates genetic influence on the basis of the difference between MZ and DZ correlations. If the MZ correlation does not exceed the DZ correlation for a particular trait, there is no genetic influence (unless assortative mating approaches unity), and model-fitting analyses must come to that conclusion or there is something wrong with the model. (p. 235, emphasis added)

In this passage, Plomin and colleagues argued that if a correlation indicates no genetic influence on a trait, but that subsequent model fitting analyses find such influence, “there is something wrong with the model.” In the 1998 CAP study, however, Plomin and colleagues found no average genetic influence on personality traits and then used “the model” to find such influence. Thus, after collecting the data, Plomin stood his previous position on its head and concluded, in effect, that there is something wrong with the *correlation*.

Moreover, it appears that model fitting analyses, like most human behavioral genetic concepts and methods, are based on several questionable assumptions (McGuire & Hirsch, 1977; Vetta & Courgeau, 2003), including the assumption “that shared genes underlie similarity between relatives” (Segal, 2012, p. 63). The claim that model fitting analyses, which *assume* that genes underlie behavioral trait similarity, can uncover genetic influences on these traits appears to be a circular one, comparable to behavioral genetic defenses of the EEA of the twin method on the basis of the circular argument that twins create their own environments because they are more similar genetically.

In the 1999 revised edition of Plomin's chapter in *Handbook of Personality: Theory and Research* (Plomin & Caspi, 1999), the previous edition's section on model fitting was removed. The 1990 second edition of Plomin and colleagues' textbook *Behavioral Genetics* contained the above quoted "black box/something wrong with the model" caution about model fitting (Plomin, DeFries, et al., 1990, p. 246), as did the 1997 third edition (Plomin, DeFries, McClearn, & Rutter, 1997, p. 310). In the 2001 and 2008 editions, however, this section was removed (for these later *Behavioral Genetics* discussions of model fitting, see Plomin, DeFries, McClearn, & McGuffin, 2001, pp. 351–371; Plomin et al., 2008, pp. 379–402).

In the TAP study (Horn & Loehlin, 2010), which found birthparent/adopted-away biological offspring personality correlations in the 0.07–0.17 range, the researchers went further than the CAP investigators and incorporated twin data into their model fitting analyses. In doing so, the TAP researchers "essentially doubled heritability estimates because of the presence of identical twins in the model fitting" (Horn & Loehlin, 2010, p. 157). Like the CAP researchers, Horn and Loehlin concluded that "nonadditive genetic influences are important for personality" (p. 157), and like Plomin and colleagues, they dismissed the idea that low adoption study correlations called into question the validity of the twin method's MZ–DZ equal environment assumption.

A full understanding of these issues requires a more detailed treatment of the technical and statistical methods involved, which is beyond the scope of the present review. My purpose here is to examine the substantive issue of how researchers and others put together and interpret data, as opposed to the technical aspects of the methods.

3.5. Evidence of Nonadditive Genetic Effects?

The title of the 1998 CAP study publication suggests that the results can be explained by the influence of nonadditive genetic effects.³ But the conclusion that these effects explain the results was mere speculation, as even Horn and Loehlin recognized (2010, p. 3).

Plomin and colleagues' decision to conclude that twin studies can detect nonadditive genetic effects, but that adoption studies cannot, appears to fall into the ad hoc hypothesis category. An ad hoc hypothesis has been defined as "any hypothesis or hypothetical explanation developed to explain

³The study was referenced in the 2001 (4th) edition of *Behavioral Genetics* with the more informative title, "Adoption Results for Self-Reported Personality: Not Much Nature or Nurture?" (Plomin et al., 2001, p. 417).

a particular set of data that does not fit into an existing theoretical framework” (Reber, 1985, p. 12). This allowed the researchers to conclude in favor of genetics on the basis of a 0.01 correlation, just as they would have if the correlation had been 0.50. There are several other areas of behavioral genetic research in which investigators have utilized this genetically-biased “heads I win, tails you lose” approach to data analysis (Joseph, 2004, 2006). In a 2012 defense of the twin method’s EEA, for example, behavioral genetic researchers in the field of political science wrote that even if the critics are “wholly correct” that the causes of MZ–DZ correlation differences are “exclusively environmental,” this finding would “provide reasons for political science to pay more rather than less attention to the biological basis of [political] attitudes and behaviors” (Smith et al., 2012, p. 17; see also Joseph, 2010b, *in press*).

3.6. Undue Weight Given to Researchers’ Conclusions

A general problem in scientific research is that too much weight is given to what researchers decide to conclude about the data they collect (Joseph & Baldwin, 2000). An observer noted the “pervasive...manner in which scientists can deliberately or, more often, unconsciously work in such a way that their conclusions are bound to support a particular position, policy, or action” (Savan, 1988, p. 26). Most researchers have strong beliefs about their areas of research, and few are willing to entertain the possibility that their life’s work has been mistaken. (The same statement can be made about critics of genetic research.) Given these understandable biases, science must do a better job of providing independent evaluation of data. A step in this direction would be to discard the myth that science is a largely objective enterprise (Gould, 1981; Savan, 1988).

In a previous publication (Joseph, 2005), I briefly mentioned the 1998 CAP study’s results and that this study is not often discussed in the literature. In response, a supporter of behavioral genetics (Miele, 2005) reproduced the study’s abstract and italicized Plomin and colleagues’ emphasis on the discrepancy between twin and adoption studies, and their conclusion that adoption studies’ inability to detect nonadditive genetic influences is the likely explanation of their results. He then commented, “The authors of the study are hardly attributing their results to environmental influences and dismissing the effects of genes on personality. The lead author, Robert Plomin, is one of the premier researchers in behavior genetics” (Miele, 2005, p. 28). Very true, but it is not surprising that genetic researchers tend to interpret their results in terms of genetics.

Although Miele implied that the researchers' conclusions trumped their results, and also implied that they deserve to have the last word on the issue, he actually provided additional evidence in support of critics' longtime contention that genetically oriented researchers' beliefs and biases strongly influence their research conclusions. As Gould has shown, there is a long history of such researchers "shifting criteria to work through good data toward desired conclusions" (Gould, 1981, p. 102) and creating conditions in which "data" are not allowed to "overthrow...assumptions" (p. 89).



4. THE 1998 CAP STUDY AND THE TWIN METHOD

In the early stages of the CAP, Plomin and DeFries wrote in *Science*, "We believe that well-designed adoption studies can provide the best information about the relative importance of heredity as a cause of individual differences in human behavior" (Plomin & DeFries, 1976, p. 12). In their 1978 *Annual Review of Psychology* contribution, DeFries and Plomin discussed the issue in more detail:

Although we do not wish to denigrate the usefulness of family study and twin study methods, it is our opinion that adoption studies provide a more convincing demonstration of genetic influence upon human behavioral characters. Adoption studies disentangle genetic and environmental factors common to members of natural families by studying genetically unrelated individuals living together (to test environmental influences common to family members) and genetically related individuals reared apart (to test genetic influences).

(DeFries & Plomin, 1978, p. 481)

In 1985, they wrote, "The adoption study is generally considered to be the most powerful method in human behavioral genetics" (Plomin & DeFries, 1985, p. 17). And 5 years after the publication of the 1998 CAP study, Plomin and colleagues continued to maintain, "Although family and twin designs yield important information, it is generally recognized that the adoption design provides the most convincing evidence upon which to base estimates of genetic and environmental influences" (Petrill, Plomin, DeFries, & Hewitt, 2003a, p. 4).

Clearly, Plomin and colleagues were in the camp of those seeing adoption studies as providing a "cleaner, crisper, separation" between genetic and environmental factors than that provided by twin studies. It follows that they should have concluded that the results of the 1998 CAP study indicated that there is something wrong with twin studies of personality, and that previous interpretations of twin data in favor of genetics should be reevaluated. Instead, we have seen that they used the results of twin

studies to negate their zero correlation finding, even though they had previously written that adoption studies provide “more convincing” evidence than twin studies and, as late as 2003, that “the adoption design provides the most convincing evidence.”

Rather than take a hard second look at twin studies and their underlying assumptions, Plomin et al. wrote that “tests of the equal environments assumption of the twin method generally support the reasonableness of the assumption” (Plomin et al., 1998, p. 216; for a critique of the EEA test literature, see Joseph, 2006). But from another perspective, one could view the 1998 CAP study results as a test of the equal environmental assumption of the twin method. Because the researchers chose to evaluate their results in the context of (less convincing) twin studies, it appears that the validity of the EEA is crucial not only for twin studies but, by extrapolation, it appears to underlie adoption studies as well.



5. THE 1998 CAP STUDY AND THE MINNESOTA STUDY OF TWINS REARED APART (MISTRA)

As we have seen, the results stand in direct contrast to the widely cited MISTRA (Bouchard et al., 1990; Segal, 2012). The MISTRA studies, and their authors’ conclusion that personality traits and IQ are strongly influenced by heredity, have been highlighted in numerous books, magazine articles, and scientific papers since the early 1980s. There have been several other TRA (twins reared apart) studies, with the first appearing in the 1930s (Newman, Freeman, & Holzinger, 1937).

TRA studies are problematic on several grounds, however, including (1) the questionable “separation” of MZ twin pairs, who in many cases were reared together for several years and had quite a bit of contact over much of their lives. According to Susan Farber, in her exhaustive review of the TRA literature, these studies assess “MZ twins *partially* reared apart” (Farber, 1981, p. 273, emphasis added); (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; and (4) the impact that the researchers’ bias in favor of genetic explanations may have had on their results and conclusions (for evidence supporting points (1)–(4), see Farber, 1981; Joseph, 2004, 2010a; Kamin, 1974; Kamin & Goldberger, 2002; Lewontin Rose, & Kamin, 1984).

The biggest problem with TRA studies, however, is that we would expect any two infants of the same sex (whether 100% genetically identical MZ twin pairs or genetically unrelated pairs), who are placed into different

families around the same time, to subsequently share many adult characteristics and traits simply because they are the same sex and grow up in the same era (Joseph, 2004; McGue & Bouchard, 1984; Rose, 1982). Such pairs usually also share national, regional, ethnic, religious, and economic class influences. Together with age and sex effects (and the fact that MZ twins will evoke more similar treatment on the basis of their similar appearance), these numerous common environmental influences plausibly explain any additional reported reared apart MZ behavioral trait resemblance not covered in points (1)–(4) above (Joseph, 2004, 2010a). On the other hand, Plomin, DeFries, McClearn, and other leading genetic researchers continue to claim, quite mistakenly, that reared apart MZ twin pairs “do not share any environmental influences” and that any resemblance between them “must be attributable to the influence of shared genes” (Plomin et al., 2008, p. 383).

According to Robert Sternberg, “the method of separated identical twins [has its own] limitations, such as the confounding variable that identical twins tend to be placed in similar, and hence correlated, environments, so that effects that may appear to be a result of genetic factors may, in fact, not be a result of such factors” (Sternberg, 2007, p. 292). For example, it is likely that a male MZ pair separated at birth and raised in different Pennsylvania Amish families would, for non-genetic reasons, display many similarities in personality, behavior, religious beliefs, clothing, facial hair, and so on. It is also likely that a pair of age-matched but genetically unrelated male infants placed into different Pennsylvania Amish families around the same time would, for the same reasons, display many similarities.

Comparable with the other TRA studies, the MISTRA researchers reported a personality correlation of 0.50 for 44 reared apart MZ pairs (Bouchard et al., 1990), whereas the 1998 CAP study found no personality correlation. In 1994, Bouchard wrote that the “well-replicated finding in behavior genetics, and its implications are straightforward. The similarity we see in personality between biological relatives is almost entirely genetic in origin” (Bouchard, 1994, p. 1701). The results of the 1998 CAP study did not lead Bouchard to revise this position, and like Plomin et al., he speculated about nonadditive genetic effects and was of the opinion that, in the 1998 CAP study, “a variety of methodological and measurement problems (age at measurement, comparability of measures, sampling, etc.) cannot be ruled out” (Bouchard & McGue, 2003, p. 23). Thus, as is often seen in human behavioral genetics, studies finding little or no genetic effects are downplayed or are seen as having “methodological and measurement problems,” whereas massively flawed studies based on implausible assumptions

are cited uncritically and become “landmark studies” (the Loehlin & Nichols, 1976 twin study is a prime example; see Joseph, 2004, 2006, for a critique of this study).



6. THE 1998 CAP STUDY'S LACK OF IMPACT: A REVIEW OF THE LITERATURE

In their discussions of the “genetics of personality” topic, the authors of many influential psychology textbooks and works discussing behavioral genetic research, including texts focusing specifically on personality research, did not cite or discuss the 1998 CAP study. This also holds true for several works popularizing behavioral genetic research and theories (Barondes, 2012; Harris, 2006; Pinker, 2002; Ridley, 2003; Rutter, 2006). This does not mean that the authors of these texts intentionally omitted reference to a study they saw as important, but only that the study is not well known and certainly has never been referred to as a “seminal” or “landmark” study by the competing textbooks and secondary sources that textbook authors at times rely on (Paul, 1985). Because negative findings in the field of psychology are frequently not published or are downplayed as anomalous (Kuhn, 1996), it would not be surprising to find that authors evaluating behavioral genetic research share this tendency. Some examples of influential academic texts, introductory psychology textbooks, and popular works discussing the genetics of personality topic, without referencing the 1998 CAP study, are seen in Table 5.1. (Several edited volumes listed in Table 5.1 contain chapters by leading behavioral genetic researchers.)

I will now briefly survey the publications that did reference the 1998 CAP study to show how the results have been described by the authors of secondary sources, the majority of whom are supportive of behavioral genetic research and theories.

A July 2nd, 2012, PsycINFO database search for publications citing the study listed 48 such publications. If we add the 2001 and 2008 editions of Plomin and colleagues' textbook *Behavioral Genetics*, and a 1999 Plomin and Caspi book chapter (Plomin & Caspi, 1999), which were not listed in PsycINFO, the total rises to 51. Two chapters in a 2003 edited book about the CAP (Petrill, Plomin, DeFries, & Hewitt, 2003b) are also missing from PsycINFO, which increases the total to 53. Subtracting four listed publications by the author of the present article yields a total of 49 publications appearing between 1998 and mid-2012 citing the 1998 CAP study. Of the publications listed in PsycINFO, five were written or co-written by psychologist

Table 5.1 Forty-five texts discussing the genetics of personality topic without referencing the 1998 CAP personality adoption study (Plomin et al., 1998)

Author(s)	Title
Watson, (2000)	<i>Mood and Temperament</i>
Bjorklund & Pellegrini, (2002)	<i>The Origins of Human Nature</i>
Hughes, (2002)	<i>Paving Pathways</i>
McGuffin et al., (2002) (Eds.)	<i>Psychiatric Genetics and Genomics</i>
Nuffield Council on Bioethics (2002)	<i>Genetics and Human Behaviour: The Ethical Context</i>
Pinker, (2002)	<i>The Blank Slate: The Modern Denial of Human Nature</i>
Westen, (2002)	<i>Psychology: Brain, Behavior, and Culture (3rd ed.)</i>
Gazzaniga & Heatherton (2003)	<i>Psychological Science: Mind, Brain, and Behavior</i>
Pennington, (2003)	<i>Essential Personality</i>
Plomin et al., (2003) (Eds.)	<i>Behavioral Genetics in the Postgenomic Era*</i>
Ridley, (2003)	<i>The Agile Gene: How Nature Turns on Nurture</i>
Santrock, (2003)	<i>Psychology (7th ed.)</i>
Kassin, (2004)	<i>Psychology (4th ed.)</i>
Chamorro-Premuzic & Furnham, (2005)	<i>Personality and Intellectual Competence</i>
Derlega et al., (2005) (Eds.)	<i>Personality: Contemporary Theory and Research (3rd ed.)</i>
Jang, (2005)	<i>The Behavioral Genetics of Psychopathology: A Clinical Guide</i>
Kosslyn & Rosenberg, (2005)	<i>Fundamentals of Psychology (2nd ed.)</i>
Pervin et al., (2005)	<i>Personality: Theory and Research. (9th ed.)</i>
Zuckerman, (2005)	<i>Psychobiology of Personality (2nd ed.)</i>
Canli, (2006) (Ed.)	<i>Biology of Personality and Individual Differences</i>
Corr, (2006)	<i>Understanding Biological Psychology</i>
Harris, (2006)	<i>No Two Alike: Human Nature and Human Individuality</i>
Kendler & Prescott, (2006)	<i>Genes, Environment, and Psychopathology</i>
Rutter, (2006)	<i>Genes and Behavior: Nature–Nurture Interplay Explained</i>
Strack, (2006) (Ed.)	<i>Differentiating Normal and Abnormal Psychology (2nd ed.)</i>
Wade & Tavris (2006)	<i>Psychology (8th ed.)</i>
Zimbardo et al., (2006)	<i>Psychology: Core Concepts (5th ed.)</i>
Ashton, (2007)	<i>Individual Differences and Personality</i>
Berk, (2007)	<i>Development Through the Lifespan (4th ed.)</i>
Chamorro-Premuzic, (2007)	<i>Personality and Individual Differences</i>

Table 5.1 Forty-five texts discussing the genetics of personality topic without referencing the 1998 CAP personality adoption study (Plomin et al., 1998)—cont'd

Author(s)	Title
Hockenbury & Hockenbury, (2007)	<i>Discovering Psychology</i> (4th ed.)
Lahey, (2007)	<i>Psychology: An Introduction</i> (9th ed.)
Zuckerman, (2007)	<i>Sensation Seeking and Risky Behavior</i>
Feldman, (2008)	<i>Understanding Psychology</i> (8th ed.)
Rathus, (2008)	<i>Psychology: Concepts and Connections</i> (9th ed.)
Carducci, (2009)	<i>The Psychology of Personality</i> (2nd ed.)
Corr & Matthews (2009) (Eds.)	<i>The Cambridge Handbook of Personality Psychology</i>
Kim, (2009) (Ed.)	<i>Handbook of Behavior Genetics</i>
Flint et al., (2010)	<i>How Genes Influence Behavior</i>
Buss & Hawley (2011) (Eds.)	<i>The Evolution of Personality and Individual Differences</i>
Chamorro-Premuzic et al., (2011) (Eds.)	<i>The Wiley-Blackwell Handbook of Individual Differences</i>
Barondes, (2012)	<i>Making Sense of People: Decoding the Mysteries of Personality</i>
Buss, (2012)	<i>Pathways to Individuality</i>
Deaux & Snyder, (2012) (Eds.)	<i>The Oxford Handbook of Personality and Social Psychology</i>
Segal, (2012)	<i>Born Together—Reared Apart: The Landmark Minnesota Twin Study</i>

*Other than a citation in the Grigorenko chapter (p. 256) in the context of a discussion of epistasis.

David Watson, five others by behavioral geneticist John C. Loehlin, and four others by behavioral geneticist Robert F. Krueger. All three authors are supportive of Plomin and colleagues' views on the genetics of personality.

A review of these 49 publications finds that the authors discussed the study in five main ways. I list these topics in the rough order of how often they were addressed, with topic A appearing with the greatest frequency. In the two editions of *Behavioral Genetics* published after 1998 (Plomin et al., 2001, 2008), the only reference to the study in the genetics of personality context is seen in the topic A quotation below. The five categories are:

- A.** The CAP investigators found evidence that nonadditive genetic factors play an important role in personality formation.
- B.** The study produced lower correlations or heritability estimates than those produced by twin studies.
- C.** The study produced evidence that genes influence personality traits.

- D. The results suggest that genes have little or no influence on personality formation.
- E. The study is cited, but there is no reference to the birthparent/adopted-away biological offspring correlation.

Few of the authors citing the study informed their readers that it found no significant personality test score correlation between birthparents and their adopted-away biological offspring. Of the authors citing the study in support of genetics, none questioned the validity of the original researchers' transformation of zero correlation into a 14% heritability estimate. Moreover, few authors suggested that the results may call into question previous interpretations of twin studies of personality (whether using reared together or "reared apart" twins) as supporting a role for genetic factors.

Here, I present selected quotations from the 49 publications, arranged by topic. In each case, the author(s) directly referenced the 1998 CAP study.

6.1. Topic A: Evidence of Nonadditive Genetic Influences

Lower heritability in adoption than in twin studies could be due to nonadditive genetic variance, which makes identical twins more than twice as similar as first-degree relatives....It could also be due to a special environmental effect that boosts identical twin similarity.

(Plomin et al., 2001, p. 236; repeated in Plomin et al., 2008, p. 240)

Another powerful adoption design involves studying correlations between the personalities of parents and their adopted-away offspring. Such studies produce smaller heritability estimates than do twin studies... Various explanations for such findings are possible, but one prime candidate is that a portion of the genetic effect on personality may be nonadditive.

(Krueger & Markon, 2002, p. 46, emphasis in original)

Adoption studies yield lower—yet still substantial—heritability estimates, but this may be due largely to their inability to model nonadditive genetic variance.

(Watson, Kotov, & Gamez, 2006, p. 10)

Studies using adoption and extended twin-family design have demonstrated that nonadditive genetic effects significantly contribute to the genetic factors found for various personality traits.

(Hur, 2007, p. 373)

The authors speculate that [their results] may be due to personality having a substantial component of its genetic effects non-additive.

(Horn & Loehlin, 2010, p. 3)

6.2. Topic B: Lower Correlations or Heritability Estimates

Adoption studies suggest less genetic influence than twins studies.

(Plomin & Caspi, 1999, p. 254)

We cannot comfortably explain why almost no heritabilities are found in an adoptive study but are substantial in twin studies.

(Block, 2002, p. 182)

Family and adoption studies of personality yield lower estimates of genetic influences than twin studies.

(Caspi & Shiner, 2006, p. 332)

Parent-child resemblances involving personality traits tend to be quite modest so that large samples are required to establish relationships.

(Loehlin, Horn, & Ernst, 2009, p. 3)

6.3. Topic C: Evidence that Genes Influence Personality

At least for self-report questionnaires of personality, it seems clear that genetic factors contribute importantly to individual differences in personality.

(Plomin & Caspi, 1999, p. 256)

Human personality is influenced by inherited components.

(Bookman, Taylor, Adams-Campbell, & Kittles, 2002, p. 786)

Adoptees' traits bear more similarities to their biological parents than to their caregiving adoptive parents.

(Myers, 2005, p. 80)

6.4. Topic D: Little or No Genetic Influence on Personality

In CAP, parent-offspring analyses using self-reports of temperament in early adolescence...show no genetic effects. The average correlation between biological parents and their adopted-away offspring's self-rated temperament was .01.

(Gagne, Saudino, & Cherny, 2003, p. 167)

Interestingly, recent studies have also shown only very slight similarities in personality between adopted children and their biological parents.

(Hewstone, Fincham, & Foster, 2005, p. 304)

The Colorado adoption project tested the 16-year-old adoptees, their adoptive and biological parents, and control parents on the EASI temperament survey...The resulting correlations...provide only weak evidence for genetic contributions.

(Matthews, Deary, & Whiteman, 2009, p. 165)

6.5. Topic E: Study Cited, No Reference Made to the Birthparent/Adopted-Away Biological Offspring Correlation

All these findings are consistent with the results of adoption studies...which showed that children bear little resemblance to either their adoptive parents or their adoptive siblings. Neither parental role modeling nor the parenting practices that would affect all children in a family seem to have much influence on personality traits.

(McCrea et al., 2000, p. 176)

In contrast to the abundant literature reviewing adopted children's behavior problems...studies about adopted children's personality are quite rare.

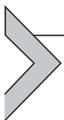
(Juffer, Stams, & van Ijzendoorn, 2004, p. 697)

Typically, however, it is considerably more challenging to gather adoption data, and hence there is much less adoption research on personality than twin research on personality.

(Krueger & Tackett, 2007, p. 75)

Although a few authors reported that the study found little evidence in favor of genetics, the consensus of the authors citing the study was that its results are generally consistent with behavioral genetic positions that genetic factors play an important role in personality development. Only one publication (Gagne et al., 2003) reported the 0.01 EASI correlation. These authors concluded that the study found “no genetic effects.” Typically, the study was mentioned only in passing, and no authors conducted a detailed analysis of the findings.

And as previously mentioned (Table 5.1), the most important aspect of how the study has been reported is the frequent failure to mention it at all in authoritative texts. This stands in contrast to the widespread reporting of the results of twin studies and the largely uncritical acceptance of genetic interpretations of their results.



7. THE 1998 CAP STUDY IN THE CONTEXT OF THE FRUITLESS SEARCH FOR “PERSONALITY GENES”

In the final paragraph of their study, Plomin and colleagues cited reports of mid-1990s molecular genetic associations of genes and personality traits such as “novelty seeking” and “neuroticism.” Although they recognized that the associations needed to be replicated, they believed that these studies constituted a “watershed for molecular genetic research on personality” (Plomin et al., 1998, p. 218). During that period, Plomin and his colleagues were writing in leading scientific journals about gene discoveries for psychological traits and psychiatric disorders as something

that had already occurred, or as something that was in the process of occurring (Joseph, 2011; Plomin, Owen, & McGuffin, 1994; Plomin & Rutter, 1998).

As it turned out, however, the early findings were not replicated, and nearly 20 years of subsequent molecular genetic attempts in psychiatry and psychology to identify genes in the “post-genomic era” have come up empty (Joseph, 2011; Joseph & Ratner, 2013; Wahlsten, 2012). This is true for personality traits (see below), the normal range of cognitive ability (IQ; Deary, 2012), psychiatric conditions such as schizophrenia (Collins et al., 2012), traits studied in the social sciences (Benjamin et al., 2012), and other behavioral traits. The following quotations from leading behavioral genetic researchers and others make it clear that despite the completion of the Human Genome Project and well-funded international efforts and cutting-edge technologies such as genome-wide association studies (GWAS), the search for genes underlying personality has been a failure:

Personality inventories became the basis of several studies designed to identify putative loci for personality. However, something unexpected happened. Despite the stability of the phenotype and consistency of the heritability estimates, years of intense molecular genetic research has been unable to consistently identify the loci underlying any of the major personality traits.

(Jang & Yamagata, 2009, p. 223)

The first candidate gene studies of human personality promised much but, in the fifteen years since their publication, have delivered little in the way of clear evidence for the contribution of specific genetic variants to observed variation in personality traits.

(Munafò & Flint, 2011, p. 395)

To this day, GWAS on well-established heritable traits such as...personality...have yielded disappointing results despite fine-grained approaches with up to 1 million SNPs in the analysis and increasingly large samples.

(Spinath & Johnson, 2011, pp. 294–295)

To the great surprise of almost everyone, the molecular genetic project has foundered on the...shoals of developmental complexity.

(Turkheimer, 2011a, p. 600)

It was widely thought that the Human Genome Project would deliver the vindication of quantitative genetics, especially as it applied to human behavior...Everyone assumed that once the human genome was sequenced the “genes for” the phenomena that had been demonstrated to be heritable would be just around the corner, but it hasn’t happened.

(Turkheimer, 2011b, p. 231)

In human behavior genetics...powerful new methods have failed to reveal even one bona fide, replicable gene effect pertinent to the normal range of variation in intelligence and personality.

(Wahlsten, 2012, p. 475)

Progress has been slow in finding genes associated with behavior.

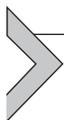
(Plomin, 2013, p. 104)

Since 2008, many leading molecular genetic researchers have adopted the position of “missing heritability” as an explanation for their failure to discover genes (Joseph, 2012; Maher, 2008; Plomin, 2013). The missing heritability interpretation of negative results has been developed in the context of the ongoing failure to uncover most of the genes that genetic researchers believe underlie common medical disorders, and virtually all of the genes that they believe underlie the major psychiatric disorders and psychological trait variation. Proponents of this position argue that genes (heritability) are “missing” because researchers must find better methods to uncover them, as opposed to some critics’ contention that the decades-long failure to discover genes suggests that they do not exist (Joseph, 2011, 2012; Joseph & Ratner, 2013; Latham & Wilson, 2010). From the “missing heritability” standpoint, genetic variants that underlie disorders and psychological trait variation will be found once researchers develop better methods and collect larger samples.

In a 2013 review, Plomin recognized that it “has been much more difficult than expected to identify genes responsible for the heritability of complex traits and common disorders” (Plomin, 2013, p. 104), which he attributed to the “the missing heritability problem” (p. 108). While recognizing the possibility that “heritability has been overestimated,” Plomin continued to maintain that “family, adoption and twin designs generally converge on similar estimates of heritability” (p. 110). He therefore believed that genes will eventually be found, arguing that “the missing heritability is likely to be due to many [still undiscovered] DNA variants of small effect” (p. 114).

However, instead of invoking the ad hoc “missing heritability” hypothesis and “triangulating” the 1998 CAP zero birthparent/biological offspring personality correlation with the results of twin research (as Plomin and colleagues did in 1998), we could plausibly “triangulate” (1) the 1998 CAP study zero correlation finding, (2) the biases and untenable assumptions of twin research, and (3) the negative results of molecular genetic research. We could then conclude that genes for personality trait variation do not appear to exist.

Behavioral genetic researchers are free to interpret the results of twin and adoption studies any way they wish, and to ignore or dismiss the arguments of their critics, but they will never be able to produce “personality genes” if they do not exist.



8. CONCLUSIONS

The 1998 CAP longitudinal adoption study of personality is perhaps the most methodologically sound and least environmentally biased study that human behavioral genetics has ever produced. However, the finding of no significant personality correlation between birthparents and their 240 adopted-away biological offspring is largely unknown. In contrast, the popular press and authoritative secondary sources such as psychology textbooks have widely reported claims of important genetic influences on the basis of reared apart twin studies, often accompanied by photographs of reunited twin pairs and a list of their supposed similarities. And yet, a plausible interpretation of the 1998 CAP study is that, in the context of the current “missing heritability” stage of molecular genetic research, it provides additional evidence that family and twin studies of personality and behavior have recorded nothing more than research bias and the impact of environmental influences. This conclusion is consistent with the position that family, social, cultural, economic, religious, and political environments—and not genetics—are the main factors underlying variation in human behavioral traits.

The results of this lost study should be at least as well known as the celebrated (yet greatly flawed) twin studies, and should have led to a serious reevaluation of the genetics of personality and behavior question in the first decade of the twenty-first century. However, it didn't. Instead, the study has had little impact and does not hold a position of importance in either psychology or behavioral genetics.

Instead of accepting Plomin and colleagues' final conclusion that their study produced a 14% heritability estimate, and that adoption studies may not be able to detect nonadditive genetic influences, the behavioral sciences would do better to accept the researchers' preliminary conclusion that the 0.01 birthparent/adopted-away biological offspring correlation, a correlation that they believed “directly indexes genetic influence,” suggests that “genetic factors correlated with parents' self-reported personality have little effect” (p. 212).

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