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REPLY

Problems in Psychiatric Genetic Research: A Reply to Faraone and Biederman

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A previous paper (Joseph, 2000) discussed the lack of evidence supporting genetic influences on behaviors given the label ''attention-deficit hyperactivity disorder'' (ADHD). Faraone and Biederman (2000) subjected this paper to a critical analysis, writing that Joseph's review ''is based on errors in scientific logic compromised by an incomplete review of relevant data.'' The present paper answers the most important criticisms by Faraone and Biederman. It is reiterated that possible genetic and environmental influences in both ADHD family and twin studies are confounded, and therefore no inferences about genetic factors can be drawn from these studies. In addition, several invalidating flaws of ADHD adoption studies are briefly reviewed. Problems with ADHD segregation analyses and molecular genetic studies are also highlighted. These studies were mentioned by Faraone and Biederman in the context of Joseph's alleged ''incomplete literature review.'' Additional topics include the appropriateness of using psychotropic drugs to treat children diagnosed with ADHD, past social and political misuse of the findings of genetic research, and alternative explanations for ADHD-type behavior. It is concluded that Joseph's previous position—that the available evidence does not support a genetic basis for ADHD—is sustained.

In their essay Faraone and Biederman (2000) raise several points about my article (Joseph, 2000) which must be addressed in order to clarify certain issues for the reader. In response to my conclusion that little evidence exists to support the genetic basis of ADHD, the authors note that this position ''runs counter to the prevailing views in the scientific community . . . [and] is based on errors in scientific logic compromised by an incomplete review of relevant data.'' My purpose here is to answer the second charge. As for the first charge, I can only say that most of the scientific knowledge we

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possess today at one time ran counter to the prevailing views in the scientific community.

Faraone and Biederman discuss five steps used in the process of investigating the genetic basis of ADHD: (1) family studies, (2) twin studies, (3) adoption studies, (4) segregation analysis, and (5) molecular genetic studies. Let us briefly look at each of these.

Family Studies

Faraone and Biederman (2000) agree with my observation that family studies cannot determine whether ADHD is genetically transmitted, since family members share both common genes and a common environment. Contrary to the claims of the authors, I neither wrote nor implied that family studies are ''irrelevant.'' If properly carried out, family studies can provide useful information about *possible* modes of transmission, but little more than that. The finding that children resemble their parents for a particular trait does not necessarily mean that it is ''transmitted'' from parent to child. Parents and children living together share a common environment and are likely to be more similarly affected by environmental factors, even if coming from outside the family. A ''testable psychosocial theory,'' therefore, must show that environmental influences are ''transmitted from parents to children'' *or* that parents and children are exposed to common environmental factors.

Twin Studies

Faraone and Biederman (2000) argue that the basic theoretical assumption of the twin method—the equal environment assumption (EEA)—is valid. However, they fail to address the main point of my article, which is that the twin method is confounded by environmental factors in much the same way as family studies are confounded. In family studies, an affected individual shares a more similar environment with members of his or her family than with members of the general population. In *twin studies,* identical twins share a more common environment and a greater psychological association than fraternal twins. Faraone and Biederman do not dispute these claims but argue that the correlation between common genes and common environment confounds family studies *but does not confound the twin method.* The argument they use in defense of the twin method could just as easily be used to support genetic inferences from family studies. They could argue, for example, that although family members share a more common environment than shared by a group of randomly selected members of the population, critics (of drawing genetic conclusions from family studies) incorrectly ''infer'' from this finding that family members are more likely to be exposed to ''trait-relevant'' environmental factors.

But Faraone and Biederman do not make this argument. Instead, they reserve the trait-relevant EEA *for the twin method* while correctly acknowledging that environmental factors confound genetic inferences from family stud-

ies. Faraone and Biederman thereby commit the logical fallacy of ''special pleading''—that is, they apply a set of standards to one research method but refuse to apply them to another method equally worthy of these standards.

Faraone and Biederman (2000) point to the Thapar, Hervas, and McGuffin (1995) twin study, which found that an index of environmental sharing ''did not predict twin similarity for ADHD scores, i.e., it was not trait relevant'' (Faraone and Biederman, 2000). Similar claims have been made in twin studies looking at other traits. However, like Thapar and colleagues, few methodological details are given and it is often unclear whether unreported comparisons might lead to different conclusions. More importantly, the Thapar et al. study suffers from several flaws apart from its reliance on the equal environment assumption. All of the data, including zygosity determination, were obtained from mailed questionnaires. The study was therefore subject to problems such as respondent bias and the way questionnaire responses were arrived at (i.e., they were completed at home, outside of the researchers' control). The hyperactivity scores reported by Thapar et al. are found in Table II of their paper (Thapar et al., 1995, p. 540). In this table it is reported that the hyperactivity score correlation for same-sex male identical twins (MZ; $N = 88$) was .71, while for same-sex male fraternal twins (DZ; $N = 90$), it was *negative* .22. A negative or zero DZ correlation is impossible to explain on genetic grounds but is easily explained on the basis of poorly collected and biased data.

To summarize, Faraone and Biederman are unable to demonstrate that the traditional or trait-relevant EEA definitions are viable. The twin method therefore remains an environmentally confounded research method which cannot be relied upon to tell us anything about the possible role of genetic factors for a psychological trait or condition (see Joseph, 1998).

Adoption Studies

Faraone and Biederman (2000) acknowledge that the problems with ADHD adoption studies ''limit the strength of any inferences we can draw from these studies,'' although the methodological problems they dismiss as ''minor'' are actually *massive.* These problems include (1) the failure to compare diagnoses among the biological and adoptive relatives *of the same* adoptee; (2) nonblinded diagnoses, which were sometimes made on the basis of relatives' recollections; (3) vague definitions of the dependent variable; (4) the inability to control for environmental confounds; (5) the inability to control for the status of adoptive parents as people who were typically screened for psychiatric disorders; and (6) researcher bias. While Faraone and Biederman agree that these studies contain flaws which limit their ability to tell us much about possible genetic factors, the authors' claim that the genetic theory correctly predicts that ''ADHD should be transmitted through biological, not adoptive family relationships'' rests on these flawed adoption studies.

Faraone and Biederman (2000) write that a ''testable psychosocial theory'' must be able to explain ''the elevated rates of ADHD and associated traits among the biological relatives of adopted away ADHD children,'' implying that elevated rates have been found among these relatives. In fact, no adoption study has investigated the biological relatives of adopted-away ADHD children (see Joseph, 2000). The ''Adoptive Parents'' model, which was used in the most frequently cited studies (Alberts-Corush, Firestone, & Goodman, 1986; Cantwell, 1975; Morrison & Stewart, 1973), compares diagnoses in a group consisting of *adopted-away ADHD children* and their adoptive families, vs a group consisting of families of *other ADHD children* living with their biological parents.

On the basis of the results from ADHD family, twin, and adoption studies, Faraone and Biederman (2000) argue that the ''standard interpretation'' would be that ''the theory that genes influence ADHD has not been disproven.'' Of course, I did not claim to ''disprove'' anything—I merely pointed out that the evidence in favor of genetic factors is extremely weak and that the existence of these factors should therefore not be accepted. Numerous discredited scientific theories ''have not been disproven.'' What we can do is examine the relevant evidence and determine if a theory warrants acceptance.

Segregation Analysis

Faraone and Biederman write that a failure to discuss segregation analysis exemplifies my ''incomplete review of the literature.'' I did not discuss segregation analysis because it is based on the assumption that the genetic basis of a condition has already been established. Faraone and Tsuang (1995) have written that the mode of transmission is investigated ''after demonstrating that a disorder is influenced by genetic factors'' (p. 93), but the purpose of my article was to demonstrate that there is little reason to accept that genetic influences are operating in ADHD.

Segregation analysis utilizes complex mathematical formulas and looks at patterns of familial transmission for the purpose of determining which type of genetic transmission is operating. Segregation analyses often test a nongenetic or ''cultural transmission'' model in addition to genetic models. However, researchers cannot know a priori what a purely environmental transmission might look like or if it resembles a known genetic model. Thus segregation analysis might be useful in determining the type of genetic transmission of a *proven* genetic condition, but it is not a valid method for distinguishing genetic from environmental causation.

Like other methods in psychiatric genetics, segregation analysis is based on several theoretical assumptions. For example, Faraone and Tsuang (1995, pp. 120–124) discuss the ''popular'' segregation analysis model of Morton and MacLean (1974). In this model, according to Faraone and Tsuang, ''a genetic trait is assumed to be due to the influence of a major locus with

mendelian transmission, a polygenic component, and random environmental effects'' (p. 121). But a model *assuming* a genetic basis for a condition cannot provide evidence in favor of genetic causation. Morton and MacLean based their model on several highly improbable assumptions: ''We have . . . assumed random mating, no gene–environment correlation, and no environment common to parents and children'' (1974, p. 501). In the real world random mating for most traits is infrequent, there is plenty of gene–environment interaction, and parents and children usually share common environments.

As a demonstration of how the expression of a culturally transmitted trait can *simulate* a genetic model, McGuffin and Huckle (1990) showed that the results of a complex segregation analysis of the families of British medical students, which tested for a gene *for attending medical school*, ''are compatible with a recessive-gene hypothesis.'' The authors pointed out that a researcher could conclude ''that we have more consistent, and somewhat more persuasive, evidence of a major gene for attending medical school than for any of the neuropsychiatric disorders recently investigated in linkage studies'' (McGuffin & Huckle, 1990, p. 998).

Molecular Genetic Studies

Another example of my alleged ''incomplete literature review'' is the topic of molecular genetic studies of ADHD. Faraone and Biederman (2000) cite several studies finding an association between ADHD and specific genes as well as several studies failing to replicate these findings. Undoubtedly, this constitutes at best an inconclusive body of evidence, in spite of a meta-analysis (Faraone, 1999) claiming the existence of a significant association between a specific gene and ADHD. A disease-causing gene cannot be discovered by taking numbers from a body of inconclusive research and running them through a computer, as Faraone and Biederman seem to suggest. Even in studies cited by Faraone and Biederman finding a significant association, the results are treated with caution. For example, the authors of the Faraone et al. (1999) study linking ADHD to the 7-repeat allele of the DRD4 gene found a statistically significant association, but also noted that ''58% of the subjects without the 7-repeat allele had ADHD. . . . suggest[ing] that the 7-repeat allele cannot be viewed as a necessary cause of ADHD'' (p. 770).

It is apparent from the evidence presented by Faraone and Biederman (2000) that there is no proof that any specific genes cause ADHD. The genetic literature is filled with claims of the discovery of specific genes for psychiatric conditions, which failed to be replicated and were soon forgotten. Examples include the Sherrington et al. (1988) ''schizophrenia gene'' discovery and Egeland and associates' claim to have found a gene for manic-depression (Egeland, Gerhard, Pauls, Sussex, & Kidd, 1987). Given these and other highly publicized yet nonreplicated claims, inconclusive evidence in favor of a specific association will not do; unequivocal proof of the opera-

tion of a specific gene or genes must be demonstrated, as it has been for real diseases such as Huntington's chorea. Indeed, Faraone and Tsuang (1995) have acknowledged that ''with the exception of Alzheimer's disease, attempts to find genes for psychiatric illness have been disappointing'' (p. 124). This is not surprising when we realize that Alzheimer's disease is one of the few DSM-IV conditions that qualifies as a true (i.e., proven) brain disease. As noted by a prominent genetic researcher, ''psychiatric genetics appears to be at a crossroads or crisis'' (DeLisi, 2000, p. 190).

I end this discussion with a prediction similar to the one I made in a previous publication that looked at the genetics of schizophrenia (Joseph, 1999): *A gene (or genes) for ADHD will not be discovered because it does not exist.* Psychiatric geneticists and molecular geneticists will one day give up on this thankless and futile endeavor, and researchers will focus on the environmental factors influencing ADHD-type behavior—some of which are summarized by Faraone and Biederman (2000).

Theories and Predictions

Faraone and Biederman (2000) argue further that the genetic theory of ADHD ''has consistently made predictions which turn out to be correct.'' But this claim is contingent upon what predictions are made and which questions are asked. An alternative analysis would review facts about ADHD not typically discussed in the context of genetics, but which make genetic explanations unlikely: (1) ADHD-type behavior is often exhibited by an individual in some situations but not in others (APA, 1994). According to Barkley, ''all the primary symptoms of ADHD show significant fluctuations across various settings and caregivers'' (1998b, p. 73). In other words, children with alleged genetic defects and shrunken brain areas (Barkley, 1998a) are often fine when playing baseball and Nintendo, but display ''symptoms'' in boring and unstimulating environments. (2) ADHD symptoms typically do not persist into adulthood, or in the words of the DSM-IV, ''In most individuals, symptoms attenuate during late adolescence and adulthood . . .'' (APA, 1994, p. 82). (3) ADHD is diagnosed from 3 to 10 times more often in boys than in girls (Barkley, 1998b). (4) ADHD has been widely recognized as a problem for only about 30 years (Arnold & Jensen, 1995). (5) Individual types of ADHD-like behavior are found in a large percentage of ''normal'' children (Barkley, 1998b). (6) Over four million children in the United States consume stimulants, while in a country like France (population 60 million), less than 6000 children receive these drugs (David Cohen, personal communication, 6/10/2000).

While individually none of these points rule out genetic factors, together they argue against the idea that genes are involved. This position is strengthened by the evidence in my article (Joseph, 2000) showing that family, twin, and adoption studies do not support the genetic position. The predictions mentioned by Faraone and Biederman are based on a body of confounded, methodologically unsound, and inconclusive research.

Other Issues Discussed by Faraone and Biederman

Faraone and Biederman (2000) point out that psychosocial theories of causation can stigmatize families, which is of course a valid observation. However, there is a difference between the stigma of being a ''bad parent'' and the stigma of being seen as the carrier of ''bad genes'' (or ''hereditary taint'' as it used to be called in psychiatric genetics; see Kallmann, 1938 as an example). This brings us to Faraone and Biederman's objection to my quite modest statement that ''history has shown that the results of genetic studies have often been used to stigmatize individuals and groups'' (Joseph, 2000). Faraone and Biederman imply that this has not occurred, with the exception of the Nazi's ''use'' of psychiatric genetic data to justify ''sterilization and murder.'' Their position overlooks the entire history of the eugenics movement in the United States and Europe and the *collusion* between German psychiatric geneticists and Hitler's regime. The case of Ernst Rüdin illustrates this last point.

Rüdin was the founder of psychiatric genetics and has been referred to as one of its ''great pioneers'' (Faraone & Tsuang, 1995, p. 124). Rüdin was also a pioneer of the German eugenics movement and in 1905 was one of the founding members of the German Society for Racial Hygiene (Proctor, 1988). After the Nazi seizure of power in 1933, Rüdin helped draft ''The Law for the Prevention of Genetically Diseased Offspring,'' which mandated the eugenic sterilization of people with, among other conditions, schizophrenia and ''manic-depressive insanity.'' It is estimated that between 300,000 and 400,000 people were sterilized under this law (Proctor, 1988, p. 108). In 1935, Rüdin proposed that the sterilization law be extended to include ''valueless individuals . . . all who were socially inferior psychopaths on account of moral confusion or severe ethical defects'' (quoted in Müller-Hill, 1988/1998, p. 33). According to his biographer, Rüdin ''played a major role in the propagation of racial hygiene doctrines in the 'Third Reich''' (Weber, 1996, p. 328). Hitler awarded Rüdin the Goethe medal for art and science in 1939, which was accompanied by a telegram from Interior minister Wilhelm Frick which read, ''To the indefatigable champion of racial hygiene and meritorious pioneer of the racial-hygienic measures of the Third Reich I send . . . my heartiest congratulations'' (quoted in Weinreich, 1946, pp. 32–33).

In the late 1930s, the regime took ''racial hygiene'' and eugenic sterilization to its logical conclusion and instituted an extermination campaign against mental patients and ''defectives,'' resulting in the murder of approximately 70,000 people (Proctor, 1988, p. 177). While Rüdin apparently was not personally involved in the Nazi ''euthanasia'' campaign, in 1942 he stressed[1] ''the value of eliminating young children of clearly inferior qual-

[1] The word ''stressed'' is Weber's.

ity'' (quoted in Weber, 1996, p. 329; one might speculate about the type of ''treatment'' Rüdin would have had in mind for ADHD children). Rüdin wrote the following in 1942, when the Holocaust was already underway:

> The results of our science had earlier attracted much attention (both support and opposition) in national and international circles. Nevertheless, it will always remain the undying, historic achievement of Adolf Hitler and his followers that they dared to take the first trail-blazing and decisive steps toward such brilliant race-hygienic achievement in and for the German people. In so doing, they went beyond the boundaries of purely scientific knowledge. He and his followers were concerned with putting into practice the theories and advances of Nordic race-conceptions . . . the fight against parasitic alien races such as the Jews and Gypsies . . . and preventing the breeding of those with hereditary diseases and those of inferior stock. (Quoted in Müller-Hill, 1988/1998, p. 67)

Rüdin's history is discussed here as an important example; there were many other lesser figures in German psychiatry who held similar views and performed similar functions (see Burleigh, 1994; Lifton, 1986; Müller-Hill, 1988/1998; Proctor, 1988).

Faraone and Biederman also failed to mention the existence of compulsory eugenic sterilization laws in over 24 American states (Lindman & McIntyre, 1961; Reilly, 1991), in the Scandinavian countries (Broberg & Roll-Hansen, 1996), and elsewhere. These laws were passed on the basis of the results of *research*, and (including Germany) hundreds of thousands of people were sterilized or died undergoing the sterilization procedures. In the United States, more than 60,000 people were involuntarily sterilized for eugenic purposes (Reilly, 1991, p. 94). Naturally, this does not mean that contemporary genetic researchers are responsible for the crimes of the past or that genetic research is wrong—in fact, it is vitally important in some areas—but the history of its misuse and the past role of some of the ''scientists'' undertaking this research must be understood. Today, according to Faraone and Biederman (2000), ''we know of no data showing that genetic studies lead to stigma.'' This statement fails to take into account social and political realities. It is not a question of ''data,'' but rather an understanding that people who are seen as carrying a genetic predisposition for socially undesirable traits are stigmatized to varying degrees in different cultures and eras. As Manfred Bleuler wrote about mid-20th century European families of people diagnosed with schizophrenia:

> If one knows schizophrenics and their families well, it is sometimes a matter for despair to see how much they suffer under the terrible concept of ''familial tainting.'' Like a sinister shadow it darkens the lives of many people and of entire families. The stifling, uncertain fear of coming from an ''inferior breed,'' of carrying within one's self the seeds of something pathological, morbid, and evil (I am speaking in the jargon the afflicted apply to themselves), like a curse that you must pass on to someone else, causes oppressive feeling of inferiority. (Bleuler, 1978, p. 473)

Too often, ''ADHD children'' are seen as ''data'' and ''cases'' when in

reality these children (like most ''normal'' children) have intense feelings and needs (which may vary from child to child but are obscured by the simple broad label of ADHD); are expected to grow up and thrive in an increasingly hurried, complicated, and alienating society; must endure underfunded schools and social services; and sometimes experience abusive family conditions (McCubbin & Cohen, 1999).

Faraone and Biederman (2000) point to my ''misleading conclusion'' that genetic studies have been used to support the use of psychotropic drugs. In a sense they are correct, since the opposite is usually the case: The ''effectiveness'' of drugs to modify behavior is often used (albeit incorrectly) as evidence of an underlying biological malfunction or genetic predisposition. In another sense, I was merely paraphrasing the advice that Faraone has already given to clinicians:

> Another influence of genetic findings is in the area of education and medication compliance. Many parents are reluctant for their children to take psychotropic medication and others find it difficult to maintain the prescribed regimen. *These problems are mitigated by discussing the genetic etiology of ADHD* [italics added]. . . . For many psychiatric disorders, genetic data provide the quickest and most convincing means of showing patients how biology plays a role in their condition. (Faraone, 1996, p. 598)

On the subject of ''therapeutic nihilism'' discussed by Faraone and Biederman (2000), I did not write and I do not believe that people should be ''denied'' psychotropic drugs, in spite of their dangerous adverse effects and frequent lack of effectiveness (see Breggin, 1998, 1997; Cohen, 1997). In my opinion an adult has the right to take most psychotropic medications, provided that he or she receives all of the relevant information regarding the drug's efficacy, side effects, and potential for long-term harm to the brain and body. Additionally, alternative treatments and theories about the cause of the person's problems should be discussed. However, I would not necessarily extend this right to include giving Ritalin or similar drugs to children (a decision typically made by parents). If ADHD-type behavior is the result of social or psychological factors, then what society is essentially doing is pumping potentially harmful chemicals (Breggin, 1998) into the bodies of growing children as a way of making them conform to current societal or educational norms. In the case of ADHD, etiology is relevant because while it may be necessary to give children potentially harmful medication in order to treat actual physical diseases, it is ethically questionable to ''medicate'' children in order to control undesirable behavior caused by psychological or social problems or to boost their academic performance (McCubbin & Cohen, 1999). I am certain that Faraone and Biederman would agree that it would be wrong to give a ''tooth brushing'' drug to an 8-year-old boy who refuses to brush his teeth before going to bed, yet ''medicating'' misbehaving, hyperactive, or inattentive children is not a qualitatively greater step than this.

As Breggin (1998) has stressed, ADHD diagnostic criteria are not based on or concerned with the *feelings* of a child. Rather, as seen in the DSM-IV (APA, 1994, pp. 83–85), children frequently receive the diagnosis on the basis of behavior that is disturbing to others (e.g., parents and teachers). Faraone and Biederman's hypothetical examples of people asking for medication who should not ''be denied'' describe people who see a clinician in order to alleviate personal suffering. In the case of ADHD children, it is more often *people in their environment* who are suffering, meaning that drugs such as Ritalin are often given to children in order to alleviate the suffering *of others*.

It is true that my article (Joseph, 2000) touched only briefly on alternative explanations for ADHD. One reason is that the condition is not a valid disease or illness. DeGrandpre (2000, p. 13) has written that the ADHD label ''is at best a poor description of a cluster of developmental and psychological problems stemming from a myriad of causal pathways; at worst, it is a sham and pseudo-scientific explanation,'' taking the place of a real explanation ''that links symptoms to independent causal factors, pointing the way to real solutions.'' To the extent that children and adults do exhibit these behaviors, we should look more closely at societal conditions for the answer. Arnold and Jensen (1995), who hold many mainstream views on ADHD, noted the ''probable interaction between the complexity of environmental demands and manifestation of the symptoms of ADHD.'' They went on to acknowledge that ''it is . . . possible that today's complex environments are overstimulating'' and that,

> Children who assimilate a steady diet of video games, television, multiple afterschool activities, harried parents, and interchangeable caretakers may have their attentional systems down-regulated as a means of reducing the noise. They may become used to many novel, complex stimuli, and their attentional systems may not respond to the lower-level stimuli involved in academic work. (Arnold & Jensen, 1995, p. 2300)

Even as an oversimplified thesis, I find this a plausible explanation for the apparent increase of ADHD-type behavior in late 20th- and early 21st-century North America.

Conclusion

As I have argued here and elsewhere (Joseph, 2000), the body of evidence typically cited in support of the genetic basis of ADHD consists of studies that (1) contain invalidating methodological errors, (2) provide inconclusive results, and (3) are confounded by environmental factors. Faraone and Biederman (2000) agree with me that ADHD family studies do not tell us anything important about genetic causation, and they acknowledge that only limited inferences can be drawn from the ADHD adoption studies. Additionally, I have discussed some of the problems with segregation analysis and molecular genetic studies. Thus, the validity of the twin method and the

equal environment assumption remains central to the genetic hypothesis, yet Faraone and Biederman offer little reason to doubt that the twin method is hopelessly confounded by the greater environmental similarity of identical vs fraternal twins. Therefore, my original conclusion is sustained: The evidence does not support a genetic basis for ADHD, and psychosocial causes of ADHD-type behavior should be the focus of future research. If the most that Faraone and Biederman (2000) can conclude is that the genetic basis of ADHD is the most parsimonious explanation and ''has not been disproven,'' then we should let this stand for the record as evidence that even leading ADHD genetic researchers cannot demonstrate that there is a convincing body of evidence pointing toward genetic factors.

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