The field of genetic epidemiology includes three sets of studies: family, twin, and adoption studies. This commentary argues that findings from these studies are sometimes used to blame parents for the development of developmental disabilities, emotional problems, or problem behaviors in their children. For this reason, this paper concludes that genetics as explanations of psychopathology among children is sometimes the enemy of parents and those parents should avoid mental health practitioners who emphasize genetics in the explanation of why children develop such psychopathology.

Keywords: Parents; Genetics; Family studies; Twin studies; Adoption studies
the same similarities and same mental disorder between the same adoptive child and his or her adoptive parents. The general findings in adoptive studies is that the similarities of a given mental disorder (e.g., symptoms of depression, anxiety, ADHD) are significantly correlated (associated) between the adopted child and his or her biological parents, relative to a lesser correlation in such similarities between the adopted child and his or her adopted parents [7,3,12]. An example is the study by Tharp [13] with emphasis on the genetics of attention-deficit hyperactivity disorder (ADHD). Tharp found that “the rate of ADHD...is much higher in the biological relatives of affected individuals than among adoptive relatives” (pp. 446-447).

**Genetics as the enemy of parents**

The overall conclusion among family, twin, and adoptive studies is that the “gene” is a critical factor in the explanation for the presence of a given mental disorder shared by some members of a given family. Despite the rejection of the null hypothesis in all studies with emphasis on variants of genetic epidemiology (e.g., twin studies), the statistically significant results are correlational rather than causal. That is, the only valid assertion the investigator can make is that event A (e.g., a twin with autism) is correlated with event B (a sibling may also develop symptoms of autism), but that investigator is not in the position to explicitly state that A is the cause for B. In this context, the investigator asserts the validity of a “correlational” explanation with the help of statistical tricks. For example, the investigator learned during his or he statistical training that it is easier to reject the null hypothesis with criterion levels of statistical significance = 0.05, in comparison with 0.01 where it is more difficult to reject that hypothesis. Similarly, it is more difficult to reject the null hypothesis with two-tailed tests, in comparison with one-tailed test [14]. But despite these tricks, in the case of “correlational” explanations the researcher will never be able to assert without doubt that A is actually what caused B.

Correlational explanations derived from all variants of genetic epidemiology reinforce the belief of some mental health practitioners in that there must be a “gene” responsible for the commonality of symptoms of a given mental disorders among family members [4]. I suggest, however, that the emphasis on findings based on genetic explanations that are essentially correlational in nature sometimes may function as the enemy of parents. The reason for this suggestion is that in such explanations parents are often blamed for the development of mental disorders in their children just because they happen to be their parents. For example, [15] reviewed the genetic literature with emphasis on families and ADHD and found that “a history of maternal depression uniquely predicts negative long-term outcome for children with ADHD, including the development of later conduct problems, depression, and suicidal behavior” (p. 198). These authors also found that “there is substantial evidence of links between parental antisocial behaviors and both child ADHD and disruptive problems [e.g., conduct disorder]” (p. 199), and that “genetically informed studies have revealed the strongest evidence for disorder-specific risk transmission for psychopathology from parents to child” (p. 199). In addition, in that review of the literature Johnston and Chronis-Tuscano also found that some studies “found support for a genetic link between maternal alcohol use disorder and offspring risk for ADHD” (p.200). These authors [15], however, did not reveal a crucial point in their review of the literature, namely, that all these findings are correlational and that suggesting that children develop symptoms of ADHD and other “psychopathological” problems because these children inherited such problems from their parents is clearly a case of using genetic arguments against parents [16-17].

The negative consequence of findings derived from all variants of genetic epidemiology is that some mental health practitioners aware of such findings would conduct the initial clinical interview (intake) with the belief that the child’s symptoms for a given mental disorder (e.g., ADHD, depression, anxiety, alcohol-related problems, etc.) are the result of a “bad” gene the child received from their parents who also experience similar disorders in the past. This situation should alert parents that genetics is its enemy in certain circumstances. Therefore, if parents “feel” that the mental health practitioner is using such correlational findings to blame them for the mental disorder, emotional disability, or problematic behavior their child is currently experiencing, my suggestion is that parents should quickly search for a different mental health practitioner who is not mentally or cognitively “infected” by those correlational findings. This recommendation is particularly important to remember in those clinical cases where the family history does not show any evidence of parents’ psychopathological problems, but the mental health practitioner evaluating the case still blames parents for their child’s psychopathology.

**References**

1. Merikangas KR, Swendsen JD (1997) Genetic epidemiology of psychiatric disorders Epidemiologic Review 19[1]: 144-155.
2. DiLalla LF (2004) Behavior genetics principles: Perspectives in development, personality, and psychopathology. American Psychological Association. Washington, DC, USA.
3. Duncan LE, Pollastril AR, Smoller JW (2014) Mind the gap: Why may geneticists and psychological scientists have discrepant view about gene-environment interactions (GxE) research. American Psychologists 69[3]: 249-268.
4. Barkley RA (2015) Etiologies of ADHD (Ed.) Attention-Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment. Guilford Press, New York, USA pp. 356-390.
5. Chaste P, Devlin B (2015) Architecture of the genetic risk for autism. In M Leboyer & P. Chaste (Eds.) Autism spectrum disorders: Phenotypes, mechanisms and treatment Basel, Switzerland 180: pp. 80-96.
6. Fanone SV, Tsuang MT, Tsuang DW (1999) Genetics Genetics of Mental Disorders. J Psychiatry Neurosci 27[4]: 291–292.
7. Fisher SE (2003) Isolation of the genetic factors underlying speech and language disorder
8. Barkley R A (2013) Taking charge of ADHD: The complete, authoritative guide for parents. Guilford Press, New York, USA.
9. Willcutt EG, Defries JC, Pennington BF, Smith SD, Cardon LR, et al. (2003) Genetic etiology of comorbid reading difficulties and ADHD. In R Plomin, JC Defries, IW Craig, P McGuffin, E G DeFries, JC Pennington, BF Smith, SD, Cardon LR, (Eds.), Behavioral genetics in the post genomic era, American Psychological Association, Washington, DC, USA, pp. 227-246.

10. Kalidindi S, McGuffin, P (2003) The genetics of affective disorders: Present and future. In R Plomin JC Defries, IW Craig, P McGuffin, Robert plomin Behavioral genetics in the post genomic era (Eds.) American Psychological Association, Washington, DC, USA.

11. Smith BH, Barkley RA, Shapiro CJ (2006) Attention-deficit/ hyperactivity disorder. In EJ Mash, RA Barkley (Eds.), Treatment of childhood disorders (3rd Ed.), NY: Guilford Press pp. 65-136.

12. Barkley R A (1997) ADHD and the nature of self control. Guilford Press, New York, USA.

13. Thapar A (2003) Attention deficit hyperactivity disorder: New genetic findings, new directions. In R. Plomin JC, Defries IW Craig, P McGuffin (Eds.), Behavioral genetics in the postgenomic era American Psychological Association, Washington, DC, USA.

14. Kline Rex B (2013) Beyond significance testing: Statistics reforms in the behavioral sciences. American Psychological Association, Washington, DC, USA, pp.349.

15. Johnston C, Chronis Tuscano A (2015) Families and ADHD. In RA Barkley (Ed.) Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment (4th ed.), Guilford Press, NY, USA, pp. 191-209.

16. Pericak Vance MA (2003) The genetics of autistic disorder. In R Plomin, JC Defries, IW Craig, P McGuffin (Eds.), Behavioral genetics in the post genomic era. American Psychological Association, Washington DC, USA.

17. Plomin R, Defries JC, Craff IW, McGuffin P (2003) Behavioral genetics in the post genomic era. Washington, DC: American Psychological Association.

This opinion is an abbreviated version of a more extensive discussion published by the author in his book entitled Informed Parents, Healthy Kids: Information you Need to Know to Find the Right Mental Health Practitioner, published by Nova Science Publishers, 2018, Hauppauge, New York