Behavior Genetics, Vol. 34, No. 1, January 2004 (© 2004)

Are Personality Disorders Psychological Manifestations of Executive Function Deficits? Bivariate Heritability Evidence from a Twin Study

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Received 25 June 2002—Final 4 Nov. 2002

This study tested whether personality disorders may be the psychological manifestations of executive function deficits by examining their bivariate heritability in a community sample of 314 twins (ages 5–17 years; *M* age = 9.7; 96 monozygotic pairs and 61 dizygotic pairs). The parents of the twins completed the Coolidge Personality and Neuropsychological Inventory (Coolidge, 1998; Coolidge *et al.,* 2002). Heritability was estimated by structural equation modeling. Executive function deficits and personality disorders were significantly heritable (executive function deficits, .77; 11 out of 12 personality disorders, median = .69). The proportion of the observed correlation attributable to heritable factors or bivariate heritability between executive function deficits and the personality disorder scales ranged from .27 for schizoid to .64 for histrionic. These findings may provide some insight as to why individuals diagnosed with specific personality disorders frequently exhibit chronic difficulties with everyday decisions, selective attention and inhibition, judgments, choices, planning, and flexibility.

KEY WORDS: Personality disorders; executive function deficits; bivariate heritability.

INTRODUCTION

The Russian neuropsychologist Luria (1966) noted that patients with frontal lobe damage frequently had their speech, motor abilities, and sensations intact, yet their complex psychological activities were tremendously impaired. He observed that these patients were often unable to carry out complex, purposive, and goal-directed actions. Furthermore, he found that they could not accurately evaluate the success or failure of their behaviors, especially in terms of using the information to change their future behavior. Luria found these patients unconcerned with their failures, hesitant, indecisive, and indifferent to the loss of their critical awareness of their own behaviors. These general symptoms have come to be known as executive function deficits of the frontal lobes.

Lezak (1982) wrote that the executive functions of the frontal lobes were ". . . the heart of all socially useful, personally enhancing, constructive, and creative abilities . . ." (p. 281). Furthermore, she thought that the impairment or loss of executive functions severely compromised a person's ability to be independent, constructively self-serving, and socially productive, regardless of how well preserved the other cognitive abilities were. She noted that some of the more psychological or behavioral changes that may result might be poor self-control, lack of self-direction, emotional lability, flattened affect, irritability, impulsivity, carelessness, rigidity, and difficulty in shifting attention.

Interestingly, executive functions have been defined much more narrowly and often without the personal or social ramifications in much of the other neuropsychological literature. Welsh and Pennington (1988) defined executive functions as the ability to maintain an appropriate problem solving set for the attainment of a future goal. They also added interference

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control, inhibition, and integration across space and time as other aspects of executive function. They did note, however, that many complex behaviors require executive functions, particularly human social behavior. Later, Pennington and Ozonoff (1996) defined executive functions as a unique domain of abilities that involves organization in space and time, selective inhibition, response preparation, goal-attainment, planning, and flexibility.

Barkley (2001) has recently approached the issue of defining executive functions from an evolutionary perspective and has taken to task traditional definitions and laboratory measures of executive functions. He viewed executive functions as a biological adaptation to interpersonal competition in social interactions. Barkley thought executive functions served as a social self-defense against resource theft (including spouse) and against interpersonal manipulation. He also saw them as advantageous in social exchanges (like reciprocal altruism or selfish cooperation) and useful in imitating and learning from others without the dangers inherent in trial and error. Barkley noted that traditional measures of executive functions such as the Wisconsin Card Sorting Task, the Tower of Hanoi, and digit span tasks often correlate poorly to the patient's decision-making, planning, and social judgments in natural settings. He wrote, ". . . extant EF measures tell precious little about what is ultimately lost in adaptive functionality in those patients suffering injury to their executive system" (p. 4).

Sarazin *et al.* (1998) also noted a dissociation in some frontal lobe damaged patients between their normal performance on laboratory measures of planning and execution and measures that show highly impaired and disorganized behavior in their general lives. They further noted that the kind of planning required by daily life may require affect-laden decisions that are not being measured by traditional laboratory tests of executive functions.

There are a number of models for the neurocircuitry and functionality of executive functions. Alexander *et al.* (1986) proposed five parallel but segregated frontal-subcortical circuits. Recently, Middleton and Strick (2001) presented evidence for two additional frontal-subcortical circuits. Chow and Cummings (1999) focused on three of these frontal-subcortical circuits that are associated with neurobehavioral repercussions from damage or dysfunction: the dorsolateral prefrontal cortex, the orbitofrontal prefrontal cortex, and the anterior cingulate cortex. The dorsolateral circuit is generally associated with the classic executive functions, that is, complex problem solving, decision-making, verbal fluency, and working memory. Brodmann's areas for the dorsolateral circuit are usually identified as 8, 9, and 46. The orbitofrontal prefrontal region is more closely connected to the limbic system and is associated with the processing of emotions and the regulation of social behavior and social interactions. Brodmann's areas for the orbitofrontal regions are generally identified as 10, 11, 12, 13, 14, 45, and 47. It is important to note that both systems are closely interconnected, and the prefrontal cortex in general has extensive projections to almost all regions of the temporal and parietal lobes, some projections to the occipital lobe, and subcortical structures such as the basal ganglia, the cerebellum, and many brainstem nuclei. There is some controversy as to which Brodmann's areas are dorsolateral, orbitofrontal, or ventromedial. The gist of these interrelationships appears to be that the prefrontal cortex coordinates the processing of broad regions of the central nervous system. A third region of the prefrontal cortex is the anterior cingulate gyrus (Brodmann's areas 24, 25, and 32), and it is thought to mediate motivational systems and action selection (Pennington, 2002). The latter region is sometimes included within the orbitofrontal system (Sarazin *et al.,* 1998), and sometimes it is considered a separate system (Chow and Cummings, 1999).

Damasio *et al.* (1994) developed a "somatic marker" hypothesis to explain the interrelationship of the orbitofrontal cortex and anterior cingulate gyrus and their contributions to decision-making. They believe that complex reasoning and emotion are intertwined such that quick yet rational decisions demand an emotional valence attached to the various elements of the decision process. The lack of such a system, which would provide a fast and "gut-level" sense of what to do next and what to avoid, would result in a slower, more indecisive decision-making process with an unlimited playing field. Damasio *et al.* argue that humans cannot consciously mull over the plethora of options that any situation offers. They noted that somatic markers rapidly narrow the options by automatically determining the affective consequences of each action.

The study by Sarazin *et al.* (1998) used positron emission tomography to examine the correlations between cerebral glucose metabolism in the dorsolateral and orbitofrontal regions of 13 patients with various lesions of the prefrontal cortex. They found a clear dissociation between cognitive tasks disturbed after damage to the dorsolateral prefrontal cortex and social functioning tasks disturbed after damage to the orbitofrontal prefrontal cortex. Sarazin *et al.,* interpreted their results as partially validating the three distinct components of the prefrontal cortex. Furthermore, they

noted that planning and decision-making in daily life may more often include an affective component as opposed to the planning and decision-making that are required in laboratory tests. They finally noted that the partial independence of the dorsolateral and orbitofrontal prefrontal cortex may explain why the performance on laboratory measures of executive functions may be a poor predictor of patients with frontal lobe damage in daily life, in which affective and social interactions are predominant for decision-making.

If variations in executive functions did lead to deficits in personal and social behaviors, how would these behavioral sequelae manifest themselves? One likely candidate is the category of personality disorders, which are defined as inflexible, enduring, and maladaptive traits that cause significant interpersonal and occupational impairment. Personality disorders are universal and prevalent (e.g., 10%–13%, Coolidge and Segal, 1998). Interestingly, as delineated in the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV;* American Psychiatric Association, 1994), personality disorders may not only express themselves by problems in the regulation of affectivity and interpersonal functioning but also through cognition. Some personality disordered individuals appear to have many of the characteristics of classic executive function deficits, that is, poor judgment, decision-making difficulties, selective attention problems, impulsivity, and inflexibility. Thus, the purpose of the present study was to determine if some personality disorders and their features are the interpersonal manifestations of executive function deficits.

A review of the *DSM-IV* criteria for the borderline, dependent, depressive, histrionic, passive-aggressive, and avoidant personality disorders reveals significant overlap with executive function deficits. For example, the criteria for the borderline personality disorder include goal attainment problems, impulsivity, anxiety, and uncertainty in essential decisions such as the determination of self-image. The dependent personality disorder includes criteria such as difficulties initiating projects, doing things independently, and difficulties in making everyday decisions. The depressive personality disorder includes symptoms of unassertiveness and a tendency to follow rather than lead others. The histrionic personality disorder includes the criteria of suggestibility, that is, opinions and feelings that are easily influenced by others, particularly strong authority figures; impulsiveness in the decision-making process; low frustration tolerance for delays in gratification; and problems in attaining long-term goals or completing long-term projects. The passive-aggressive personality disorder criteria in *DSM-IV* include indecisiveness and erratic behavior. Finally, for the avoidant personality disorder, *DSM-IV* notes that these individuals often have resistance to occupational advancement. The *DSM-IV* criteria for this disorder have explicit assumptions that the resistance is due to feelings of inadequacy, doubts of social competency, and the aversion to social interactions. It would be interesting to determine, in a future study, whether the resistance to occupational advancement was actually due to decision-making difficulties or impairments in problem solving that such advancements would likely reveal.

Deficits in executive functions in childhood have been established in some psychological and neuropsychological syndromes, for example, frontal lobe damage or lesions, attention-deficit/hyperactivity disorder (ADHD), Tourette's syndrome, autism (Pennington and Ozonoff, 1996), and borderline personality disorder features in children (Coolidge *et al.,* 2000). In adults, Raine *et al.* (1992) found evidence of prefrontal cortical dysfunction associated with schizotypal personality features in a normal population. There is also some evidence of cognitive processing deficits attributable to the frontal lobes in the antisocial personality disorder (e.g., Deckel *et al.,* 1996; Elliott and Gillett, 1992; Eysenck, 1964; Lykken, 1957; see Millon and Davis, 1996, for a review of this area). Other types of cognitive deficits, but not executive function deficits, have been found to be associated with the borderline personality disorder (e.g., Andrulonis *et al.,* Glueck *et al.,* 1980; Gardner *et al.,* 1987; Kimble *et al.,* 1997; Murray, 1979; Quitkin *et al.,* 1976; Soloff and Millward, 1983; van Reekum, 1993).

There is a growing body of evidence that personality disorders are heritable (e.g., Torgersen *et al.,* 2000), and there is evidence that their heritability may be evident in childhood and adolescence (Coolidge, Thede, and Jang, 2001). For reviews of studies on the heritability of personality disorders, see Hersen and Van Hasselt, 2001, and Millon *et al.,* 2000. Certainly, there are numerous studies supporting the diagnostic validity of personality disorders in childhood and adolescence (for extensive reviews of this literature, see Bleiberg, 2001, or Kernberg *et al.,* 2000). Furthermore, there is recent evidence for the heritability of some types of parent-rated executive functions (Coolidge *et al.,* 2000) and for the validity of parent-rated measures of executive functions (Gioia *et al.,* 2000; Silver *et al.,* 1999). The present study is predicated on the assumption that personality disorders can be measured dimensionally rather than categorically and that appropriateness of the use of normal populations to study

personality disorders and their features has been previously demonstrated (e.g., Jang *et al.,* 1996; Livesley *et al.,* 1993; Raine *et al.,* 1992).

The present study used the Coolidge Personality and Neuropsychological Inventory for Children (CPNI; Coolidge, 1998; Coolidge *et al.,* 1990, 1992, 2002). It is a *DSM-IV*–based parental measure of the features of 12 personality disorders, as well as executive function deficits. The construct validity of the CPNI Executive Function Deficits scale has been demonstrated in a study of ADHD children (Friedman, 1998). Friedman found that ADHD children scored significantly lower on the scale than a matched control sample. Coolidge *et al.* (2000) found a bivariate heritability of the CPNI scales for ADHD, conduct disorder (CD), and oppositional defiant disorder (ODD) with the CPNI Executive Function Deficits scale. Thede and Coolidge (2002) found that autistic children scored significantly lower on the CPNI Executive Function Deficits scale than a matched sample. Coolidge *et al.* (1994) found that juvenile nonviolent offenders scored significantly lower on this scale than a matched group of normal adolescents, although they found no differences on the scale between first-time and repeat offenders. Coolidge *et al.* (2000) found the scale to be lower in children with borderline personality disorder features than matched controls.

If personality disorders are personal and interpersonal manifestations of executive function deficits, then a bivariate heritability (two syndromes with shared genotype) should be evident. Such evidence might help clarify and delineate the nature of the cognitive deficits that the *DSM* states often accompany personality disorders. Such a relationship might also help to explain why personality disorders are typically so often unamenable to traditional psychological treatment. The present study used a community sample of 314 child and adolescent twins to investigate, through structural equation modeling, the bivariate relationship of personality disorders (or their features or traits) to executive function deficits. Based on a review of the criteria on Axis II of *DSM-IV,* it was hypothesized that the borderline, dependent, depressive, histrionic, passive-aggressive, and avoidant personality disorders would exhibit a bivariate heritability with executive function deficits. Based on a study of adults by Raine *et al.* (1992), it was also hypothesized that the schizotypal personality disorder would share a bivariate heritability with executive function deficits. Also, based on a number of lines of evidence in adults with antisocial personality disorder (e.g., Millon and Davis, 1996) and our previous investigation with conduct disorders (Coolidge *et al.,* 2000), it was hypothesized that there would be a bivariate heritability between conduct disorder and executive function deficits. Based on a review of *DSM-IV* criteria and relevant literature, we had no reason to suspect that executive function deficits might be related to the obsessive-compulsive, paranoid, schizoid, and narcissistic personality disorders.

METHODS

Participants and Procedure

Parents of 314 twins from Colorado Springs, Colorado, USA, and Vancouver, British Columbia, Canada, were recruited by advertising (e.g., Internet, local newspapers), visits to local Parents of Multiple Births group meetings, and by university students who earned extra credit by identifying parents of twins in the community.

The sample consisted of 157 twin pairs, 96 monozygotic (MZ) pairs (44 male pairs and 52 female pairs) and 61 dizygotic (DZ) pairs (20 male pairs, 20 female pairs, and 21 male/female pairs). The mean age of the MZ pairs was 9.4 years (SD = 3.4), and the range of ages was 5 to 17 years. The mean age of the DZ pairs was 10.1 years (SD = 3.6), and the range of ages was also 5 to 17 years. The mean age of the parents was 39.5 years (SD = 6.3), and 85% of the parents had attained a level of education beyond high school. The mean maternal age at time of birth was 29.5 years (SD = 5.3). Ethnicity was as follows: MZ twins, Caucasian (83%), Hispanic (4%), Asian (6%), African American (2%), and other (5%); DZ twins, Caucasian (93%), Asian (2%), and other (5%).

Zygosity was assessed by the parents of the twins using a 10-item questionnaire based on a study by Cohen *et al.* (1975) and contained items regarding physical similarities (e.g., height, weight, hair, and eye color) and confusion of the twins by parents, family, and strangers. Their questionnaire has been demonstrated to be approximately 90% valid (compared to blood-typing).

Materials

Participating parents completed demographic information and the CPNI on each child at their home or at the university. The parents completed the two CPNI forms for their twins on separate days in order to reduce any contrast effects (preconceived views that MZ twins are more alike than DZ twins) or effects from repeating the same procedure simultaneously. Informed consent was obtained.

The 200-item, parent-as-respondent CPNI for children and adolescents (Coolidge, 1998; Coolidge *et al.,* 2002) assesses (a) the presence of 10 personality disorders according to the specific criteria on Axis II of *DSM-IV* and two personality disorders in its appendix, and (b) measures executive function deficits. The CPNI uses a 4-point Likert scale ranging from *(1) strongly false* to *(4) strongly true* and is designed to be completed by a parent. The CPNI normative sample consists of 780 children, ages 5 to 17 years old. The 12 personality disorder scales have a mean internal scale reliability of .68 and a median test-retest reliability of .81 (4- to 6-week interval). The 16-item Executive Function Deficits scale has an internal reliability of .92 and a test-retest reliability of .85. The scale was created from pertinent neuropsychological literature assessing symptomatology consistent with dysfunction of the prefrontal and frontal lobes of the cortex. The scale contains items measuring decision-making difficulties, organizational impairments, poor planning, perseveration, and sequencing difficulties. None of the 16 items of the Executive Function Deficits scale overlap with any of the items on the 12 personality disorder scales.

Statistical Methods

Behavioral genetic designs typically estimate additive genetic influences (A), environmental influences shared in common (C), and nonshared environmental influences including error (E) on the variance and covariance between variables. Additive genetic influences represent genetic effects that are passed directly from parent to offspring. Shared environmental influences affect all children within a family to the same degree (e.g., Rowe, 1994), whereas nonshared environmental factors are those events that have differential effects on individual family members such as differential parental treatment (e.g., Hetherington, Reiss, and Plomin, 1994). It should be noted that E also contains error variance, because it is estimated as a residual term after the effects of A and C have been removed.

Genetic influence on the variance of a single variable is suggested when the similarity of MZ twins, measured by bivariate correlation, exceeds the DZ twin correlation, because the increased MZ similarity can be attributed to the two-fold greater genetic similarity of MZ compared to DZ twins. In the multivariate case, the focus of the present study, common genetic influences are suggested when the MZ cross-correlation (i.e., the correlation between one twin's score on one of the variables and the other twin's score on the other variable) is greater than the DZ cross-correlation. Note, however, that the estimation of genetic covariance is limited to pairs of variables that were influenced by the same genetic and environmental components. For example, if univariate heritability analyses determined that one variable is influenced by additive genetic and nonshared environmental influences, and the other by shared and nonshared environmental influences, no genetic covariance can be estimated. As such, the first step is to estimate the univariate heritability of each variable to determine which components of variance are present.

Heritability Estimation

Heritability analyses were conducted for each of the CPNI scales in the present sample. MZ and DZ twin similarity (Pearson's r) was estimated for all variables using the computer program PRELIS 2 (Jöreskog and Sörbom, 1993). Structural equation models estimating the magnitude of genetic and environmental influences were then fit to matrices of MZ and DZ Pearson correlations, and asymptotic weights were computed using the computer program Mx (Neale *et al.,* 1999) by the method of weighted least squares (WLS) suitable for non-normally distributed data (Neale and Cardon, 1992). WLS was applicable to the present general population sample, because the distributions of some variables were skewed due to relatively few subjects endorsing extremely high levels of some behaviors. The first model fit to the data was the "full model" that specified A, C, and E influences. The full model was then systematically modified to test the significance of A, C, and E by fitting a series of "reduced" models. These models systematically removed the effects of (1) additive genetic variance (CE model), (2) shared environmental variance (AE), and (3) additive genetic and shared environmental variance (E only model).

The relative fit of each reduced model was assessed by testing the difference in likelihood ratio χ^2 values between the full and reduced models. The critical value of χ^2 to test the χ^2 difference is determined by the difference in the number of degrees of freedom (df) between the full and reduced model under consideration. The reduced model was rejected whenever the χ^2 difference exceeded the critical value of χ^2. Model-fit was also assessed in conjunction with two additional criteria: (1) the principle of parsimony, and (2) Akaike's Information Criterion (Akaike, 1987: AIC = $\chi^2 - 2df$). The model reported was the one that did not significantly increase χ^2, that accounted for the variance with the fewest number of parameters, and that yielded the smallest value of AIC.

Multivariate Genetic Analyses

The relationship between each of the CPNI personality disorder scales and the CPNI Executive Function Deficits scale was studied by estimating the genetic (r_G) and environmental (r_E) correlations. These correlations yield an index that varies between +1.0 and −1.0, which estimates the extent to which two variables are influenced by common genetic and/or environmental influences. r_G and r_E are estimated in a way similar to that used to estimate the heritability of a single variable. The magnitude of the observed correlation (r_p) between two variables (within measurement error) is explained by: $r_p = (h_i \times h_j \times r_G) + (e_i \times e_j \times r_E)$. The terms h and e are the square roots of the heritability and nonshared environmental estimates (h^2 and e^2) for variables *i* and *j*, respectively. Bivariate r_G and r_E were estimated by fitting structural equation models known as a "Cholesky decomposition" to pairs summarized in matrices of MZ and DZ correlations using the computer program Mx (Neale *et al.,* 1999) by the method of weighted least squares (WLS) suitable for non-normally distributed data (Neale and Cardon, 1992).

RESULTS

We first tested six equal environment assumptions for their relationship with executive function deficit scores. Three of the assumptions had positive correlations with the executive function deficit scores, and three had negative correlations. The average absolute value of the correlation coefficients for the six equal environment assumptions was $r = .16$. Therefore, it was concluded that there was no violation of the equal environment assumptions with regard to the Executive Function Deficits scale.

From Table I, the MZ correlations exceeded the DZ correlations for all CPNI scales, suggesting the presence of a genetic influence on all scales. The left half of Table II presents the model-fitting statistics for the CPNI scales. A model specifying only additive genetic and nonshared environmental influences (AE) yielded the most satisfactory fit to all scales except Obsessive-Compulsive and Narcissistic personality disorders. A full model specifying additive genetic, shared, and nonshared environmental influences (ACE model) provided the most satisfactory fit to the Obsessive-Compulsive personality disorder scale, and for the Narcissistic personality disorder scale, a wholly environmental model specifying shared and nonshared influences (CE model) was sufficient. The right half of Table II provides the parameter estimates and an estimate of the standard error from most satisfactory model fit to each CPNI scale.

Table III provides the heritability estimates (squared parameter estimates from Table II), the phenotypic (Pearson's r), genetic (r_G), nonshared environmental correlations (r_E), and the bivariate heritability estimates (h^2_B: the proportion of the phenotypic correlation between the additive genetic and nonshared environmental influences). Given that no heritable influences were detected for the Narcissistic personality disorder scale, no Cholesky decomposition was attempted to compute the genetic correlation with the Executive Function Deficits scale. A Cholesky decomposition specifying A and E effects provided a

Table I. Descriptive Statistics of CPNI Scales and MZ and DZ Twin Correlations

	T Scores				
Scale	Mean (SD)	Minimum	Maximum	r_{MZ}	r_{DZ}
Paranoid	47.5 (10.0)	30.8	86.6	.59**	.34*
Borderline	47.9 (9.9)	29.4	83.1	.75**	.42**
Obsessive-Compulsive	46.7 (10.0)	30.1	75.3	.76**	.56**
Dependent	50.1 (11.3)	28.1	84.4	.75**	.26
Schizotypal	48.5 (8.9)	33.9	99.0	.77**	.30
Narcissistic	46.7 (9.1)	34.6	85.9	.73**	.59**
Conduct Disorder	48.0 (8.2)	38.2	110.2	.57**	.21
Schizoid	48.5 (9.5)	32.2	84.8	.74**	.45**
Avoidant	49.0 (11.2)	35.3	88.2	.65**	.26
Histrionic	48.4 (10.4)	35.7	81.5	.78**	.40*
Passive-Aggressive	48.5 (10.1)	32.6	88.6	.69**	.30
Depressive	46.7 (10.2)	30.7	78.2	.66**	.44**
Executive Function	49.0 (11.1)	35.7	86.8	.77**	.03

Note: $N_{MZ} = 97$ pairs; $N_{DZ} = 60$ pairs. 2-tailed Signif: *$p < .01$; **$p < .001$.

Table II. Model-Fitting Statistics (likelihood ratio χ^2), Heritability Estimates with 95% Confidence Intervals

	Model parameters				Parameter standard and error estimates		
Scale	ACE	AE	CE	E	h^2	c^2	e^2
Paranoid	.00	.15	3.74	83.05	.61 (.47–.79)	—	.39 (.21–.53)
Borderline	.00	.19	7.96	281.48	.77 (.64–.94)	—	.23 (.06–.36)
Obsessive-Compulsive	.00	3.92	4.03	334.88	.40 (.22–.53)	.36 (.19–.49)	.24 (.07–.37)
Dependent	.94	.94	14.64	276.81	.77 (.64–.94)	—	.23 (.06–.37)
Schizotypal	.51	.51	13.93	324.46	.76 (.63–.93)	—	.24 (.07–.37)
Narcissistic	.00	6.67	1.92	262.36	—	.69 (.53–.86)	.31 (.16–.45)
Conduct Disorder	.39	.39	6.57	688.22	.57 (.42–.74)	—	.43 (.26–.58)
Schizoid	.00	.52	6.43	250.94	.76 (.63–.93)	—	.24 (.07–.37)
Avoidant	.29	.29	8.43	120.76	.68 (.54–.85)	—	.32 (.15–.46)
Histrionic	.00	.00	10.65	363.51	.82 (.67–.99)	—	.19 (.01–.31)
Passive-Aggressive	.15	.15	8.95	159.97	.69 (.56–.86)	—	.31 (.14–.44)
Depressive	.00	.96	3.41	140.25	.68 (.55–.85)	—	.32 (.15–.46)
Executive Function	7.12	7.12	29.05	326.39	.76 (.63–.93)	—	.24 (.07–.37)

Note: A, a = additive genetic effects; C, c = shared environmental effects; E, e = nonshared environmental effects; $df_{ACE} = 3$; $df_{AE} = 4$; $df_{CE} = 4$; $df_E = 5$.

Table III. The Phenotypic (r_P), Genetic (r_G), Environmental (r_E) Correlations with 95% Confidence Intervals, and the Bivariate Heritability Estimates (h^2_B) Between the Personality Disorder Scales and the 16 Item Executive Function Deficits Scale

	Correlations with executive functions			Model fit	
	r_P	r_G	r_E	h^2_B	χ^2
Paranoid	.39	.64 (.50–.78)	.04 (−.18–.29)	.43	16.18
Borderline	.53	.69 (.58–.79)	.32 (.11–.71)	.52	16.08
Obsessive-Compulsive	.54	—	—	—	44.83*
Dependent	.62	.78 (.68–.86)	.30 (.09–.66)	.58	12.82
Schizotypal	.48	—	—	—	26.72*
Narcissistic	.51	—	—	—	—
Conduct Disorder	.37	.51 (.35–.67)	.20 (.01–.47)	.34	9.60
Schizoid	.20	.36 (.22–.51)	−.07 (−.35–15)	.27	18.86*
Avoidant	.48	.72 (.61–.83)	.21 (.01–.49)	.50	17.84
Histrionic	.59	.83 (.75–.91)	.14 (−.09–1.00)	.64	18.56
Passive-Aggressive	.56	.77 (.67–.87)	.22 (.03–.51)	.56	13.19
Depressive	.50	.77 (.67–.87)	.06 (−.17–.29)	.55	22.56

Note: All r_P significant at $p < .001$. *$p < .05$, df = 14.

satisfactory fit to the Executive Function Deficits scale and each of the personality disorder scales except Obsessive-Compulsive, Schizotypal, and Schizoid (see last column of Table III). The poor fit of the model with these three scales was due to the excessive positive skew found in each scale's distributions directly attributable to low item endorsement rates for these pathological behaviors in a general population sample. From the successful decompositions, the genetic and environmental covariances were estimated and then converted into the genetic and environmental correlations (middle of Table III). Substantial genetic correlations were found between most of the remaining personality disorder scales. The environmental correlations were uniformly smaller. The second to last column in Table III presents the bivariate heritability estimates. These estimates ranged from .27 for the Schizoid scale to .64 for the Histrionic scale.

DISCUSSION

With regard to the original hypotheses, seven of the eight hypothesized relationships were found to be significant. There were significant genetic influences

between the Executive Function Deficits and Avoidant, Borderline, Dependent, Depressive, Histrionic, Passive-Aggressive, and Conduct Disorder personality disorder scales. We failed to find significance for the Schizotypal scale with the Executive Function Deficits scale. There were also two scales that produced significant bivariate heritabilities with executive function deficits that were not expected: the Paranoid and Schizoid scales. The reasons for these unexpected relationships are not imminently clear, although they share an emotional coldness. The finding will obviously require further study. Consistent with our previous research (Coolidge *et al.,* 2001), most of the individual personality disorder scales had high individual heritability estimates (median = .69), as did the Executive Function Deficits scale (.77). Interestingly, these individual heritability estimates are consistent with a previous twin study of adult personality disorders by Torgerson *et al*. (2000), who found a median heritability for nine adult personality disorders of .61.

These findings, if substantiated, may help to explain why individuals with personality disorders often have chronic difficulties with everyday decisions, selective attention and inhibition, judgments, choices, planning, and flexibility. For example, the Avoidant personality disorder scale had a substantial bivariate heritability of .50 with the Executive Function Deficits scale. The latter finding leads us to speculate that at least in adults the *DSM-IV* criterion "resistance to occupational advancement" may be due, in part, to a deficiency in executive functions. Furthermore, it appears possible that the feelings of inadequacy, low self-esteem, and doubts of social competence in avoidant personality–disordered individuals may be, at least in part, a consequence of their executive function deficits. Even their resistance to social interactions and reluctance to new endeavors and activities may arise as a consequence of executive function deficits. These findings may also explain why therapy with personality-disordered individuals is often considered so extraordinarily difficult (e.g., Millon and Davis, 1996). One reason may be that if therapies focus primarily on insight, they may not alleviate the symptoms with a predominant heritable influence. As Shallice and Burgess (1991) have noted, patients with executive function deficits are often aware of their difficulties in social situations and have the intellectual abilities to generate ideas that can solve their problems; yet their efforts are negligible, inappropriate, or haphazard. However, because of the preliminary nature of the present investigation, we should be wary of selecting specific criteria from the *DSM-IV,* and cautiously conclude from the current findings that perhaps *some* of the personality disorder symptoms may be due to executive function deficits.

Variations in executive functions in the present study were not measured by traditional laboratory measures (see Pennington and Ozonoff, 1996, for a review of 30 common measures), and the findings in the present study hinge on the assumption that the Executive Functions Deficit scale of the CPNI validly measures impairments in executive abilities. Traditional measures such as the Wisconsin Card Sorting Task, the Stroop Task, and measures of verbal fluency may be more sensitive to damage in the dorsolateral prefrontal cortex rather than the orbitofrontal prefrontal cortex (e.g., MacPherson *et al.,* 2002; Sarazin *et al.,* 1998). In the present study, the findings of a coheritability between executive functions and personality disorders may be due to the broader social and behavioral ramifications associated with the CPNI Executive Functions scale. It may be that the parents in the present study are rating their children's decision-making, planning, and flexibility in a more ecologically valid context, and it may be that the present scale is possibly more sensitive to dysfunction of the orbitofrontal cortex. It could be argued that the decision-making processes involving the orbitofrontal cortex are also executive functions of a different nature than those executive functions associated with the dorsolateral prefrontal cortex. As Sarazin *et al*. (1998) have noted, these two functional circuits may be relatively independent of each other, and this may explain why the laboratory performance of frontal lobe damaged patients on classic executive functions cognitive tests are a poor predictor of the patient's ability to perform in daily life. Thus, personality disorders may be manifestations of executive function deficits, because everyday decisions, inhibitions, judgments, and flexibility are expressed in an affect-laden context with emotional and social repercussions, which classic cognitive measures of executive functions lack.

The present study used only parental ratings. It might have been strengthened if we had used additional raters or if one parent evaluated personality disorders and the other parent evaluated the same child for executive function deficits in order to avoid any halo effects or expectation biases associated with the same parent completing both ratings. There is also the problem of parental expectations that MZ twins are more alike than DZ twins, and this is a distinct problem for twin studies that use parental ratings. However, the literature in this area has provided a clear consensus that demand characteristics, social desirability, and acquiescence may all play a role in a rater's response,

although a majority of the variance in a participant's response is due to the content of the question (Stone *et al.,* 2000).

Another limitation of the present study is the controversy of whether personality disorders and their features are evident and measurable in children (see Wolff, 1993, or Kernberg *et al.,* 2000, for reviews of this controversy). The *DSM-IV* clearly states that personality disorders may be diagnosed in childhood or adolescence when the maladaptive behaviors are "pervasive, persistent, and unlikely to be limited to a particular developmental stage or an episode of an Axis I disorder" (p. 631). Future researchers may wish to ascertain the presence of personality disorders or their features in children by clinical diagnostic interviews with the children and their parents.

There is also the issue of using a community-based sample for studying clinical disorders. Although future research would clearly benefit from the use of clinical samples, there is ample evidence that normal twin samples provide sufficient variation to study abnormal behavior, particularly personality disorders (e.g., Jang *et al.,* 1996; Livesley *et al.,* 1993). There was, however, such a positively skewed distribution (absence of pathology) in the present twin sample on the Schizotypal Personality Disorder scale, that previous research with a normal sample (e.g., Raine *et al.,* 1992) could not be replicated. Although few individuals were actually positive for each disorder, it is important to note that the concept of a personality disorder was approached in the present study dimensionally rather than categorically. The overall twin sample size could also be considered relatively small. This not only limits the power to detect shared environmental influences, but also to compare gender-specific genetic and environmental contributions to the traits studied.

The biological foundations of personality disorders are without question (e.g., Livesley, 1995; Millon and Davis, 1996). Also, it is stated in the *DSM-IV* that personality disorders may express themselves in the cognitive domain. However, heretofore there has been little speculation as to how disruptions in decision-making, sequencing, goal-attainment strategies, and other behaviors that are thought to constitute executive functions might influence the personality intrapsychically and interpsychically. The present study investigated this important link. As noted earlier, executive functions have been classically defined to include complex decision-making for the purpose of goal attainment. Yet, traditional laboratory measures often remain in a strict cognitive realm and may not encompass the personal and social ramifications of the decision-making involved in everyday life. The present study offers preliminary evidence that a relatively prevalent form of psychopathology, personality disorders or their features, may share a common genetic origin with executive functions. These findings might also highlight the need to evaluate executive functions in clinical assessment in a broad manner and beyond the scope of traditional measures, even when neuropsychological impairment is not suspected.

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