



Personality and neuropsychological correlates of bullying behavior[☆]

Frederick L. Coolidge^{*}, John W. DenBoer, Daniel L. Segal

Department of Psychology, University of Colorado at Colorado Springs, Colorado Springs, CO 80933-7150, USA

Received 25 November 2002; received in revised form 15 April 2003; accepted 8 June 2003

Abstract

The psychological and neuropsychological correlates of bullying behavior were examined in a group of 41 middle school students (age range 11–15 years) and group-matched controls. The students were identified as bullies by school administrators, their teachers, and self-ratings. Parents of children in both groups completed the Coolidge Personality and Neuropsychological Inventory, a 200-item, *DSM-IV-TR* aligned, parent-as-respondent, standardized measure. It was found that bullying behavior was associated more with *DSM-IV-TR* Axis I diagnoses of conduct disorder, oppositional defiant disorder, attention-deficit/hyperactivity disorder, and depressive disorder than in matched controls. Bullying behavior was also correlated more with Axis II diagnoses of passive-aggressive, histrionic, paranoid, and dependent personality disorders than in matched controls. Bullying behavior was also more correlated with measures of neuropsychological dysfunction and executive function deficits. An implication of these findings is that traditional short-term psychotherapeutic interventions for bullying behavior may be of limited value given the complex nature of the associated psychopathology.

© 2003 Elsevier Ltd. All rights reserved.

Keywords: Bullying; Personality disorders; Passive-aggressive personality disorder; Executive function deficits; Depression

1. Introduction

In a variety of forms, school violence pervades American society. Various methods of research have been undertaken in an effort to pinpoint the etiology of school violence, particularly bullying

[☆] A version of this article served as the Master's thesis for John DenBoer.

^{*} Corresponding author. Tel.: +1-719-262-4146; fax: +1-719-262-4166.

E-mail address: fcoolidg@mail.uccs.edu (F.L. Coolidge).

behavior in schools. Definitions of bullying behavior incorporate such factors as an evident power differential, physical and/or verbal abuse, and severity and duration of abuse (Atlas & Pepler, 1998). Olweus (1991) defines bullying and victimization as the exposure of an individual, repeatedly and over time, to negative actions on the part of one or more others. Bullying can take physical forms, such as hitting, pushing, kicking, or punching, and/or verbal forms, exemplified in threatening, teasing, taunting, and name calling.

A major theoretical orientation for the understanding of developmental psychopathology was offered by Spreen (1989). Spreen proposed that psychiatric disturbances are frequently associated with neuropsychological dysfunction as a result of a common biological origin. Whereas Spreen noted that toxins and the prenatal environment might be sources of influence, he emphasized that the stronger etiological agent was genetic. Support for this argument was provided by Yeudall, Fromm-Auch, and Davies (1982) in their study of 99 juvenile delinquents. They found that 84% of the delinquents had evidence of neuropsychological deficits compared to only 11% of a control sample. A multitude of related research provides support for subtle neurological deficits in children and adolescents with borderline personality disorder features and characteristics of other personality disorders (Coolidge, Segal, Stewart, & Ellett, 2000; Cowdry, Pickar, & Davies, 1985; Gardner, Lucas, & Cowdry, 1987; Quitkin, Rifkin, & Klein, 1976; Shaffer, Davidson, & Saron, 1985). However, the Yeudall et al. study did not specifically assess bullying behavior, and their application to the study of bullying behavior remains speculative.

Another theoretical framework for the understanding of bullying behavior was provided by Grigsby and Stevens (2000) who suggest that appropriate functioning of the frontal lobes serves as a basis for appropriate social behavior as well as the basis for inhibition of inappropriate and irrelevant behavior. Based on this theory, it is plausible that bullies may lack sufficient frontal lobe functioning, which would be a requirement for them to be able to follow directions from others and obey authority figures. Additionally, bullies may lack the capability to inhibit their aggressive and inappropriate verbal and physical actions. Indeed, in a group with similar problems as bullies, juvenile delinquents were found to have significantly more executive functions deficits than non-delinquent controls (Coolidge et al., 1992).

The majority of bullying research has focused on the distinctive characteristics of bullies and victims. Although empirical investigations have dealt with the attitudinal and behavioral aspects of school bullies (Atlas & Pepler, 1998; Craig, 1998; Glover, Gough, Johnson, & Cartwright, 2000; Whitney & Smith, 1993), there is presently limited information concerning bullying behavior vis-à-vis diagnoses from the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000) and the relationship of bullying to neuropsychological behavioral function.

A variety of studies have dealt with psychological disturbance in bullies and/or victims. Kumpulainen, Rasanen, and Henttonen (1999), in a longitudinal study of 1268 children aged 8 and 12 years studied at two time points for incidence of bullying behavior, found that children who bully had significantly more psychiatric symptoms than other children. Specifically, bullies exhibited greater psychopathology on externalizing disorders and greater hyperactivity than controls. Additionally, Craig (1998) found that bullies exhibited more antisocial behavior and physical aggression than non-bullies, but they also exhibited lower levels of anxiety. In contrast, victims showed increased depression and anxiety. Bullies, in Craig's study, did not show elevated levels of depression. This finding is consistent with previous research examining psychiatric

symptoms among bullies and victims (Neary & Joseph, 1994; Slee, 1995). Olweus (1991) identified 13–16-year-old bullies as having an antisocial personality combined with physical strength, whereas victims were found to have an anxious personality pattern combined with physical weakness. Although previous research has examined some psychiatric symptoms among bullies (Baldry & Farrington, 2000; Craig, 1998; Kumpulainen et al., 1998, 1999), the major deficiencies in these studies have been the failure to define operationally clinical symptoms in terms of the criteria in *DSM-IV-TR* and failure to examine the full range of personality disorders according *DSM-IV-TR*.

The purpose of the present study was to examine the association between bullying behavior and *DSM-IV-TR* clinical (Axis I) and personality disorders (Axis II), neuropsychological dysfunction, and other psychopathological behaviors. The first hypothesis was that bullies would show greater levels of Axis I conduct disorder, oppositional defiant disorder, and ADHD compared to controls. The second hypothesis was that bullies would show higher levels of some personality disorders or their traits than controls. Although the diagnosis of personality disorders in children is somewhat controversial, the *DSM IV-TR* allows the diagnosis of personality disorders under the age of 18 in cases where the behavior is pervasive, persistent (at least one year), and unlikely to be limited to a developmental stage. There is currently strong genetic evidence for the heritability of personality disorders in both children (Coolidge, Thede, & Jang, 2001) and adults (Torgersen et al., 2000). For a review of the diagnosis and treatment of personality disorders in children and adolescents, see Bleiberg (2001) and Kernberg, Weiner, and Bardenstein (2000). The third hypothesis was that bullies would show higher levels of neuropsychological behavioral dysfunction than controls. The fourth hypothesis was that bullies would show greater levels of dangerousness, aggression, emotional lability, and disinhibition compared to controls.

2. Method

2.1. Participants and procedure

This study involved 41 public middle school students (22 males, 19 females; mean age = 12.6 years, $SD = 0.9$ years, age range 11–15 years; 11 sixth-graders, 18 seventh-graders, 12 eighth-graders; 15 Whites, 16 Blacks, 7 Hispanics, 3 American Indians) who were identified by school counselors as having three or more office referrals from administrators or teachers in a school year. The referrals were a result of any of the following behaviors: name calling, fighting, relentless picking on other students, defiance toward teachers, or getting kicked out of an in-school suspension class. These students constituted the bully group. A control group was chosen, as closely matched as possible, for gender, age, and grade level ($N = 41$; 22 males, 19 females; mean age = 12.7 years, $SD = 0.9$ years, age range 11–14 years; 7 sixth-graders, 21 seventh-graders, 13 eighth-graders; 23 Whites, 7 Blacks, 6 Hispanics, 5 American Indians). They had no office referrals in the past school year. There were no significant differences between the bully and non-bully groups on their age (t test), gender, grade levels, and percentages of Whites versus minorities (chi square tests).

A school counselor contacted the parents of each child to complete the measures. Informed consent was obtained from all participants, including parents, teachers, and students. At no time

during the study were the students or parents made aware of the nature of their group classification for the present study. The parents of the children in the bully group were contacted by the school counselor as part of the standard administrative process in such referrals. The parents were asked if they wished to volunteer to complete the study questionnaires. The majority of the parents completed the measures at school during parent–teacher conferences, although some completed the surveys at home. The students completed their measures at school. The parents of the children in the control group were contacted by the school counselor and asked to volunteer their time for a project being conducted by the school counselor and a local university (identified by name).

2.2. *Materials and procedure*

The parents of the children completed the Coolidge Personality and Neuropsychological Inventory (CPNI; Coolidge, 1998; Coolidge, Thede, Stewart, & Segal, 2002), a standardized measure of children's and adolescents' (ages 5–17 years) psychological functioning. The 200-item, parent-as-respondent CPNI assesses (a) five Axis I syndromes from *DSM-IV-TR* (conduct disorder, oppositional defiant disorder, attention-deficit/hyperactivity disorder [ADHD], depressive disorder, and overanxious disorder of childhood), (b) nine personality disorders and their features (avoidant, borderline, dependent, histrionic, narcissistic, obsessive–compulsive, paranoid, schizoid, schizotypal) according to the criteria on Axis II of *DSM-IV-TR* and two personality disorders in its appendix (passive–aggressive and depressive; note: antisocial personality disorder is not assessed by the CPNI because it requires an age of 18-years-old), (c) three neuropsychological-behavioral syndromes including mild neurocognitive disorder (in the appendix of *DSM-IV-TR*), general neuropsychological dysfunction, and executive function deficits (and its three subscales: decision-making, metacognitions, and social judgment), and (d) four clinical scales, Dangerousness, Aggression, Emotional Lability, and Disinhibition. The CPNI uses a 4-point Likert scale ranging from (1) *strongly false* to (4) *strongly true*. The CPNI normative sample consists of 780 children, ages 5–17-years-old. The 11 personality disorder scales have a median internal scale reliability of 0.67 and a median test–retest reliability of 0.81 (four to six week interval). The five Axis I scales have a median internal scale reliability of 0.81 and a median test–retest reliability of 0.87. The three neuropsychological scales have a median internal scale reliability of 0.91 and a median test–retest reliability of 0.83. The four clinical scales have a median internal scale reliability of 0.61 and a median test–retest reliability of 0.64.

The general construct validity of the CPNI scales has been demonstrated in a variety of clinical and non-clinical empirical studies (Coolidge, Aksamit, & Becker, 1994; Coolidge et al., 1992; Coolidge, Segal, et al., 2000; Coolidge et al., 2001; Coolidge, Thede, & Jang, 2004; Coolidge, Thede, & Young, 2000; Coolidge, Thede, & Young, 2002). See Coolidge, Thede, Stewart, et al. (2002), for a summary of the CPNI reliability and construct validity studies.

In order to assess bullying behavior in both groups, the teachers were asked to complete a non-standardized measure, Weinhold's survey of bullying and related behaviors. The measure is a shortened version of a bullying survey adapted from the work of Espelage (Espelage, Bosworth, & Simon, 2000). Weinhold's teacher measure contains 20 items of bullying behavior observed in the last 30 days. The items are rated on a 5-point Likert scale ranging from (0) *never* to (4) *seven or more times*. Both groups of students also completed Weinhold's student self-report measure of bullying and related behaviors. This scale contains 15 items and is also measured on a 5-point

Likert scale. Because previous estimates of internal scale reliabilities for the two Weinhold measures were not available, they were calculated on the present sample ($N = 82$), and they were 0.84 for the teacher version and 0.94 for the student version.

3. Results

The validity of the diagnosis of the two groups (bullying and controls) was established by performing t tests for independent samples on the sum of the Weinhold's teacher's measure and student's measure of bullying behavior. The mean teacher's rating of bullying behavior for the bullying group ($M = 33.2$, $SD = 14.9$) was significantly higher than the control groups' mean ($M = 5.6$, $SD = 4.6$), Welch's t (44.9) = 11.06, $p < 0.0005$. The mean student's self-ratings of bullying behavior for the bullying group ($M = 14.2$, $SD = 10.2$) was also significantly higher than the control groups' mean ($M = 5.4$, $SD = 7.0$), Welch's t (67.3) = 4.43, $p < 0.0005$. Together, these results show that the referred bullies were rated by their teachers and self-rated higher on bullying scales than the controls. Thus, these findings help to establish the validity of the bullying diagnosis.

3.1. Clinical (Axis I) scales

A multivariate analysis of variance (MANOVA) was performed on the five Axis I scales of the CPNI for the main effect of group (bully and controls). The MANOVA was significant, approximate $F(5, 76) = 4.01$, $p = 0.003$. Post hoc univariate analyses of variance (ANOVA) with a modified Bonferroni correction (Holm, 1979) revealed that the Conduct Disorder, Oppositional Defiant Disorder, ADHD, and Depressive Disorder scales produced a significant main effect for the bullying diagnosis. One scale (Overanxious Disorder of Childhood) was not significant (see Table 1). All three Axis I hypotheses were supported. The bullying group produced significantly higher means than the controls for Conduct Disorder, Oppositional Disorder, and ADHD scales, and all had large effect sizes. Inspection of the bullying group revealed that 46%, 49%, and 51% of the individuals were clinically elevated (T scores ≥ 60) for the Conduct Disorder, Oppositional Disorder, and ADHD scales, respectively. Although not hypothesized, the Depressive Disorder scale was significantly and clinically elevated (T score ≥ 60) in the bullying group, and it also had a large effect size. Approximately 49% of the children in the bullying group exhibited a clinical elevation on the Depressive Disorder scale.

3.2. Personality disorder (Axis II) scales

A MANOVA was also performed on the 11 Axis II personality disorder scales. The MANOVA was again significant, approximate $F(11, 70) = 2.58$, $p = 0.008$. Post hoc ANOVA's (with the modified Bonferroni correction) revealed that only the Passive–Aggressive scale was significant. The bullying group was significantly and clinically elevated for the Passive–Aggressive scale with a large effect size. The bullying group was also elevated compared to the control group on the Histrionic, Paranoid, and Dependent scales, and each had a large effect size but the difference did not reach statistical significance with the modified correction (see Table 1).

Table 1

Means, *T* scores, *t* values, and correlation of effect size for bullies and non-bullies on the CPNI

	<i>T</i> scores		<i>t</i>	Sig.	<i>r</i> **
	Bullies (SD)	Non-bullies (SD)			
<i>Axis I</i>					
ADHD	60.4 (10.9)	51.9 (10.9)	3.64	0.001*	0.38
Conduct Dis.	59.9 (15.4)	49.6 (10.5)	3.53	0.001*	0.37
Oppos. Defiant Dis.	60.6 (13.0)	51.9 (11.6)	3.18	0.002*	0.33
Major Depress. Dis.	62.4 (15.6)	54.3 (13.1)	2.55	0.013*	0.27
Overanxious Dis.	50.1 (9.8)	51.4 (11.4)	−0.59	0.558	0.06
<i>Axis II</i>					
Passive–aggressive PD	60.9 (13.7)	50.1 (12.0)	3.80	0.001*	0.42
Paranoid PD	60.1 (10.9)	54.5 (11.1)	2.30	0.024	0.26
Histrionic PD	58.4 (13.4)	51.9 (12.7)	2.25	0.027	0.28
Dependent PD	54.0 (11.7)	49.1 (10.3)	2.01	0.048	0.25
Borderline PD	58.8 (13.1)	53.8 (11.7)	1.84	0.069	0.22
Depressive PD	55.5 (12.6)	51.9 (10.1)	1.41	0.163	0.19
Schizoid PD	56.2 (11.7)	53.7 (11.5)	0.96	0.338	0.15
Narcissistic PD	53.3 (12.6)	51.1 (10.5)	0.89	0.379	0.15
Schizotypal PD	56.2 (11.7)	52.0 (11.6)	0.79	0.432	0.14
Avoidant PD	53.1 (13.4)	53.7 (13.5)	−0.19	0.847	0.00
Obs.–comp. PD	51.8 (9.7)	52.1 (9.6)	−0.14	0.890	0.02
<i>Neuropsychological scales</i>					
Execut. Funct. Def.	61.9 (12.7)	52.2 (11.3)	3.61	0.001*	0.41
Neuropsych. Dysf.	63.1 (12.2)	54.0 (12.2)	3.39	0.001*	0.39
Mild Neurocog. Dis.	62.5 (12.2)	53.6 (11.8)	3.37	0.001*	0.39
<i>Other clinical scales</i>					
Dangerousness	60.9 (13.6)	51.8 (12.9)	3.13	0.002*	0.32
Aggression	57.6 (12.2)	50.0 (11.3)	2.93	0.004*	0.33
Disinhibition	55.8 (11.8)	49.4 (9.8)	2.66	0.009*	0.30
Emotional Lability	58.1 (11.7)	51.6 (11.5)	2.52	0.014*	0.31

Note: * Significant according to modified Bonferroni correction. ** *r* = correlation of effect size; small = 0.100, medium = 0.243, large = 0.371.

It was hypothesized, in general, that some personality disorders would be more prevalent in the bullying group compared to controls, and this hypothesis was partially supported. However, the finding that the bullying group was elevated (compared to controls) with a large effect size on the Histrionic scale was somewhat surprising. Therefore, a post hoc *t* test item analysis was conducted for the eight items on the scale. Interestingly, the item representing Criterion 7 in *DSM-IV-TR* “is suggestible, i.e., easily influenced by others or circumstances” produced the highest *t* value of the eight items, and it had a large effect size.

3.3. Neuropsychological scales

The third hypothesis that the bullying group would also have greater neuropsychological behavioral dysfunction was also supported. A MANOVA was performed on the three neuropsychological scales.

chological scales of the CPNI, and it was again significant, approximate $F(3, 78) = 4.49$, $p = 0.006$. Post hoc ANOVA's (with modified Bonferroni) revealed that the Executive Function Deficits, the General Neuropsychological Dysfunction, and Mild Neurocognitive scales produced significant group main effects. The bullying group was significantly and clinically elevated on all three scales with large effect sizes (see Table 1).

Post hoc t test analyses were also performed on the three subscales of the Executive Function Deficits scale of the CPNI between the bullying and control groups. The three subscales, derived through previous factor analyses, measure (a) decision-making, planning, and organizational problems, (b) metacognitive dysfunctions such as problems with learning, reading, memory, and concentration, and (c) social misjudgments including poor interpersonal decision-making and choices. All three subscales were significantly higher and clinically elevated for the bullying group.

3.4. Other clinical scales

The fourth hypothesis that the bullying group would be elevated on the Dangerousness, Aggression, Emotional Lability, and Disinhibition scales of the CPNI was also supported. A MANOVA was performed on the four clinical scales, and it was significant, $F(4, 77) = 2.99$, $p = 0.024$. Post hoc ANOVA's revealed that the bullying group was significantly elevated on all four scales with large effect sizes (see Table 1).

4. Discussion

The results of the present study provide support for previous findings (e.g., Kumpulainen et al., 1999; Olweus, 1991) that bullying behavior is associated with meaningful levels of psychiatric disturbances. The present study also provides a glimpse of the specific nature of these disturbances. With regard to Axis I syndromes, it was found that bullying behavior was more likely to be associated with conduct disorder, oppositional defiant disorder, ADHD, and depression but not anxiety compared to controls. The depression finding was surprising because depression had been previously found in the victims of bullying but not in the bullies themselves. An item analysis of the Depressive Disorder scale of the CPNI revealed that two of the top four items on the scale that discriminated the best between bullies and controls were both concerned with sadness, low self-esteem, feelings of worthlessness, and depression. This latter finding, if substantiated, might suggest that bullying behavior may be treated, at least in part, by interventions that relieve depression, both psychotherapeutic and psychopharmacological.

The finding that the bullying group in this study was not elevated on the Overanxious Disorder of Childhood scale appears in contrast to the Olweus' (1991) study of bullies in which it was found that bullies tended to display anxious patterns. The T scores for the bullies on this scale in this study were nearly identical to the CPNI normative sample. Perhaps, sampling differences between our sample and Olweus' much larger sample may account for the discrepancy. Certainly, the nature of anxiety among bullies deserves further examination.

The Axis II findings revealed that bullies are more likely to have a constellation of personality disorder features including passive-aggressive, histrionic, paranoid, and dependent behaviors compared to controls (although the latter three were not significant with a modified Bonferroni

correction, they all had large effect sizes). Coupled with the Axis I findings, these results, if replicated, show the deep-seated rejection of rules, institutions, and authority figures, and also show that the nature of bullies is to fail to cooperate with authority figures. Interestingly, however, the histrionic and dependent personality features may be indicative of the general indecisiveness of bullies, their ability to be influenced by others, and their tendency to be driven by excessive, although shallow, displays of emotion. The Paranoid Personality Disorder scale was elevated as well, and it may demonstrate that bullies tend not only to reject rules and authority figures, but also to distrust them and fear harm from them. Bullies with a paranoid personality style may be easily slighted and may aggress against others due to a perceived need for self-protection.

Interestingly, the bullies' elevation on the Histrionic and Dependent scales may be related to each other and related to the neuropsychological findings. The single strongest item endorsed about the bullying group on the Histrionic scale was Criterion 7 "is suggestible, i.e., easily influenced by others or circumstances". The general characteristics of the dependent personality disorder displays this same undue influence upon their behavior by others, including allowing others to make most everyday decisions for them. These findings segue nicely into the present findings that the bullying group was significantly and clinically elevated on the Executive Function Deficits scale. The Executive Function Deficits scale measures three broad areas of functioning of the frontal lobes: (1) decision-making, planning, organizing, (2) learning and integrating information, and (3) making appropriate social judgments. Our findings suggest that executive function deficits in these abilities accompany much of the bullying behavior. Although the bullying group was significantly elevated as a whole versus the control group, only 32% were actually clinically elevated on the Executive Function Deficits scale. This finding may suggest that there may be subtypes of bullies. Certainly, future research may wish to test bullies with laboratory and more traditional measures of executive functions to see if the present findings can be replicated.

With regard to more general neuropsychological dysfunction, it appeared that such symptoms were even more pervasive than executive function deficits in the bullying group. Over 61% of the bullying group had *T* scores indicative of a significant clinical elevation (≥ 60). The parents of the children in the bullying group rated 34% of their children as having a significant reading problem, 37% said their children had problems with math, 27% of the parents said their children had a learning problem, and 56% of the parents said their children had trouble concentrating.

The other clinical scales examined in the present study, Dangerousness, Aggression, Emotional Lability, and Disinhibition were all elevated for the bullying group. These findings support the previous literature suggesting that bullies have problems with impulse and emotional control as well as significant problems with anger management. These findings are also consistent with the Axis I elevations in bullies for conduct disorder and oppositional defiant disorder. These syndromes share the symptoms of a lack of inhibitory capabilities of aggressive behavior, a pronounced lack of empathy, and defiance towards authority.

The results of the present investigation point to the early development (by the beginning of middle school) of a constellation of personality disordered traits and neuropsychological dysfunction in children and adolescents who bully other children. Interestingly, estimates from twin and adoption studies show that conduct disorder and ADHD have a strong genetic component (0.74 and 0.82, respectively; Coolidge et al., 2000). Coolidge et al. have also found that executive function deficits are highly heritable (0.77) and have a bivariate heritability with conduct disorder (0.37) and ADHD (0.79). In another twin study of children and adolescents, Coolidge et al. (2001)

found the passive–aggressive (0.50), histrionic (0.79), paranoid (0.50), and dependent personality disorders (0.81) to be highly heritable also. An important implication of these studies for the present investigation is that the clinical levels of Axis I, Axis II, neuropsychological symptoms and other clinical syndromes in bullies may not be easily ameliorated if these syndromes have a substantial genetic basis. These results might further call into question some conventional treatment methods for bullies such as those designed simply to boost self-esteem or the assumption that a major cause of bullying behavior is simply poor parenting.

As noted earlier, appropriate executive functioning of the frontal lobes allows one to engage actively and appropriately in life and to inhibit irrelevant or inappropriate behavior (Grigsby & Stevens, 2000). From the results of the present study, it appears that many bullies lack the appropriate frontal lobe functioning necessary to follow or implement directions from peers or authority figures. Additionally, it also appears that many bullies lack the capability to inhibit their aggressive and inappropriate verbal and physical actions. More recently, Coolidge et al. (2004) have found genetic evidence from child and adolescent twin studies that personality disorders may be the psychological manifestations of executive function deficits of the frontal lobes. Thus, the comorbidity of Axis I, Axis II, neuropsychological dysfunction, and executive function deficits in the present study of bullies may not be all that surprising.

With regard to treatment programs for bullies, these results have important implications for intervention and prevention programs. From an individual psychotherapeutic treatment perspective, a primary implication of these findings is that the treatment of personality disorders in children may be highly complex. It is also important to note that if neuropsychological dysfunction is determined to be intimately and biologically related to personality disorders in children and adolescents, then traditional psychotherapeutic approaches may be of limited benefit.

The present sample is small, and the results should be deemed preliminary. Future research should examine the impact of gender, ethnicity, and socio-economic status. Our sample was fairly homogeneous in that the bullies were severe enough to be referred to the school counselor. It is possible that less severe bullies would not show as much psychopathological and neuropsychological deficits, and these potential differences should be examined further. Additionally, study of normal personality traits of bullies (in addition to psychopathology) would also be of value. Our study would also be enhanced if both parents, where possible, could complete the psychological measures and clinical interviews could be conducted. It would also be useful to have traditional measures of neuropsychological dysfunction and executive function deficits on the bullies and the control group. Another limitation is that we did not assess possible victimization of the bullies by others, and future studies should include this information. A final limitation is that there was some overlap among our requirements for a diagnosis of bullying and some Axis I disorders, most notably oppositional defiant disorder. However, it would be difficult to study bullies without any oppositional features. In conclusion, bullying is clearly a complex phenomenon. Understanding its multiple causal agents should be an important focus of future work.

Acknowledgements

The authors wish to thank Professor Barry Weinhold, Melissa Mincic, Douglas Asher, and Lisa Barber-Tucci for their help with the study.

References

- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.—Text Revision). Washington, DC: Author.
- Atlas, R. S., & Pepler, D. J. (1998). Observations of bullying in the classroom. *Journal of Educational Research*, 92, 86–99.
- Baldry, A. C., & Farrington, D. P. (2000). Bullies and delinquents: personal characteristics and parental styles. *Journal of Community & Applied Social Psychology*, 10, 17–31.
- Bleiberg, E. (2001). *Treating personality disorders in children and adolescents: A relational approach*. New York: Guilford.
- Coolidge, F. L. (1998). Coolidge personality and neuropsychological inventory for children (CPNI): Manual. Colorado Springs, CO: Author.
- Coolidge, F. L., Aksamit, C. R., & Becker, L. A. (1994). Prediction of recidivism in juvenile offenders: a neurobehavioural investigation. *Indian Journal of Psychological Issues*, 2, 1–6.
- Coolidge, F. L., Reilman, B. J., Becker, L. A., Cass, V. J., Coolidge, R. L., & Stocker, R. (1992). Emotional problems and neuropsychological symptoms in juvenile non-violent offenders. *Journal of Personality and Clinical Studies*, 8, 7–13.
- Coolidge, F. L., Segal, D. L., Stewart, S. E., & Ellett, J. A. C. (2000). Neuropsychological dysfunction in children with borderline personality disorder features: a preliminary investigation. *Journal of Research in Personality*, 34, 554–561.
- Coolidge, F. L., Thede, L. L., & Jang, K. L. (2001). Heritability of personality disorders in childhood. *Journal of Personality Disorder*, 15, 33–40.
- Coolidge, F. L., Thede, L. L., & Jang, K. L. (2004). Are personality disorders psychological manifestations of executive function deficits? Bivariate heritability evidence from a twin study. *Behavior Genetics*, 34, in press.
- Coolidge, F. L., Thede, L. L., Stewart, S. E., & Segal, D. L. (2002). The Coolidge personality and neuropsychological inventory for children (CPNI): preliminary psychometric characteristics. *Behavior Modification*, 26, 550–566.
- Coolidge, F. L., Thede, L. L., & Young, S. E. (2000). Heritability and the comorbidity of attention deficit hyperactivity disorder with behavioral disorders and executive function deficits. *Developmental Neuropsychology*, 17, 273–287.
- Coolidge, F. L., Thede, L. L., & Young, S. E. (2002). The heritability of gender identity disorder in a child and adolescent twin sample. *Behavior Genetics*, 32, 251–257.
- Cowdry, R. W., Pickar, D., & Davies, R. (1985). Symptoms and EEG findings in the borderline syndrome. *International Journal of Psychiatry in Medicine*, 15, 201–210.
- Craig, W. M. (1998). The relationship among bullying, victimization, depression, anxiety, and aggression in elementary school children. *Personality and Individual Differences*, 24, 123–130.
- Espelage, D. L., Bosworth, K., & Simon, T. R. (2000). Examining the social context of bullying behaviors in early adolescence. *Journal of Counseling and Development*, 78, 326–333.
- Gardner, D., Lucas, P. B., & Cowdry, R. W. (1987). Soft sign neurological abnormalities in borderline personality disorder and normal control subjects. *Journal of Nervous and Mental Disease*, 175, 177–180.
- Glover, D., Gough, G., Johnson, M., & Cartwright, N. (2000). Bullying in 25 secondary schools: Incidence, impact, and intervention. *Educational Research*, 42, 141–156.
- Grigsby, J., & Stevens, D. (2000). *The neurodynamics of personality*. New York: Guilford.
- Holm, S. (1979). A simple sequentially rejective multiple test procedure. *Scandinavian Journal of Statistics*, 6, 65–70.
- Kernberg, P. F., Weiner, A. S., & Bardenstein, K. K. (2000). *Personality disorders in children and adolescents*. New York: Basic Books.
- Kumpulainen, K., Rasanen, E., & Henttonen, I. (1999). Children involved in bullying: psychological disturbance and the persistence of involvement. *Child Abuse & Neglect*, 23, 1253–1261.
- Kumpulainen, K., Rasanen, E., Henttonen, I., Almqvist, F., Kresanov, K., Linna, S. L., Piha, J., Puura, K., & Tamminen, Y. (1998). Bullying and psychiatric symptoms among elementary school-age children. *Child Abuse & Neglect*, 22, 705–717.
- Neary, A., & Joseph, S. (1994). Peer victimization and its relationship to self-concept and depression among schoolgirls. *Personality and Individual Differences*, 16, 183–186.

- Olweus, D. (1991). Bully/victim problems among school children: basic facts and effects of a school-based intervention program. In D. Pepler & K. Rubin (Eds.), *The development and treatment of childhood aggression*. Hillsdale, NJ: Erlbaum.
- Quitkin, F., Rifkin, A., & Klein, D. F. (1976). Neurologic soft signs in schizophrenia and character disorders. *Archives of General Psychiatry*, 33, 845–853.
- Shaffer, C. E., Davidson, R. J., & Saron, C. (1985). Frontal and parietal EEG asymmetries in depressed and nondepressed subjects. *Biological Psychiatry*, 18, 753–762.
- Slee, P. T. (1995). Peer victimization and its relationship to feelings of depression among Australian primary school students. *Personality and Individual Differences*, 18, 57–62.
- Spreen, O. (1989). The relationship between learning disability, emotional disorders, and neuropsychology: some results and observations. *Journal of Clinical and Experimental Neuropsychology*, 11, 117–140.
- Torgersen, S., Lygren, S., Oien, P. A., Skre, I., Onstad, S., Edvardsen, J., Tambs, K., & Kringlen, E. (2000). A twin study of personality disorders. *Comprehensive Psychiatry*, 41, 416–425.
- Whitney, I., & Smith, P. K. (1993). A survey of the nature and extent of bullying in junior/middle and secondary schools. *Educational Research*, 35, 3–25.
- Yeudall, L. T., Fromm-Auch, D., & Davies, P. (1982). Neuropsychological impairment of persistent delinquency. *Journal of Nervous and Mental Disease*, 170, 257–265.