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Intermittent Explosive Disorder and aversive parental care

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ABSTRACT

Parental bonding has been shown to have lasting impacts on the psychological development of children. Despite a growing body of research examining trauma as it relates to Intermittent Explosive Disorder (IED), no prior research has examined the relationship between parental bonding and IED. Six hundred fifty eight subjects were studied and categorized into one of three groups: Normal Control (no history of current or lifetime Axis I or Axis II disorder), Psychiatric Control (current and/or lifetime Axis I and/or Axis II disorders without IED), and IED (met current and/or lifetime criteria for IED). Self-reported parental care was assessed using the Parental Bonding Inventory (PBI). PBI Care scores were lowest among IED subjects, which were lower than among Psychiatric Control subjects, which were lower than among Normal Control subjects. PBI Control scores were highest among IED and Psychiatric Control subjects, which were higher than among Normal Control subjects. The diagnostic group differences in PBI Care/PBI Control scores were not impacted by the number of Axis I/II diagnoses. The findings in this study expand the link between childhood trauma exposure, violent behavior, and IED. This is the first report of an association of IED with an aversive childhood parenting environment.

1. Introduction

Intermittent Explosive Disorder (IED) is the DSM-IV clinical description of problematic impulsive aggression, with an estimated prevalence between 5.4% and 6.9% (Coccaro, 2012). Although serotonergic abnormalities have been consistently linked to impulsive aggression, details regarding the mechanism whereby IED develops remain unclear. Data from the National Comorbidity Survey Replication (NCSR) study have provided some clues about the development of IED. In adults with IED, the mean age of onset of IED symptoms is 14 (Kessler et al., 2006). The prevalence of IED in adolescents, according to data from the NCSR Adolescent Supplement, is 7.8% (McLaughlin et al., 2012; Meaney, 2010). These findings highlight the need to examine developmental factors present in childhood and adolescence that play an etiological role in IED. Although genetic factors have an unequivocal role in brain development, early life environment plays an important role in shaping the development of emotional traits relevant to a wide range of psychiatric disorders (reviewed in Meaney (2010)). The experience of traumatic events has previously been found to be associated with abnormal aggressive behavior (Fincham et al., 2009; Silove et al., 2009). To date, a single study has examined the relationship of trauma in childhood with IED specifically. Analysis of data from the NCSR found that a diagnosis of IED was associated with exposure to both childhood trauma (51.28% of persons with IED) and adult trauma (19.88% of persons with IED) (Nickerson et al., 2012). Interpersonal trauma had a stronger relationship to IED than trauma resulting from acts of nature or accidents. The study did not specifically examine aversive parenting practices, although the relationship between IED and childhood interpersonal traumas indicates that parental care needs to be examined in relation to IED. Parental care is protective against parental abuse and neglect, and is associated with opposing neurobiological effects in clinical samples (Lee et al., 2006). A large body of work has found that childhood trauma in the form of abuse and/or neglect represents a risk factor for psychiatric disorders broadly (Hildyard and Wolfe, 2002; Schafer and Fisher, 2011). Furthermore, there is a limited, but growing, body of longitudinal and cross-sectional evidence identifying experienced abuse in childhood as a risk factor contributing to aggression and violence in adulthood. In particular, it may be a significant risk factor for intimate partner violence (reviewed in Gil-González et al. (2008)).

Qualitative and quantitative research on parental bonding resulted in the identification of two opposing factors: 1. Parental warmth *vs.* parental rejection, and 2. Control *vs.* autonomy (Parker and Brown, 1979; Schaefer, 1965). The first factor describes positive evaluation, sharing, expressing of affection, emotional

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support, and fair treatment; it is opposed by ignoring, neglect, and rejection. The second factor describes intrusiveness and parental direction through guilt; it is opposed by encouragement of autonomy and independent thinking. Laxity in discipline, which was part of Schaefer's conceptualization of the second "control" factor, was de-emphasized by Parker, and was subsequently found to be less predictive of developmental problems (Safford et al., 2007).

No study has yet examined the relationship between parental bonding and IED. The present study addresses this gap by comparing scores between IED subjects and control subjects on a validated questionnaire measure of parental bonding, the 25-item Parental Bonding Inventory (PBI) (Parker and Brown, 1979). PBI scores were compared between adults with IED and Normal Controls (without Axis I or Axis II disorders) as well as with Psychiatric Controls (with a non-IED Axis I or Axis II diagnosis). This study had four hypotheses: 1. IED subjects would have lower scores on a measure reflecting their perception of the degree of care and involvement of their parents with the subject, compared with Normal Control and Psychiatric Control subjects. 2. IED subjects would have higher scores on a measure reflecting their perception of the degree of control and overprotection by their parents toward the subject, compared with Normal Control and Psychiatric Control subjects. 3. These differences would be accounted for by differences in aggression and impulsivity but not by any differences in Axis I or II psychopathology or by variability in dimension of personality. 4. Similar findings would be noted for history of self-directed aggression (history of suicide attempt and self-injurious behavior).

2. Methods

2.1. Subjects

Six-hundred-fifty-eight physically healthy subjects participated in this study. All subjects were medically healthy and were systematically evaluated as part of a larger program designed to study correlates of impulsive aggressive and personality disorder related behaviors in human subjects. Subjects were recruited from public service announcements and through newspaper advertisements seeking out individuals who: a) reported psychosocial difficulty related to one or more Axis I and Axis II conditions or, b) had no evidence of psychopathology. All subjects gave informed consent and signed the informed consent document approved by our Committee for the Protection of Human Subjects (IRB). Of this group, 187 subjects had no history of current or lifetime Axis I or Axis II disorder and were designated as the Normal Control group. Two-hundred-six subjects with current and/or lifetime Axis I and/or Axis II disorders were designated as the Psychiatric Control group (by definition, individuals in this group did not meet criteria for a current or lifetime diagnosis of IED). The remaining 265 subjects met current and/or lifetime criteria for IED and were designated as the IED group. No subject met current diagnostic criteria for alcohol or other drug dependence and none had a life history of mania/hypomania, schizophrenia, or delusional, disorder.

2.2. Diagnostic assessment

Axis I and Axis II Personality Disorder diagnoses were made according to DSM-IV criteria (American Psychiatric Association, 2000). The diagnosis of Intermittent Explosive Disorder was made by Research Criteria, which specify: 1) bi-weekly verbal or physical aggression towards other individuals, animals, or property for 1 month; or three episodes or physical aggression within 1 year, 2) the aggression must be disproportionate to provocation or instigating stressors, 3) the aggressive behavior is impulsive and not goal oriented, 4) the aggressive behavior causes concern in the individual, and finally 5) the aggressive behavior is not better accounted for by another mitigating condition such as a mental disorder, substance abuse, or general medical condition. (Coccaro, 2011, 2012). Diagnoses were made using information from: (a) the Structured Clinical Interview for DSM Diagnoses (SCID-I; First, 1997) for Axis I disorders and the Structured Interview for the Diagnosis of DSM Personality Disorder (Pfohl et al., 1997) for Axis II disorders; (b) clinical interview by a research psychiatrist. The research diagnostic interviews were conducted by individuals with a masters, or doctorate, degree in Clinical Psychology. All diagnostic raters went through a rigorous training program that included lectures on DSM diagnoses and rating systems, videos of expert raters conducting SCID/SIDP interviews, and practice interviews and ratings until the rater was deemed reliable with the trainer. This process resulted in good to excellent inter-rater reliabilities (mean kappa of 0.84 ± 0.05; range: 0.79–0.93) across mood, anxiety, substance use, impulse control, and personality disorders. Kappa for the diagnosis of specific PD, and for PD-NOS, was 0.84 and 0.83, respectively. Final diagnoses were assigned by team best-estimate consensus procedures (Klein et al., 1994; Leckman et al., 1982) involving research psychiatrists and clinical psychologists as previously described (Coccaro et al., 2012). This methodology has previously been shown to enhance the accuracy of diagnosis over direct interview alone (Kosten and Rounsaville, 1992).

By definition, none of the Normal Control subjects had a current or lifetime history of any Axis I or II disorder. Of the 206 Psychiatric Control subjects, most (74%) had a lifetime history, of at least one Axis I disorder. Lifetime Axis I disorders were as follows: Any Depressive Mood Disorder ($n=68$); Any Anxiety Disorder ($n=39$); Alcohol or Drug Dependence ($n=44$); Other Impulse Control Disorder ($n=5$); Eating Disorder ($n=9$); Somatoform Disorder ($n=1$); Adjustment Disorder ($n=16$). Seventy-seven (37%) of the Psychiatric Control subjects met DSM-IV criteria for a specific personality disorder as follows: a) Cluster A ($n=19$): Paranoid ($n=15$), Schizoid ($n=7$), Schizotypal ($n=2$); b) Cluster B ($n=41$): Antisocial ($n=7$), Borderline ($n=15$), Histrionic ($n=4$), Narcissistic ($n=19$); c) Cluster C ($n=38$): Avoidant ($n=13$); Dependent ($n=1$), Obsessive–Compulsive ($n=26$). An additional 62 (30%) subjects were diagnosed as Personality Disorder-Not Otherwise Specified (PD-NOS). These subjects met DSM-IV General Diagnostic Criteria for Personality Disorder, had pathological personality traits from a variety of personality disorder categories and had clear evidence of impaired psychosocial functioning [Mean ($\pm$S.D.) Global Assessment of Function (GAF) score$=64.4 \pm 7.7$].

Of the 265 IED subjects, all had a lifetime history, of at least one Axis I disorder. Lifetime Axis I disorders were as follows: Any Depressive Mood Disorder (65%); Any Anxiety Disorder (38%); Alcohol or Drug Dependence Disorders (43%); Other Impulse Control Disorder (9%); Eating Disorder (9%); Somatoform Disorder (2%); Adjustment Disorder (5%). One-hundred-eighty-three (62%) of the IED subjects met DSM-IV criteria for a specific personality disorder as follows: a) Cluster A (26%): Paranoid (25%), Schizoid (1%); b) Cluster B (70%): Antisocial (34%), Borderline (44%), Histrionic (7%), Narcissistic (26%); c) Cluster C (39%): Avoidant (17%); Dependent (2%), Obsessive–Compulsive (26%). An additional 82 (31%) subjects were diagnosed as Personality Disorder-Not Otherwise Specified (PD-NOS). Similar to the corresponding Psychiatric Control subjects with PDNOS, these subjects also had clear evidence of impaired psychosocial functioning [Mean ($\pm$S.D.) GAF score$=59.6 \pm 7.1$].

2.3. Measures of perceived parental care and control

Self-reported parental care, the subjective perception and recollection of quality of parental care received, and the degree of parental control experienced, in childhood, was assessed using the Parental Bonding Inventory (PBI; Parker and Brown, 1979). The PBI is a 25-item self-rating scale, with each item measuring on a 4-point Likert scale qualitative aspects of individual parent (mother and father) behavior. The PBI parental care (PBI Care) subscale score represents a summation of data regarding perceived parental care from the mother and father from one of two dimensions of parental care (care *vs.* neglect). PBI Care describes experienced affection, emotional warmth, empathy and closeness *vs.* emotional coldness, indifference, and neglect. The second PBI dimension, Parental Control, describes intrusiveness and parental direction through guilt *vs.* encouragement of autonomy and independent thinking. The two PBI variables were inversely correlated in this sample ($r=-0.42$, $p<0.001$, $n=658$) suggesting that perceived parental care runs counter to perceived parental control.

2.4. Measures of aggression, impulsivity and relevant measures of personality

Aggression was assessed using the Aggression scale from the Life History of Aggression assessment (Coccaro et al., 1997) and impulsivity was assessed using the Barratt Impulsivity Scale (BIS-11; Patton et al., 1995). LHA Aggression contains five items related to life frequency of temper tantrums, general fighting, specific physical assault, specific property assault, and verbal assault. LHA Aggression has high internal consistency (alpha$=0.87$), excellent inter-rater reliability (ICC$=0.95$), and good test–retest reliability up to 1 year ($r=0.80$; Coccaro et al., 1997). The BIS-11 is a 34 item self-report questionnaire developed to assess impulsivity as a personality trait, taking into account the multi-factorial nature of the construct. Each item is scored on a four-point scale from: 1 ("rarely/never") to 4 ("almost always/always") and items are summed to yield a total impulsivity score. The psychometric properties of the BIS-11 are well documented (Patton et al., 1995). Self-directed aggression was assessed by history of suicidal behavior and self-injurious behavior as assessed during the SCID interviews (First, 1997). General personality and temperament were assessed using the Neuroticism, Psychoticism, and Extraversion scales from the Eysenck Personality Questionnaire (EPQ; Eysenck and Eysenck, 1975) and the Novelty Seeking, Harm Avoidance, and Reward Dependence scales from the Temperament Personality Questionnaire (TPQ; Cloninger, 1994)

2.5. Statistical analysis

Comparisons of between-group variables were performed by X^2, Fisher's Exact Test (FET), analysis of variance/covariance (ANOVA/ANCOVA) and multivariate analysis of variance/covariance (MANOVA/MANCOVA) followed by Tukey HSD post-hoc testing ($p < 0.05$). Correlational analyses included Pearson correlation, partial correlation, and multiple regression. A two-tailed alpha value of 0.05 was used to denote statistical significance for all analyses. Multiple regression ($F(4,653) = 6.77$, $p < 0.001$) revealed that PBI Care scores correlated uniquely, though relatively weakly ($R^2 = 0.03$), with age (part $r = -0.19$, $p < 0.001$) and gender (part $r = -0.07$, $p < 0.09$) but not with race or socioeconomic status score, together accounting for about 3% of variance in Total PBI Care scores. Similar analysis ($F[4,653] = 14.65$, $p < 0.001$) revealed that PBI Control scores correlated uniquely, but modestly ($R^2 = 0.08$), with age (part $r = 0.13$, $p = 0.001$), gender (part $r = 0.16$, $p < 0.001$), race (part $r = 0.19$, $p < 0.001$), and socioeconomic status score, together accounting for about 8% of variance in Total PBI Control scores. Adjusting for the influence of these demographic variables, however, did not affect the results of the analyses. Thus, the results reported below are based on unadjusted PBI Care and Control score data. The number of lifetime Axis I disorder diagnoses was used as a proxy for severity in Axis I psychopathology and the number of Axis II personality disorder diagnoses was used as a proxy of Axis II psychopathology. Both of these variables are positively and significantly correlated with DSM-IV Global Assessment of Function (GAF) score ($r = -0.671$, $p < 001$ and $r = -0.687$, $p < 001$ respectively). Multiple regression analyses ($F(2,655) = 411.40$, $p < 0.001$; $R^2 = 0.56$) revealed that total number of Axis I/II diagnoses correlated uniquely, and strongly ($R^2 = 0.56$), with LHA Aggression (part $r = 0.52$, $p < 0.001$) and BIS Impulsivity (part $r = 0.26$, $p < 0.001$) scores. Accordingly, this variable, reflecting severity of overall psychopathology, was adjusted for LHA Aggression and BIS Impulsivity scores in analyses where the independent effect of overall psychopathology was examined. This variable was adjusted by using the residual of overall psychopathology after multiple regression with total number of Axis I/II diagnoses as dependent variable and LHA Aggression and BIS Impulsivity scores as independent variables. For the same reason, corresponding adjustments were made to the following variables: History of Any Mood Disorder, Any Anxiety Disorder, Any Substance Dependence Disorder, Any Personality Disorder, and for EPQ and TPQ personality variables (so that these variables were free of any overlapping variance with LHA Aggression and BIS Impulsivity). Exploratory analyses examined high *vs.* low PBI Care IED subjects as defined by median split of the IED group based on the mean PBI Care score of the Normal Control subjects on severity of aggression and Axis II comorbidities.

3. Results

3.1. Characteristics of the sample (Table 1)

Normal Control, Psychiatric Control, and IED subjects displayed modest, but statistically significant, differences in the distribution of gender and race. More robust differences were observed in age and in Hollingshead Socio-Economic Class score. Despite these group differences in demographic variables, as stated above, adjusting for variance in these variables did not affect the results reported below. The pattern of DSM-IV lifetime and current Axis I and current Axis II psychiatric comorbidities revealed that IED subjects had the greatest mean number of Axes I and II comorbidities, followed by Psychiatric Control, then Normal Control subjects. The IED group compared with the Psychiatric Control group had significantly higher rates of lifetime alcohol dependence ($p < 0.001$), any lifetime drug abuse disorder ($p < 0.001$), and any drug dependence disorder ($p < 0.001$), but did not differ on lifetime rates of alcohol abuse ($p = 0.33$). This data largely parallels what was seen in comparisons of number Axis I and Axis II conditions across the three groups (Table 1).

3.2. Relationship between aggression, impulsivity, PBI Care, and PBI Control

Both LHA Aggression and BIS Impulsivity scores correlated significantly with PBI Care (Aggression: $r = -0.30$, $p < 0.001$; Impulsivity: $r = -0.31$, $p < 0.001$) and PBI Control (Aggression: $r = 0.19$, $p < 0.001$; Impulsivity: $r = 0.20$, $p < 0.001$), though in opposite directions. Multiple regression revealed that both LHA and BIS variables uniquely, and independently, correlated with PBI Care to the same degree ($F(2,655) = 46.25$, $p < 0.001$, $R^2 = 0.12$; part r for Aggression $= -0.18$, $p < 0.001$; part r for Impulsivity $= -0.20$, $p < 0.001$). The same was true for PBI Control ($F(2,655) = 19.25$, $p < 0.001$, $R^2 = 0.05$; part r for Aggression $= 0.11$, $p = 0.003$; part r for Impulsivity $= 0.14$, $p < 0.001$).

3.3. Total PBI Care and Control scores as a function of diagnostic group

Multivariate ANOVA revealed a significant difference for both PBI Care and PBI Control scores as a function of diagnostic group (Wilks $\lambda = 0.88$, $F(4,1308) = 22.18$, $p < 0.001$) with both PBI scales reaching a high level of statistical significance (Care: $F[2,655] = 40.90$, $p < 0.001$; Control: $F(2,655) = 19.55$, $p < 0.001$). Post-hoc testing demonstrated that PBI Care scores were lowest among IED subjects, and significantly lower than among Psychiatric Control subjects, whose scores were significantly lower than among Normal Control subjects (see Table 2). PBI Control scores were highest among both IED and Psychiatric Control subjects which were both higher than those scores among Normal Control subjects (see Table 3). Adding the total number of Axis I/II diagnoses to the ANOVA model did not meaningfully affect these results (PBI Care: IED: 20.48 ± 7.67 *vs.* Psychiatric Control: 22.71 ± 7.49 *vs.* Normal Control: 26.85 ± 7.72; PBI Control: IED: 25.74 ± 7.20 *vs.* Psychiatric Control: 15.01 ± 6.97 *vs.* Normal Control: 12.04 ± 7.19). Adding the variables of Any Mood Disorder, Any Anxiety Disorder, Any Substance Dependence Disorder, or Any Personality Disorder to the models, also, did not affect the results, nor were the results affected by adding EPQ or TPQ personality variables to the model. In contrast, adding both LHA Aggression and BIS Impulsivity scores to the model eliminated the difference in PBI Care scores between IED and Psychiatric Control subjects (IED: 21.87 ± 10.06 *vs.* Psychiatric Control: 22.31 ± 7.84) but not the difference between IED/Psychiatric Control and Normal Control subjects (Normal Control: 25.31 ± 9.20). Adding LHA Aggression and BIS Impulsivity had little impact on PBI Control scores (IED: 15.05 ± 9.51 *vs.* Psychiatric Control: 15.22 ± 7.39 *vs.* Normal Control: 12.79 ± 8.68).

3.4. PBI subscale scores as a function of PBI Mother/Father and diagnostic group

PBI scores for Mother were significantly higher than those for Father for both PBI Care and PBI Control, with greater group differences for PBI Care compared with PBI Control (PBI Care: paired $t_{595} = 9.18$, $p < 0.001$, effect size: $z = 0.38$; PBI Control: paired $t_{594} = 4.47$, $p < 0.001$, effect size: $z = 0.18$). Subsequent MANOVA revealed a significant difference as a function of diagnostic group (Wilks $\lambda = 0.86$, $F(8,1178) = 11.85$, $p < 0.001$) with each PBI subscale score reaching a high level of statistical significance (Care for Mother: $F(2,592) = 25.69$, $p < 0.001$; Care for Father: $F(2,592) = 27.93$, $p < 0.001$; Control for Mother: $F[2,592] = 12.78$, $p < 0.001$; Control for Father: $F[2,592] = 18.36$, $p < 0.001$). Post-hoc testing demonstrated that the PBI Care subscales for Mother were lowest among IED subjects, and significantly lower than among Psychiatric Control subjects, whose scores were significantly lower than among Normal Control subjects (see Table 2). For the remaining PBI subscales, scores were lowest (or highest) among IED and Psychiatric Control subjects whose scores were significantly different compared with scores among Normal Control subjects (PBI Care Father: IED: see Table 2; PBI Control Father: see Table 3; PBI Control Mother: see Table 3).

Table 1
Demographic data for sample.

	Normal Controls ($N=187$)	Psychiatric Controls ($N=206$)	Intermittent Explosive Disorder ($N=265$)	p value
Age (years)	30.3 ± 8.4	33.3 ± 9.6	37.0 ± 10.0	<0.001[1]
Gender (M/F)	105/82	134/72	143/122	<0.050[2]
Race (white/AA/other)	117/45/25	116/76/14	140/96/29	$=0.015$[2]
SES score	39.0 ± 14.0	33.8 ± 13.8	37.2 ± 13.0	<0.001[1]
No. of current Axis I Dx	0	0.44 ± 0.8	2.25 ± 1.6	<0.001[1]
No. of lifetime Axis I Dx	0	1.7 ± 1.8	5.4 ± 2.5	<0.001[1]
No. of Axis II Dx	0	0.9 ± 1.5	1.65 ± 1.6	<0.001[1]

[1] Significance after ANOVA. Post-hoc (THD: $p<0.05$) for age: IED > PC > NC; for SES score: NC=IED > PC; for current Axis I: IED > PC > NC; for lifetime Axis I: IED > PC > NC; for Axis II: IED > PC > NC.
[2] Significance after X^2 test.

Table 2
PBI Care/involvement mean scores.

	Normal Controls ($N=187$)	Psychiatric Controls ($N=206$)	Intermittent Explosive Disorder ($N=265$)	p value
Parental	26.89 ± 7.48	22.71 ± 7.48	20.44 ± 7.49	<0.001[1]
Maternal	28.48 ± 9.15	24.42 ± 9.13	22.23 ± 9.13	<0.001[1]
Paternal	25.02 ± 9.45	20.43 ± 9.40	18.33 ± 9.41	$=0.001$[1]

[1] Significance after MANOVA.

3.5. Relationship between self-directed aggression and total PBI scores in Psychiatric Control/IED subjects

Adding lifetime history of suicide attempt (SA) to the MANOVA model revealed a main effect for history of SA (Wilks $\lambda=0.97$, $F(1,467)=8.40$, $p<0.001$; PBI Care: SA+: 18.67 ± 7.87 *vs.* SA−: 22.01 ± 7.80; PBI Control: SA+: 18.50 ± 7.25 *vs.* SA−: 15.00 ± 7.17) and for diagnostic group ($F(1,467)=3.77$, $p<0.025$) but not for the interaction of the two. Only a marginal effect of history of SIB was observed on Total PBI Care or Control scores when the SIB variable was added to the model (Wilks $\lambda=0.99$, $F(1,467)=2.59$, $p=0.076$), despite the fact that 40% (25 of 63 subjects) of the SA+ group also had a history of SIB. These results were not meaningfully affected by adding the total number of Axis I/II diagnoses to the model, by examining the effect of history of Any Mood Disorder, Any Anxiety Disorder, Substance Dependence Disorder, or Any Personality Disorder, or by adding EPQ or TPQ personality variables to the model. However, unlike the diagnostic group differences, noted above, adding LHA Aggression and BIS Impulsivity scores to the model involving the SA variable did not affect Total CTQ scores as a function of history of SA (PBI Care: SA+: 19.31 ± 7.87 *vs.* SA−: 21.80 ± 7.80; PBI Control: SA+: 18.11 ± 7.88 *vs.* SA−: 15.12 ± 7.25).

3.6. Relationship between self-directed aggression and PBI subscale scores

MANOVA analysis revealed only an overall trend for statistical significance for the PBI subscales as a function of lifetime history of SA (Wilks $\lambda=0.98$, $F(4,422)=2.50$. $p=0.06$). Stepwise multiple regression, with SA status as the dependent variable confirmed that, while all PBI subscales (together) were related to lifetime history of SA ($F(2,422)=2.50$, $p<0.05$, $R^2=0.01$), no subscale was uniquely, or independently, associated with lifetime history of SA.

Table 3
PBI Control/overprotection mean scores.

	Normal Controls ($N=187$)	Psychiatric Controls ($N=206$)	Intermittent Explosive Disorder ($N=265$)	p value
Parental	11.83 ± 6.97	14.98 ± 6.97	15.91 ± 6.98	<0.001[1]
Maternal	12.29 ± 8.27	15.19 ± 8.25	16.23 ± 8.25	<0.001[1]
Paternal	10.29 ± 8.23	14.84 ± 8.20	14.37 ± 8.21	<0.001[1]

[1] Significance after MANOVA.

3.7. Effect of severity of overall psychopathology on PBI scores

While severity of overall psychopathology, expressed as total number of Axis I/II diagnoses did not influence group differences in PBI scores, PBI scores did correlate with raw scores for severity of general psychopathology (PBI Care: All Subjects: $r=-0.34$, $n=658$, $p<0.001$; Psychiatric Control/IED Subjects: $r=-0.22$, $n=472$, $p<0.001$; PBI Control: All Subjects: $r=0.26$, $n=658$, $p<0.001$; Psychiatric Control/IED Subjects: $r=0.17$, $n=472$, $p<0.001$). Removal of shared variance with LHA Aggression and BIS Impulsivity Scores, however, resulted in weaker correlations with PBI scores (PBI Care: All Subjects: $r=-0.12$, $n=658$, $p<0.001$; Psychiatric Control/IED Subjects: $r=-0.07$, $n=471$, $p=0.11$; PBI Control: All Subjects: $r=0.13$, $n=658$, $p<0.001$; Psychiatric Control/IED Subjects: $r=0.10$, $n=471$, $p<0.05$). Multiple regression revealed that severity of general psychopathology was uniquely, and independently, related to PBI Care and PBI Control scores. Analysis of the raw total of number of Axis I/II diagnoses revealed a stronger relationship with PBI Care (All Subjects: part $r=0.25$, $p<0.001$; Psychiatric Control/IED Subjects: part $r=-0.16$, $p<0.001$), compared with PBI Control (All Subjects: part $r=0.13$, $p<0.001$; Psychiatric Control/IED Subjects: part $r=0.10$, $p=0.033$), scores. However, analyses of the adjusted scores (*i.e.*, residual scores after removal of variance shared with LHA Aggression and BIS Impulsivity) revealed a nearly equal relationship with both PBI scale scores of marginal (or of no) statistical significance (PBI Care: All Subjects: part $r=-0.07$, $p=0.063$, Psychiatric Control/IED Subjects: part $r=-0.04$, $p=0.376$; PBI Control: All Subjects: part $r=0.09$, $p=0.021$, Psychiatric Control/IED: part $r=0.07$, $p=0.112$).

3.8. Exploratory data regarding IED subtypes

Median split of the IED group based on mean PBI Care score (26) of the Normal Control population created a low PBI IED group ($n=179$) and a high PBI IED group ($n=69$). Independent samples t-tests comparing the two groups on LHA subscale scores revealed that mean scores were higher for most if not all of the LHA subscale scores in the low PBI group. LHA subscales that crossed the threshold for statistical significance (uncorrected $p<0.05$) included LHA Difficulties with Authority ($t(1, 245)=2.547$, $p=0.01$) and LHA Fighting ($t(1, 246)=2.457$, $p=0.02$), as well as LHA Total Score ($t(1, 245)=2.514$, $p=0.01$). Further comparisons between the low PBI IED group and high PBI IED group using X^2 (2-sided) tests revealed that the low PBI IED group had higher rates of Obsessive Compulsive Personality Disorder ($X^2=4.483$, $p=0.032$) and Antisocial Personality Disorder ($X^2=5.747$, $p=0.02$).

4. Discussion

In this study reports of parental care were related to dimensional measures of aggression as well as a categorical diagnosis of the aggression-related psychiatric disorder, IED. In the entire sample, parental care (PBI Care) was inversely correlated with aggression (LHA-Aggression) and impulsivity (BIS Impulsivity) while parental control (PBI Control) was positively correlated with aggression and impulsivity. With regards to the categorical findings, IED was associated with: 1. lower PBI Care score relative to both Normal Controls and Psychiatric Controls and 2. higher PBI Control scores relative to Normal Controls, but not significantly higher than Psychiatric Controls. These results were consistent with the overall hypothesis of an association between IED and aversive parenting. This is the first report of an association of IED with an aversive childhood parenting environment. The association is shared with other psychopathologies, although IED may be more specifically related to low levels of parental care and warmth.

Some evidence was found for a unique relationship between IED and maternal care. The PBI subscale Maternal Care showed a similar pattern as PBI Care, with IED associated with lower Maternal Care than both control groups. For Paternal Care, IED subjects had lower scores than Normal Controls, but were comparable to Psychiatric Controls. For Maternal Control and Paternal Control, IED subjects had higher scores than normal subjects, but were comparable to Psychiatric Controls. This pattern of findings, if replicated, suggests that IED may be specifically associated with low maternal warmth.

Although no previous research has addressed parental bonding in IED, the findings contribute to a growing body of work that links trauma exposure to violent behavior as well as the more specific diagnosis of IED. Exposure to violence and trauma accounts for 24–50% of the variance in violent behavior in children (Singer et al., 1999; Song et al., 1998). However, given that not all violence is caused by IED; it is of interest that two studies have linked trauma exposure to IED. The first study, conducted in 4351 South African adults, found that exposure to multiple traumas was associated with IED (Fincham et al., 2009). Childhood trauma, in the form of parental abuse and/or neglect, was not specifically assessed in this study. The second study, based on data from the NCSR, examined 4844 trauma-exposed and 731 non trauma-exposed adults and replicated the association of IED with trauma exposure (Nickerson et al., 2012) but additionally found that the strongest associations between trauma and IED were with childhood, interpersonal trauma. The observational, cross-sectional design of this study is unable to conclusively prove that aversive parenting plays a causal role in the development of IED. An alternate possibility is that the association is caused by response bias, due to the subjective nature of the PBI. Aversive emotional states have been demonstrated to alter the perception and experience of emotion related information (Becker and Leinenger, 2011; Koster et al., 2010). Other non-causal possibilities could include correlated Gene $\times$ Environment effects. Nevertheless, the finding that aversive parenting is correlated with trait-like dimensions of aggression and impulsivity suggests that aversive parenting may lead to IED via altered neurobiology (reviewed in Pedersen (2004)). If so, it is likely that etiological pathways may be complex and bidirectional, as illustrated by a recent report that childhood emotional neglect moderates the relationship between negative emotional reactivity to social scenarios and adult aggression (Chen et al., 2012). Given that approximately 9% of variance in LHA score was driven by measures of experienced parental care, this means other factors are important in the etiology of aggression. Previous work suggest that aggression is highly heritable (Burt and Klump, 2012; Yeh et al., 2010), although the precise genetic factors underlying human aggression have not yet be convincingly identified.

Subsequent exploratory analyses revealed that suicide attempt history is associated with lower parental care and warmth and increased parental control. IED was also associated with increased suicide attempt rate, above and beyond psychopathology. These findings add to work persuasively linking suicide with aggressive behavior (reviewed in Gvion and Apter (2011)) and aversive parenting (Rhodes et al., 2012).

This is the first report of an association of IED with decreased PBI Care and increased PBI Control scores. It adds to a growing literature that links IED to risk factors in early life. The credibility of these links is strengthened by recent findings of a relatively early onset of symptoms of the disorder (Kessler et al., 2006; McLaughlin et al., 2012). Limitations of the study include retrospective reporting of parental care, lack of objective confirmation of these reports, and the use of a single measure of experienced parental care, the PBI. It is also likely that other factors, such as genetic factors or other environmental factors, may moderate or mediate the relationship between parental care and impulsive aggression.

While this study attempts to illuminate the connection between parental bonding and the later development of IED, further research is warranted exploring the complex impact of the parent-child relationship on the development of IED. Understanding the causes of IED may someday help to improve therapeutic approaches to the disorder by revealing the underlying biological and psychological mechanisms of impulsive aggressive behavior.

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