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Prior Exposure to Interpersonal Violence and Long-term Treatment Response for Boys with a Disruptive Behavior Disorder

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Abstract

Interpersonal violence (IPV) is common in children with a disruptive behavior disorder (DBD) and increases the risk for greater DBD symptom severity, callous-unemotional (CU) traits, and neuroendocrine disruption. Thus, IPV may make it difficult to change symptom trajectories for families receiving DBD interventions given these relationships. The current study examined whether IPV prior to receiving treatment for a DBD predicted trajectories of a variety of associated outcomes, specifically DBD symptoms, CU traits, and cortisol concentrations. Boys with a DBD diagnosis ($N = 66$; age range = 6–11 years; 54.5% of whom experienced IPV prior to treatment) of either oppositional defiant disorder or conduct disorder participated in a randomized clinical trial and were assessed 3 years following treatment. Multilevel modeling demonstrated that prior IPV predicted smaller rates of change in DBD symptoms, CU traits, and cortisol trajectories, indicating less benefit from intervention. The effect size magnitudes of IPV were large for each outcome ($d = 0.88 - 1.07$). These results suggest that IPV is a predictor of the long-term treatment response for boys with a DBD. Including trauma-focused components into existing DBD interventions may be worth testing to improve treatment effectiveness for boys with a prior history of IPV.

Disruptive behavior disorders (DBDs), specifically oppositional defiant disorder (ODD) and conduct disorder (CD), are among the most prevalent psychiatric disorders in childhood and adolescence (Roberts, Roberts, & Xing, 2007). There are 16 well-established behavioral interventions for DBDs (Eyberg, Nelson, & Boggs, 2008) demonstrating clear, short-term benefit for children and their families (Comer, Chow, Chan, Cooper-Vince, & Wilson, 2013). The long-term benefit of these interventions, however, remains questionable with some

studies reporting a maintenance of treatment gains (Reid, Webster-Stratton, & Hammond, 2003) and others reporting declines (Hemphill & Littlefield, 2001) 1 to 2 years after treatment. Thirty-three to 53% of children will meet diagnostic criteria for a DBD 3 to 5 years after treatment (Drugli, Larsson, Fossum, & Mørch, 2010; Kolko et al., 2009), suggesting that some children benefit from treatment immediately and over long-term follow-up whereas others remain at or return to pretreatment levels of functioning. Identifying predictors of this long-term variability holds considerable promise as it can identify those children at greatest risk for nonresponse with the eventual hope of improving treatment effectiveness.

Research on the predictors of treatment response to DBD interventions has identified several factors associated with treatment outcomes, such as the child's sex and the family's socioeconomic status (Drugli et al., 2010; Reyno & McGrath, 2006). These factors or their consequences, however, fail to specify relevant, modifiable clinical targets that can be used during the course of a DBD intervention to improve long-term response. Factors that identify children at greatest risk for nonresponse to treatment while also informing clinicians on how to augment existing interventions serve an important translational need that can ultimately improve the long-term effectiveness of DBD interventions. Exposure to interpersonal violence (IPV), specifically physical assault, sexual assault, or witnessing community or family violence, may be one such factor as it affects as many as 60% of children diagnosed with a DBD (Briggs-Gowan et al., 2010; Ford et al., 1999) and is related to the most direct measures of treatment response for DBD interventions, namely DBD symptoms and callous-unemotional (CU) traits. Studies have demonstrated that IPV is associated with a higher prevalence (Ford, Gagnon, Connor, & Pearson, 2011; Kolko, Dorn, Bukstein, & Burke, 2008) and more persistent course (Stouthamer-Loeber, Loeber, Homish, & Wei, 2001) of DBD symptoms with recent research demonstrating a dose-response relationship where more severe types of IPV are linked to greater DBD symptom severity (Milot, Ethier, St-Laurent, & Provost, 2010). IPV also increases the risk for CU traits, a stable pattern of antisocial behavior present in the most severe cases of DBD's (Frick & White, 2008), which are now used as a clinical specifier for the course and treatment of CD in the Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition (DSM-5) (American Psychiatric Association, 2013). Exposure to IPV in childhood increases the risk for CU traits even among adjudicated and incarcerated youths (Kimonis, Frick, Munoz, & Aucoin, 2008; Krischer & Sevecke, 2008) while predicting a greater severity of CU traits that extends well into young adulthood (Weiler & Widom, 1996). Given the increased risk between IPV and the longitudinal course of these outcomes, exposure to IPV before treatment may make it more difficult to change symptom trajectories for children and families receiving a traditional intervention for DBD's. Such exposure may predict variability in the long-term response to DBD interventions and identify trauma exposure, specifically IPV, as an important treatment target for those children with a DBD.

Research on the mechanisms by which IPV leads to increased DBD symptoms or CU traits remains speculative. One area has examined how early adversity affects neuroendocrine processes, particularly the hypothalamus-pituitary-adrenal (HPA) axis, that may increase the risk of engagement in disruptive and antisocial behaviors (van Goozen, Fairchild, Snoek, & Harold, 2007). Research on resting cortisol concentrations falling within a non-clinical range

of physiologic functioning indicates an attenuated HPA axis pattern in which children exposed to IPV initially display a hypercortisol profile following trauma that ultimately results in a hypocortisol profile when compared to controls and tracked longitudinally (Trickett, Noll, Susman, Shenk, & Putnam, 2010). Attenuated cortisol profiles are a central component in etiologic models of early adversity and DBDs (Susman, 2006) because of their consistent association with the hallmark features of these disorders, specifically DBD symptoms (Alink et al., 2008) and CU traits (Stadler et al., 2011). Once established, disruptive and antisocial behaviors can even lead to or maintain hypocortisol profiles (Ruttle et al., 2011), potentially creating a bidirectional process of engagement in these behaviors over an extended period of time. Thus, IPV may have predictive utility when examining relevant longitudinal processes and outcomes, such as attenuated HPA axis activity and increased DBD symptoms and CU traits, associated with the development and course of DBD's as well as the effectiveness of existing DBD interventions.

The current study tested whether prior exposure to IPV predicted variability in treatment response across several central outcomes using data from a randomized clinical trial on the treatment of DBDs: DBD symptoms, CU traits, and cortisol concentrations. The purpose of the current study is not to evaluate the effectiveness of the two interventions delivered in the trial, as previous research has already found these treatments provide equal benefit (Kolko et al., 2009). Instead, this paper focuses on whether IPV predicted overall response to these two, similar treatments. A specific focus on boys with a DBD was made because males are more likely to be diagnosed with a DBD (Roberts et al., 2007) and because of noted gender differences when examining long-term outcomes in this population (Dorn, Kolko, Shenk, Susman, & Bukstein, 2011). Because IPV is related to increased severity of DBD symptoms, CU traits and an attenuation of the HPA axis when followed longitudinally, the primary aim of the present paper was to evaluate whether exposure to IPV prior to treatment would predict differential rates of treatment response, specifically smaller rates of change in DBD symptoms, CU traits, and cortisol concentrations, when compared to those boys without a prior history of IPV.

Method

Participants

Families were recruited through program sites affiliated with the University of Pittsburgh Medical Center. Two multiple-gate screening phases were used to determine the sample. First, a clinic screen was conducted with families by phone or interview to obtain information regarding the child's psychiatric diagnosis, behavioral problems, and treatment needs. Second, a formal diagnostic assessment was conducted to determine the presence of DBDs. Inclusion criteria were: (a) children aged 6-11 years, (b) residence with at least one parent or guardian, (c) intellectual level no more than two standard deviations below age norms, and (d) an ODD or CD diagnosis. Exclusion criteria were: (a) concurrent individual or family participation in a treatment program for a DBD, (b) current psychosis, bipolar disorder, or major depressive disorder marked by significant vegetative signs, substance abuse, or eating disorder, or (c) suicidality with a plan or homicidality.

The total number of boys randomized to treatment was 118. Funding for the collection of saliva samples was secured 1 year after the initiation of the clinical trial, thereby excluding 49 boys from the current analyses who completed their pretreatment evaluation without providing saliva samples. Three additional participants were unwilling or unable to provide saliva samples and were also excluded, resulting in a total available sample size of 66. Mean age for the current sample at entry into the study was 8.98 ($SD = 1.71$) years, with the average participant coming from a low to moderate socioeconomic background ($M = 36.50$, $SD = 11.74$). The mean IQ was 99.29 ($SD = 13.33$) with 66.7% of the sample coming from single-parent homes. The racial demographic, as reported by the parent, was comprised of Caucasian (45.5%), African-American (45.5%), Hispanic (1.5%), and Bi-racial (7.6%) children. A total of 80.3% of the sample received a diagnosis of ODD with 19.7% diagnosed with CD.

Procedure

All procedures were approved by the University of Pittsburgh Institutional Review Board. Parental consent and child assent were obtained after reviewing all procedures with the family and before proceeding with any part of the study. Families participated in one of two modular-based, behavioral interventions for DBDs: one delivered in a community-based format (COMM) and the other in a clinic-based format (CLIN). COMM and CLIN treatments were similar in content and techniques used to achieve a family's goals for treating DBDs. The treatments differed, however, in the ecological contexts in which they were delivered with the COMM condition receiving treatment in the family's home, school, and/or community settings and with the CLIN condition receiving outpatient treatment in a traditional clinic setting. The majority of services provided to children in both conditions were parent management training, cognitive-behavioral therapy, and family therapy and the average length of treatment ranged from 20-24 weeks.

Trained research assistants conducted treatment evaluations with a parent/guardian and child at several time-points: pre-treatment, post-treatment, and 6-, 12-, 24-, and 36-month follow-up. Saliva samples and CU traits were obtained at all six treatment evaluations, whereas DBD symptoms were assessed at only five evaluations (DBD symptoms were not assessed at the 6-month follow-up). The procedure for collecting saliva samples was integrated into the ongoing clinical trial evaluations. Participants were instructed not to brush their teeth or eat 2 hours before each collection and to rinse their mouth with water prior to passively drooling into collection tubes. Saliva samples were collected at two different times during each treatment evaluation. Sample 1 was collected 15 minutes after arrival to the evaluation. The average time of day for collecting Sample 1 was 1:07 pm ($SD = 2.88$ hours). Participants then completed a semi-structured diagnostic interview assessing DBD symptoms, which lasted on average 53.06 minutes ($SD = 20.63$ minutes). Sample 2 was collected immediately following completion of the diagnostic interview except at the 6-month follow-up evaluation. The average time of day that Sample 1 was collected at the 6-month evaluation was 1:53 pm ($SD = 2.88$ hours), with Sample 2 collected on average 50.02 minutes ($SD = 18.03$ minutes) later. Participants were given \$10 for completing an evaluation with each child and parent receiving the benefit of treatment services at no cost. All participants were given an additional \$10 for providing saliva samples.

Measures

The Traumatic Events Screening Inventory for Children (TESI-C; Ford & Rogers, 1997) is a 15-item clinician-administered interview conducted with the child to determine whether he or she has ever experienced a potentially traumatic event, such as IPV. The TESI-C has moderate to good interrater reliability across different events ($\kappa = .50$ – 1.00 ; Ford & Rogers, 1997) with noted criterion and convergent validity (Daviss et al., 2000). The current study administered the four items of the TESI-C assessing IPV: (a) sexual assault, (b) physical assault, (c) witnessing domestic or family violence, and (d) witnessing community violence. The pre-treatment evaluation determined whether a child experienced IPV at any point prior in their lifetime. Answers to the TESI-C items resulted in assignment to a dummy coded variable indicating prior IPV exposure (*Yes* = 1, *No* = 0). See Table 1 for study-related information based on prior IPV exposure. These same four TESI-C items were administered at each subsequent treatment evaluation and assessed exposure to IPV throughout the 3-year follow-up.

The Schedule for Affective Disorders and Schizophrenia for School-Aged Children for DSM-IV, Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997) diagnostic interview is a well-established instrument with known reliability and validity. Interviews were conducted with the child and parent together by trained, Master's-level clinicians. Diagnoses were made in consultation with the study psychiatrist. Inter-rater reliabilities obtained from the clinical trial were moderate to high for ODD ($\kappa = .79$) and CD ($\kappa = .74$). The number of ODD and CD symptoms endorsed at each treatment evaluation was summed to create a composite index of DBD symptoms that was used in subsequent analyses.

The Antisocial Process Screening Device (ASPD; Frick & Hare, 2001) is a 20-item screening measure assessing three domains of antisocial behavior in children and adolescents, impulsivity, narcissism, and CU traits, with demonstrated construct and predictive validity (Frick & White, 2008). Each item is rated on a dimensional scale ranging from 0 = *not at all true* to 2 = *definitely true*. The teacher-report version of the ASPD was administered to track long-term changes in CU traits in response to treatment. The CU subscale consists of six items and the internal consistency of the ASPD-CU subscale in the current sample was $\alpha = .79$. Total scores on the ASPD-CU subscale were used in analysis.

All saliva was stored at -80°C until assayed in duplicate using a highly sensitive enzyme immunoassay at Salimetrics (State College, PA). The test has a lower limit of sensitivity of $<.003\text{ }\mu\text{g/dL}$ and average intra- and inter-assay coefficients of variation ranging from 3.4% to 3.7% and 3.8% to 6.4%, respectively. Saliva was assayed to establish individual cortisol concentrations from Samples 1 and 2 that were then averaged to derive a mean value of cortisol used in statistical analysis.

Information on current psychotropic medication use was collected from caregivers at each treatment evaluation and categorized into major groups (e.g., stimulants, antidepressants) by a nurse practitioner. Use of steroid medications (inhaled, oral, and topical) within the past two weeks was also assessed at each evaluation. Participants reporting steroid medication use at the time of the treatment evaluation had their data for that particular evaluation

omitted from analysis. Socioeconomic status (SES) was measured with the widely-used Hollingshead scale (Hollingshead, 1975).

Data Analysis

Multilevel modeling assessed changes in the repeated measurements of DBD symptoms, CU traits, and cortisol (Level 1 variables) that were nested within study participants (Level 2). Time was coded in the following manner consistent with the time intervals between treatment evaluations: Pre-treatment = 0; Post-treatment = 0.50, 6-month Follow-up = 1.00, 12-month Follow-up = 1.50, 24-month Follow-up = 2.50, and 36-month Follow-up = 3.50. Trend parameters (e.g., linear, quadratic) assessed average within-subject change in DBD symptoms, CU traits, and cortisol in unconditional models. The quadratic trend failed to achieve statistical significance across each outcome and therefore the linear trend was retained in all analyses. Prior IPV (Yes = 1; No = 0) was entered as a Level 2 predictor of linear growth across all outcomes throughout the 36-month follow-up period. The model for cortisol added a main effect for the time of day in which the first cortisol sample was collected, along with its interaction with time, to control for diurnal variation in cortisol estimates due to sampling time variability. Treatment condition (COMM = 1; CLIN = 0), along with its interaction with time, were also entered as statistical covariates into each multilevel model to adjust for any potential confounds with treatment group assignment. Missing data were addressed through maximum-likelihood estimation under the missing at random assumption. Effect size estimates (d) for multilevel models are reported for each outcome (Feingold, 2009).

Results

At pre-treatment, 4.5% ($n = 3$) of the sample reported prior sexual assault, 34.8% ($n = 23$) reported prior physical assault, 21.2% ($n = 14$) reported prior community violence exposure, and 12.1% ($n = 8$) reported prior exposure to domestic violence. Thirty participants experienced no prior IPV, 25 experienced one event, 10 experienced two events, and 1 participant experienced three events. Multivariate analysis of variance and chi-square tests examined differences in demographic variables across the prior IPV ($n = 36$) and no prior IPV ($n = 30$) groups to identify potential confounders. Age, $F(1, 58) = 0.22$, *ns*; SES, $F(1, 58) = 0.10$, *ns*; IQ, $F(1, 58) = 1.20$, *ns*; DBD symptom severity, $F(1, 58) = 0.05$, *ns*; and CU traits at pre-treatment, $F(1, 58) = 0.01$, *ns*; along with total number of treatment sessions received, $F(1, 58) = 0.03$, *ns*; did not differ significantly between IPV groups. Rates of comorbid attention deficit-hyperactivity disorder were high in both the prior IPV (69.4%) and no prior IPV groups (76.7%) but did not differ significantly between groups, $\chi^2(1, N = 66) = 0.43$, *ns*. The number of single-parent homes, $\chi^2(1, N = 66) = 2.48$, *ns*; as well as rates of ODD, $\chi^2(1, N = 66) = 1.41$, *ns*; and CD, $\chi^2(1, N = 66) = 1.41$, *ns*; also did not differ significantly between IPV groups. Use of psychotropic medication neither differed between IPV groups at pre-treatment, $\chi^2(1, N = 66) = 0.34$, *ns*, nor was IPV group membership associated with psychotropic medication use throughout follow-up, $r = -.07$, *ns*. Minority status (Caucasian = 0, all other racial groups = 1) differed significantly, however, between IPV groups at pre-treatment, $\chi^2(1, N = 66) = 7.09$, $p = .008$, indicating that minority groups experienced significantly higher rates of IPV prior to the trial. Minority status was therefore

included as a covariate in all subsequent statistical modeling. All outcome variables were screened to determine the percent of missing data throughout the study. Over the course of the 3-year follow-up, the total amount of missing data for DBD symptoms, CU traits, and cortisol was 3.9%, 17.4%, and 13.9%, respectively. Outlying values (1.0%) of cortisol were winsorized (Dixon, 1960) to ± 3 standard deviations of the mean to minimize bias resulting from extreme values.

It was expected that prior IPV would predict a smaller rate of change in DBD symptoms over time when compared to those without prior IPV. Results demonstrated that prior IPV did predict a smaller rate of change in the severity of DBD symptoms throughout the 3-year follow-up after controlling for minority status and treatment condition, prior IPV $\times$ Time: $\beta = .65$, $SE = .28$, $p = .023$, $d = 1.07$. While both IPV groups experienced reductions in DBD symptoms over time, the average rate of decline in DBD symptoms was of a significantly weaker magnitude for those boys with an IPV history. The difference in the rates of decline for DBD symptoms resulted in a large effect-size difference between the prior IPV groups (see Figure 1).

It was hypothesized that prior IPV would predict a smaller rate of change in CU traits over time when compared to those boys without prior IPV. After controlling for minority status and treatment condition, model results demonstrated that prior IPV predicted a smaller rate of change in CU traits throughout follow-up, prior IPV $\times$ Time: $\beta = .61$, $SE = .29$, $p = .042$, $d = 0.88$. Unlike DBD symptoms however, CU traits remained stable over follow-up for those boys with a prior IPV history while those without prior IPV experienced a decline (see Figure 1). This resulted in a large effect size difference between prior IPV groups by the final study assessment.

It was expected that prior IPV would predict a smaller rate of change, or attenuation, of HPA axis functioning characterized by higher initial and lower subsequent cortisol concentrations. A significant main effect was observed for prior IPV after controlling for minority status, treatment condition and saliva sampling time, $\beta = .03$, $SE = .02$, $p = .049$, indicating that prior IPV was associated with significantly higher concentrations of cortisol at the initial study assessment. The interaction of prior IPV over time was also significant, prior IPV $\times$ Time: $\beta = -.02$, $SE = .01$, $p = .049$, $d = 0.88$, demonstrating that prior IPV was associated with significantly lower concentrations of cortisol by the last study assessment that resulted in a large effect size difference between groups (Figure 1).

Of note, the TESI-C also measured Subsequent IPV (Yes = 1; No = 0) that occurred in the time between each follow-up visit. Subsequent IPV was added to all prior multilevel models as both a main effect and as an interaction with time when predicting trajectories of each outcome. Model results for all three outcomes produced non-significant parameter estimates for both main (p 's = .23 – .40) and interaction (p 's = .33 – .78) terms without any significant improvement in -2 log likelihood estimates of model fit.

Discussion

This study is the first to examine whether prior exposure to IPV predicted variability in the long-term treatment response for DBDs. The results from this study extend current research in several important ways with implications for the assessment and treatment of boys with a DBD. First, a majority of the sample experienced IPV before receiving treatment. This rate is consistent with prior clinical research (Ford et al., 1999) and is considerably higher than rates of IPV found in community samples (McDonald, Jouriles, Ramisetty-Mikler, Caetano, & Green, 2006). This suggests that IPV is common to DBD populations, especially boys, and is critical to assess prior to beginning treatment. Assessment of IPV is particularly relevant for those boys with a DBD who are of diverse racial and ethnic origins, as minority status was significantly and positively related to IPV exposure before treatment. Given the methods used in this study, IPV can be assessed efficiently without significant burden to practitioners or patients at intake so that all boys, especially those from minority groups, can be screened for IPV to promote access to appropriate treatment services.

Second, prior exposure to IPV predicted a worse response to treatment as demonstrated across multiple outcomes using a variety of methods and informants. Disruptive and antisocial behaviors provide the most obvious and direct measures of treatment response for DBD interventions. Overall, boys in this study experienced reductions in DBD symptoms over time. This is consistent with previous research (Comer et al., 2013) and suggests that the behavioral interventions delivered in the trial were helpful for reducing DBD symptoms regardless of IPV history. However, the magnitude of change in DBD symptom reduction differed significantly based on exposure to IPV. This finding extends current literature and demonstrates that boys previously exposed to IPV are more likely to experience persistent disruptive behaviors, and therefore respond less effectively to intervention, 3 years after treatment. With respect to antisocial behaviors, prior clinical trials research has shown that DBD interventions aimed at reducing harsh and inconsistent parenting are effective in reducing CU traits in children 4 to 9 years of age when compared to treatment as usual (McDonald, Dodson, Rosenfield, & Jouriles, 2011). Findings from the current study support this research but only for those boys who did not have an IPV history when they began treatment. Having a history of IPV predicted a more severe and stable course of CU traits that was unresponsive to treatment when compared to those boys without a prior IPV history. Together, these results provide evidence that IPV is systematically related to persistent courses of disruptive and antisocial behaviors throughout long-term follow-up that increase the risk for non-response to DBD interventions.

Third, support for an attenuation of the HPA axis was found in this study, as prior IPV was associated with significantly higher cortisol concentrations at study entry and significantly lower concentrations by the end of follow-up. Further support for an attenuation of the HPA axis lies in the examination of the entire cortisol trajectory for each group. Like previous research (Trickett et al., 2010), support for an attenuation of the HPA axis is relative to the trajectory of cortisol concentrations observed in a comparison group, such as those boys with a DBD but who did not have a prior history of IPV. Results from the current study support prior clinical trials research (Dorn et al., 2011) and demonstrate that behavioral interventions for DBDs can change cortisol trajectories but primarily for those boys without

an IPV history. On average, cortisol concentrations did not change across 3 years of follow-up for those boys with an IPV history. The lack of change in the average cortisol trajectory for the IPV group, along with significant group differences at the initial and final evaluations, suggests that IPV is associated with an attenuation of the HPA axis that is insensitive to DBD interventions when compared to the change in the average cortisol trajectory for non-traumatized boys with a DBD. Viewing attenuation in this manner may serve as a more relevant index of overall treatment response for those exposed to IPV, given the weaker reductions in disruptive and antisocial behaviors observed for this group during this same time.

The results of this study should be interpreted in light of several important limitations. First, only children 6-11 years of age and diagnosed with either ODD or CD were eligible for the clinical trial. Even with the three-year follow-up, it is not appropriate to generalize findings to older children or adolescents diagnosed with a DBD. This sample is most generalizable to early-onset ODD or CD, and not the more common, adolescent-onset ODD or CD. Second, the range of time when saliva samples were collected is also a limitation. Because the hormone component of the current study merged with an existing clinical trial, procedures for collecting samples were dictated largely by the procedures of the clinical trial already in place. While cortisol samples were aggregated and sampling time was used as a statistical covariate, collecting samples at the same time of day across multiple days would further reduce concerns about diurnal variation in cortisol samples. Third, the effects of puberty were also not assessed in the parent study. Future research will need to examine the influence of stage or timing of puberty on cortisol and treatment response. Fourth, analyses are based on a sample of boys with DBD and, therefore, findings cannot generalize to girls with a DBD. Finally, this study did not test whether specific types of prior violence exposure, severity of exposure, or recency of exposure are systematically related to clinical outcomes. It is likely these factors may strengthen the relationship between IPV and treatment response.

These limitations notwithstanding, the collective findings show that standard treatments for DBDs are not fully addressing the needs of traumatized boys with a DBD. Incorporating evidence-based, trauma-focused treatment components to address the unique consequences of IPV may help improve rates of long-term response. For instance, existing interventions for children with a DBD are now being modified to include empirically supported, trauma-specific components, such as emotion regulation skills, imaginal exposure, and parent-child communication around traumatic events, into the intervention (Kolko, Iselin, & Gully, 2011; Smith & Chamberlain, 2010). Such adaptations hold considerable promise in tailoring interventions with known efficacy to DBD sub-populations that present with specific risk factors linked to decreased treatment response. Such efforts are likely to enhance the overall efficacy of DBD interventions through better allocation of treatment services that more effectively target the pressing clinical needs of individual patients.

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Figure 1.

Trajectories of disruptive behavior disorder (DBD) symptoms, callous-unemotional (CU) traits, and cortisol concentrations by prior interpersonal violence (IPV) group. Group x time parameter estimates presented for DBD symptoms (Figure 1.A.; $n = 66$), CU traits (Figure 1.B.; $n = 66$), and cortisol (Figure 1.C.; $n = 66$) are significant at $p < .05$.

Table 1

Means and Standard Deviations for Each Outcome by IPV Group

Outcomes	Prior IPV						No Prior IPV					
	Pre		Post		6-month		1-year		2-year		3-year	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
DBD Sxs	8.03	2.17	5.57	2.97	-	-	5.59	2.99	6.56	5.22	5.69	4.12
CU Traits	7.30	2.57	6.12	2.66	6.00	2.05	6.46	1.79	6.27	1.91	5.65	2.93
Cortisol	0.13	0.06	0.19	0.11	0.13	0.07	0.15	0.07	0.14	0.07	0.13	0.06

Note. Diagnostic interviews not conducted at the 6-month follow-up assessment. IPV = interpersonal violence; DBD Sxs = disruptive behavior disorder symptoms; CU = callous-unemotional. Sample sizes for each point of data collection are as follows: Pre ($n = 66$); Post ($n = 65$); 6-month ($n = 65$); 1-year ($n = 64$); 2-year ($n = 64$); 3-year ($n = 59$).