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Adolescent Physiological and Behavioral Patterns of Emotion Dysregulation Predict Multisystemic Therapy Response

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Abstract

This study examined whether physiological and behavioral indicators of emotion dysregulation assessed over the course of Multisystemic Therapy (MST) were related to treatment response. Participants were 180 ethnically diverse adolescents (n=120 males), ranging in age from 12 to 17 years. Treatment response was assessed through therapist report and official arrest records. Changes in cortisol reactivity and changes in scores on a behavioral dysregulation subscale of the Child Behavior Checklist were used as indicators of emotion dysregulation. Hierarchical linear modeling analyses examined whether a less favorable treatment response was associated with cortisol reactivity measures (a) collected early in treatment and (b) over the course of treatment, as well as with behavioral reports of emotion dysregulation reported (c) early in treatment, and (d) over the course of treatment. Sex was explored as a moderator of these associations. Results indicated that both cortisol and behavioral indices of emotion dysregulation early in treatment and over the course of therapy predicted treatment responsiveness. This relationship was moderated by sex: girls were more likely to evidence a pattern of increasing emotion regulation prior to successful therapy response. The results lend further support to the notion of incorporating emotion regulation techniques into treatment protocols for delinquent behavior.

Keywords

Externalizing behavior; delinquency; developmental psychopathology

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Adolescents who engage in delinquent behaviors have problems in academic and interpersonal functioning (Barrett, Katsiyannis, & Zhang, 2010; Hinshaw, 1992), and an increased risk of comorbid mental health problems (Wade, 2001). Adolescent delinquency costs the United States millions of dollars annually, and has devastating impacts on the individual, as well as on family systems and communities (Zigler, Taussig, & Black, 1992).

A variety of interventions exist for treating youth with delinquent behavior problems. In general, traditional individual therapy does not lead to substantial short or long-term gains (Carr, 2009). Instead, evidence-based treatments (EBTs) like Parent Management Training (PMT) and Multisystemic Therapy (MST; for an overview, please see (Henggeler et al., 1998) consistently yield more effective results (Borduin et al., 1995; Kazdin, 1997; Schaeffer & Borduin, 2005; Timmons-Mitchell, Bender, Kishna, & Mitchell, 2006). Despite demonstrated effectiveness of the EBTs discussed above, they are by no means a panacea for eliminating delinquency. In fact, across decades of research, success rates and overall effect sizes of EBTs for conduct problems substantially vary (e.g., Webster-Stratton & Hammond, 1997; Webster-Stratton, Reid, & Hammond, 2004; Cook et al., 2008), and are heavily influenced by a number of variables, including youth age. This suggests that there continues to be significant variance not accounted for by existing treatments, and that perhaps one method of improving current EBTs is to more meticulously evaluate specific factors related to successful treatment response. Nevertheless, relatively few studies to date have examined specific factors that relate to effectiveness of existing EBTs (Carr, 2009; Ruma, Burke, & Thompson, 1996). Studies aimed at better identifying the unique variables that contribute to more favorable treatment outcomes can inform the next iteration of existing EBTs.

One client characteristic that might predict MST treatment response is emotion regulation. Emotion regulation is an active process whereby an individual influences which emotions he or she has, when to experience them, and how those emotions are experienced and expressed to others (Gross, 2013). Individuals who display deficits in their ability to regulate emotions are often referred to as “dysregulated.” Emotion dysregulation has been linked to increased levels of externalizing behavior problems (Blandon, Calkins, Keane, & O’Brien, 2008), including delinquent and aggressive behaviors. For example, a recent study found that emotion dysregulation mediated the relationship between stressful life events and aggressive behavior in adolescents (Herts, McLaughlin, & Hatzenbuehler, 2012). Relatedly, one meta-analysis found that programs that enhance social and emotional learning in schools result in significant reductions in child conduct problems (Durlak et al., 2011). Taken together, these results suggest that improvements in the regulation of emotion over time should be associated with decreases in conduct problems in both community and intervention samples, even when emotion dysregulation is not explicitly targeted by the intervention.

Although there is no consensus in the literature about the most appropriate way to measure emotion regulation, it is often measured through proxy variables such as cardiovascular or autonomic indices (e.g., Gross & Levenson, 1993; Egloff et al., 2006) or participant self-report (e.g., Gross & John, 2003). The present study utilized physiological (i.e., cortisol reactivity) and behavioral (i.e., parent-rated standardized index) proxies of emotion dysregulation. Cortisol is a glucocorticoid produced by the adrenal gland that acts as a regulatory factor in the human stress response (Gunnar & Quevedo, 2007). Previous studies

have suggested that prolonged exposure to certain kinds of stressors contributes to dysregulated HPA axis functioning (Fisher, Kim, Bruce, & Pears, 2012), and that dysregulation of the HPA axis is involved in the pathogenesis of child behavior and mood disorders, including delinquent behavior (Tyrka et al., 2012). Studies also suggest that sex (Oldehinkel & Bouma, 2011) and clinical status (Dietrich et al., 2013) moderate the relationship between cortisol and behavior. Previous studies have also suggested that high cortisol responses in response to a stressor can be conceptualized as a physiological correlate of emotion regulation strategies (Lam, Dickerson, Zoccola, & Zaldivar, 2009).

A small number of studies have examined cortisol levels as markers of treatment success in children undergoing treatment for externalizing behavior. For example, Schechter, Brennan, Cunningham, Foster, and Whitmore (2012) found that high morning cortisol levels measured early in MST predicted less of a decline in externalizing behaviors in males over the course of treatment. This study, however, did not evaluate sex differences or changes in cortisol over the course of treatment. A separate study assessing basal cortisol and cortisol stress responsivity before and after a psychotherapeutic intervention for 22 children diagnosed with Disruptive Behavior Disorder (DBD; Van De Wiel, Van Goozen, Matthys, Snoek, & Van Engeland, 2004) found that cortisol reactivity, but not baseline cortisol, predicted treatment outcome. Together these findings suggest that youth with higher cortisol reactivity at the outset of treatment might be less likely to benefit from the “full dose” of treatment than their less physiologically dysregulated counterparts, a question we examined in the present study.

Only a limited number of studies have assessed whether adolescent cortisol levels change over the course of treatment for externalizing disorders. One such study showed that boys diagnosed with DBD showed a significant decline in diurnal cortisol change over the course of a three-year treatment compared to a sample of healthy controls (Dorn, Kolko, Shenk, Susman, & Bukstein, 2011). Although these results preclude the conclusion that the treatment itself contributed to changes in diurnal cortisol profiles, they do inform the literature by demonstrating that physiological indicators over the course of treatment could be examined as markers of treatment utility in future studies. The present study will extend these findings by exploring whether, among youth receiving an EST for delinquency, physiological markers of emotion dysregulation can be used to distinguish treatment responders from non-responders. In addition, whereas the aforementioned studies have examined the relationship between baseline cortisol and externalizing behavior problems, this study will be among the first to evaluate the cortisol response to stress as a predictor of treatment response.

Another way of better understanding dysregulated emotional patterns in children with conduct problems is to study the emotion-related behaviors themselves. For example, in a study by Snyder, Schrepferman, and St. Peter (1997), higher levels of emotion dysregulation and negative reinforcement of aggressive behavior were found to covary with subjects’ irritability toward parents, and to predict child antisocial behavior. The authors suggested developing specific environmental contingencies in treatment protocols that would aim to treat specific problematic behaviors indicating emotion dysregulation in children. Others have suggested that school-based interventions would benefit from incorporating social-

emotional learning curricula since emotion regulatory factors are an important mechanism of change in children with behavioral and educational problems (Bradshaw, Goldweber, Fishbein, & Greenberg, 2012). Collectively, these studies highlight the importance of emotion dysregulation as a mechanism through which aggressive behaviors occur and can be modified or reduced. However, to our knowledge, specific behavioral measures of emotion dysregulation have not been examined as predictors of treatment outcome in children referred for externalizing behavior disorders. The current study fills this gap in the literature by examining how behavioral measures of emotion dysregulation predict outcomes in a treatment designed specifically for delinquency.

Previous research has not explicitly examined the moderating role of sex on treatment response. Nevertheless, sex differences in the cortisol-delinquency relationship as well as in the use of specific emotion regulatory strategies have emerged in previous studies. For example, Marsman and colleagues (2009) suggest that there may be a positive association between HPA-axis activity and externalizing behavior in girls, but an inverse relationship in boys. Other studies further corroborate the influence of sex on cortisol levels (e.g., Banks & Dabbs, 1996; Marsman et al., 2008). In studies of emotion regulation, adult men have been shown to use less effort (evaluated through neural imaging) when using cognitive regulation, whereas women are more likely to reappraise negative emotions by focusing on positive emotions more so than men (McRae, Ochsner, Mauss, Gabrieli, & Gross, 2008). Given that previous research has clearly identified sex differences in use of emotion regulation strategies as well as cortisol levels, it is important to more closely examine sex as a moderator of these variables in a treatment responsiveness context.

Our inclusion of two proxy variables for emotional dysregulation does not require response coherence, that is, a correlation between these behavioral and physiological responses; instead we are conceptualizing behavioral reactivity and cortisol reactivity as separate proxies of emotion dysregulation, and are evaluating each as an independent predictor of treatment response. Increases in physiological responding suggestive of emotion regulation may still occur in the absence of overt behaviors (Gross, 2002). Previous studies have found mixed results regarding response coherence (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005); some authors suggest modest associations between the three responses (experiential, behavioral, physiological) in relation to certain emotional states, like fear (Bradley & Lang, 2000), and others suggest no associations at all (Edelmann & Baker, 2002).

The current study seeks to explore whether behavioral and physiological indices of emotion dysregulation contribute to treatment responsiveness, as well as to examine whether these indices of dysregulation differentially predict outcomes for males and females. These questions will be answered by evaluating treatment responsiveness among a sample of youth undergoing Multisystemic Therapy, which has been previously established as an efficacious, empirically supported treatment for adolescent delinquency (Cunningham & Henggeler, 1999; Henggeler, 1999). In the present study, hierarchical linear modeling analyses examined whether three indicators of treatment response were associated with changes in cortisol reactivity and behavioral indicators of emotion dysregulation over the course of treatment. We also examined whether emotion dysregulation at the beginning of treatment

predicted worse outcome. We hypothesized that treatment nonresponse would be predicted by (a) higher cortisol reactivity at the onset of treatment; (b) increasing cortisol reactivity over the course of treatment; (c) higher scores on the behavioral measure of emotion dysregulation at the onset of treatment; and (d) increasing reactivity in behavior over the course of treatment. Furthermore, we explored whether sex would moderate these relationships.

Method

Participants

The participants in the study were 180 adolescents (n = 120 males), ranging in age from 12 to 17 years, and adult caregivers. This developmental age range was chosen because adolescents are the primary recipients of MST. Descriptive statistics are provided in Table 1. The overall sample of participants was ethnically diverse: Over 47% were Caucasian, 27.8% were Latino/a, 19.8% were African American, and 4.3% identified as “Other.” All adolescent participants received MST from one of four licensed MST programs serving the Denver Metropolitan area. Youth were referred by either the juvenile justice system or social service agencies. To be included in the present study, adolescents had to meet the following inclusion criteria: (a) referral for crimes against others, property offenses, substance abuse, or other externalizing problem behaviors; (b) residence in the current caregiver’s home with no immediate plans for placement elsewhere within the treatment timeframe; and (c) one caregiver who was willing to participate in the study. Because previous research has shown that HPA axis regulation differs as a function of parity status (Kivlighan, DiPietro, Costigan, & Laudenslager, 2008), 5 pregnant females who also participated were excluded from analyses in this study.

Setting

A total of 52 therapists provided Multisystemic Therapy. Therapists were predominantly female (73%, n=38) and Caucasian (80%, n=42). Therapists had to meet their agency’s requirements for hiring and complete their agency’s training requirements prior to participation (for an overview of therapist requirements, see Crandal et al., 2015). This study was approved by the Institutional Review Boards of the University of Colorado, the Medical University of South Carolina, Emory University, and Alliant International University. Caregivers and therapists provided consent for study participation, and adolescents provided assent. Therapists, caregivers and adolescents were compensated for their involvement in the study. The first assessment (T1) was conducted as close to treatment onset as scheduling permitted and was, on average, 3.1 weeks (SD = 2.04) from intake. The second assessment (T2) occurred during mid-treatment on average 9.3 (SD=2.73) weeks from treatment start, and the final assessment (T4) occurred immediately post-treatment, on average 19.3 weeks after treatment start (SD = 7.48). Because time between assessments differed across participants, it was controlled in analyses. (Table 1 about here)

Measures and Procedures

Cortisol Reactivity—Saliva samples were collected in the participants’ homes twice during each visit: before and after a math stressor task in order to assess cortisol reactivity to

a stressful situation. The majority (85%) of samples were collected between 1:00 p.m. and 4:00 p.m. in the afternoon. For the math stressor task, youth were instructed to count backwards by thirteen from high numbers, and were provided with very stern critical evaluations of their performance (e.g., “that is incorrect, most people did this quickly”). Cortisol was collected via passive drool before and about 20 minutes after the task. Once collected, samples were frozen at −20°C and then shipped on dry ice to the Endocrine Core Laboratory at the Yerkes National Primate Research Center at Emory University to be assayed. On the day of assay, samples were thawed, vortexed, and centrifuged to remove any particulate matter, and cortisol was assayed using an enzyme immunoassay kit (DSL, Webster, TX), catalogue number DSL-10-67100. Each sample was assayed in duplicate, and duplicate tests that generated errors of more than 20 percent were retested.

Changes in cortisol over time were evaluated by measuring differences in cortisol reactivity (calculated as post-stressor task level minus pre-task level) across three different time points in treatment (T1: early in treatment, T2: mid-treatment, and T4: at the end of treatment). Higher cortisol reactivity was suggestive of greater emotional dysregulation.

Behavioral Reactivity—Adolescents’ caregivers were administered behavioral questionnaires during each visit. Specifically, the Child Behavior Checklist (CBCL; Achenbach, 1991) an empirically validated measure of youth behavior problems, was used to assess behavioral indicators of emotion dysregulation. The CBCL consists of 113 behavior problem items with responses are coded on a scale of 0 (Never) to 2 (Almost Always). For the purposes of the present study, we used the CBCL-Posttraumatic Stress Problems Scale (PTSP; Wolfe, Gentile, & Wolfe, 1989) as our behavioral index of emotion dysregulation. Although there has been disagreement in the literature regarding whether the PTSP scale is best conceptualized as a measure of posttraumatic stress (Wolfe et al., 1989; King et al., 2000), or as a broad indicator of emotion regulation (Althoff, Ayer, Rettew, & Hudziak, 2010), the item composition of the scale reflects both internalizing and externalizing self-regulatory problems (e.g., argues a lot, sudden changes in mood or feelings, feels others are out to get him/her) characteristic of deficits in emotional regulatory behaviors. Therefore decreases in caregivers’ scores of behavioral reactivity were considered to reflect reductions in emotion dysregulation over the course of treatment. The items on the behavioral reactivity scale evidenced high internal consistency at T1, T2, and T4 (alphas>0.80).

MST Treatment response—MST treatment response was measured in three ways, selected in particular to avoid shared method variance between predictor and criterion variables, and, with the exception of arrest data, was obtained via phone calls or in-person meetings with the therapist. Arrest record information was obtained from the Judicial Branch’s Integrated Colorado Online Network database approximately six months to a year after treatment discharge. First, the Case Discharge Summary (CDS), originally developed in a MST transportability study (Schoenwald, Sheidow, Letourneau, & Liao, 2003), required therapists to complete two questions assessing discharge circumstances: (a) reason for discharge, and (b) who made the decision to discharge. Using Schoenwald et al.’s (2003) scoring system (0=not successful, 1=successful), a successful MST response was indicated

by a therapist assessment that the family met or partially met treatment goals, and had control and involvement over discharge decisions collaboratively with the therapist. Schoenwald et al. (2003) provided data indicating that this measure of treatment success has acceptable construct validity.

The second measure of MST treatment response was the 14-item Therapist Perception of Treatment Outcome (TPTO) scale (Crandal, Foster, Cunningham, Brennan, & Whitmore, 2015). This questionnaire asks therapists at the conclusion of treatment (T4) to rate how strongly they agree with statements about caregiver and youth responses to treatment on a six-point Likert scale (1-Agree Strongly; 6-Disagree Strongly). Crandal and colleagues (2015) found that total scores for the TPTO scale administered to the therapists in this sample at T4 evidenced high internal consistency (alpha>0.90) and acceptable construct validity. High scores on the TPTO indicate more favorable treatment outcome. Lastly, we assessed MST response using adolescent arrest records. Individuals who were not arrested during the follow-up (post T4) period were considered positive treatment responders according to this measure.

Control Variables

Previous studies have suggested that health variables and pubertal development could influence HPA axis activity (e.g., Granger, Hibel, Fortunato, & Kapelewski, 2009; Netherton, Goodyer, Tamplin, & Herbert, 2004). A 20-item Child Health Questionnaire was utilized to measure variables like stimulant, antidepressant, or antihistamine use, whether the adolescent had a cold or flu, and the adolescent's time of awakening. Height and weight were measured at each time point, and the Petersen Pubertal Developmental Scale (PPDS; Petersen, Crockett, Richards, & Boxer, 1988) was administered to evaluate youth puberty status. The PPDS contains 5 items, each on a 4-point scale, about youth physical changes associated with adolescence. A total puberty score was calculated by summing the individual items and dividing by 5 (to maintain the original metric).

Statistical Analyses

Hierarchical Linear Modeling (HLM; Raudenbush, Bryk, & Congdon, 2011) techniques were used to test the primary hypotheses, and to evaluate sex as a possible moderator. HLM is frequently used in developmental research when investigators are concerned with measuring growth trajectories for individuals (Raudenbush & Bryk, 2002). The data gleaned from the present study are particularly well suited for HLM because they meet three important criteria: (a) there are at least three waves of data (T1, T2, T4); (b) there is an interest in change over time (in this case, in cortisol and behavioral dysregulation ratings); and (c) there is a sensible metric for evaluating change over time (Singer & Willett, 2003). Because HLM can only model changes over time in criterion variables, not predictors, the HLM outcome variables were cortisol and behavioral reactivity ratings, with measures of treatment response used as predictors. We deemed this to be appropriate because the intent of the study was to examine associations between patterns of change and treatment response, not causal relationships.

Results

Correlations between the youth regulation and treatment response measures are presented in Table 2. Behavioral reactivity and cortisol reactivity were significantly and negatively correlated with case discharge assessments of treatment success and TPTO. Among the therapy response variables, only treatment success and TPTO were significantly correlated with one another. Cortisol and behavioral dysregulation measures were not significantly correlated with one another. (Table 2 about here)

Preliminary Data Analysis

Before testing the primary hypotheses, it was necessary to assess whether therapist effects accounted for significant variance in the Level 1 variables (behavioral and cortisol reactivity) as youth were nested within therapists. Intraclass Correlation Coefficients (ICCs) were computed for models that included behavioral and physiological indices separately at Level 1, nested within youth (Level 2), which were nested within therapists (Level 3). Time was scored as days since enrollment in the study and entered at T1. The ICCs for Level 3 ($\tau_\beta/(\tau_\beta+\tau_\pi+\sigma^2)$ in these models were minimal ($ICC_{Behavior}$=0.0003; $ICC_{Cortisol}$=0.001), suggesting that there were no differential effects on the indices based on the therapist assigned to a family. Once a higher level of the model (Level 3) is found not to cause interdependence of the data at a lower level, it can be excluded from the model (Raudenbush, Bryk, Cheong, Congdon, & Toit, 2004). Therefore, two-level HLM models were used for hypothesis testing with changes in behavioral and cortisol reactivity included at Level 1, and therapy response variables (i.e., absence of post-treatment arrest, case discharge success rating, and TPTO scores) examined as predictors in separate models at Level 2. Additionally, to test for sex moderation, an interaction term between sex and therapy response variable was added to each of the equations at Level 2. In cases where this sex by treatment response interaction term was significant, results were reported for the sample as a whole and for each sex separately. In cases where sex did not moderate the association between dysregulation and MST response, results are reported for the sample as a whole.

Unconditional model: Cortisol and behavioral reactivity at Level 1—HLM analyses were performed to examine the trajectory of cortisol across treatment. First, an unconditional model was run with cortisol reactivity entered as the outcome and time (measured in days since T1) as the predictor at Level 1. Both intercept and slope equations were treated as random effects. The estimated mean slope of cortisol change was 0.009 (*SE*=0.005). Based on this mean trajectory, measures of youth's cortisol increased at an average of 0.009 points per observation point from onset of treatment to termination. The slope in this model was not significant at $p < 0.09$, suggesting that there was not a significant change across treatment for the sample as a whole. However, the variance component of the slope in this model indicated that there was significant variation among slopes of cortisol reactivity for the individuals in our sample (χ^2=180.43, $p = 0.014$), which suggests that some individuals showed declines in cortisol reactivity, whereas others showed increases.

A similar procedure was followed for the behavioral reactivity variable. The estimated mean slope of behavior change was −0.63 (*SE*=0.08). Based on this mean trajectory, youth's behavioral dysregulation scores (Table 1) decreased at an average of 0.63 points per observation point from onset of treatment to termination. The slope in this model was significant at $p < 0.001$, suggesting significant change across treatment. Furthermore, the variance component of the slope in this model indicated that there was significant variation among slopes of behavior for the individuals in our sample (χ^2=182.14, p=0.03).

Testing for confounds—Prior to the start of analyses for hypothesis testing, potential time varying confounds were examined at Level 1 and youth varying confounds were examined at Level 2 in HLM. Time varying variables were those that changed across the course of treatment (e.g., an individual's height), whereas youth-related variables remained consistent over the course of treatment (e.g., ethnicity). Baseline cortisol prior to the start of the math stressor task was controlled for in all cortisol reactivity analyses. Variables previously identified in the literature as confounds for cortisol were tested as potential Level 1 confounds. Only minutes after awakening (p=0.01) and height (p=0.001) were significantly related to cortisol reactivity, and were included as Level 1 controls in all subsequent cortisol analyses. Table 1 includes descriptive statistics on time-varying control variables for cortisol analyses. Additionally, puberty, sex, age, SES, and ethnicity (Black, White, and Latino, dummy-coded) of youth were tested as potential confounds at Level 2. Only SES (p=0.04) and Latino ethnicity (p< 0.05) were significant. Both variables were only significant in the intercept equations, and thus controlled for in all subsequent intercept analyses. A similar procedure was followed to identify time varying and youth varying confounds for all behavior reactivity analyses. Age, sex, ethnicity, SES, and puberty status of youth were tested in HLM. No Level 1 confounds were identified, and only SES (p=0.003) and Caucasian ethnicity (p=0.02) were significantly related to behavior dysregulation and thus controlled at Level 2. SES was only controlled in the intercept equation, but because Caucasian ethnicity significantly predicted behavior dysregulation slope, it was controlled for in both intercept and slope Level 2 equations.

Hypothesis Testing

Results for associations between treatment response and changes in behavior reactivity and cortisol reactivity over time are grouped by the type of time-varying variable (i.e., cortisol or behavioral measures of dysregulation). Effect sizes are reported for significant findings using Proportional Reduction in Variance (PRV) statistics (Peugh, 2010), which are calculated using the following equation: $(var_{NoPredictor}\text{-}var_{Predictor})/var_{NoPredictor}$. "Predictor" represents the level-1, level-2 intercept, or level-2 slope variance and "No Predictor" represents the variance estimate of the model prior to adding a predictor. As noted above, in cases where sex was found to be a significant moderator between regulation/reactivity and treatment success, results are presented for each sex separately. In these and all HLM models tested in this study, time in therapy was uncentered, and therefore the intercept term represents the value of the youth dysregulation variable at treatment onset (T1).

Cortisol reactivity and treatment response—In the first set of HLM analyses, cortisol reactivity was tested at Level 1 with time and time varying confounds included as

predictors in the Level 1 equation and youth level confounds included as predictors at Level 2. Continuous predictors at Level 2 (e.g., TPTO, SES) were grand-mean centered. Specifically, associations between treatment responsiveness measures (tested separately at Level 2) and cortisol reactivity over time (Level 1) were assessed with the following equations (dropping out sex and the interaction of sex by treatment responsiveness when the interaction was not significant):

Level 1 Equation:

$$\text{CortisolReactivity}_{\text{ti}} = \pi_{0\text{i}} + \pi_{1\text{i}}\,(\text{PreMathCortisol}_{\text{i}}) + \pi_{2\text{i}}\,(\text{MinutesAwake}_{\text{ti}}) + \pi_{3\text{i}}\,(\text{Height}_{\text{ti}}) + \pi_{4\text{i}}\,(\text{Time}_{\text{ti}})\,\text{e}_{\text{ti}}$$

Level 2 Equations:

$$\pi_{0\text{i}}\,(\text{Intercept}) = \beta_{10} + \beta_{11}\,(\text{SES}_{\text{i}}) + \beta_{12}\,(\text{Latino Ethnicity}_{i}) + \beta_{13}\,(\text{Treatment Response}) + \beta_{14}\,(\text{Sex}) + \beta_{14}\,(\text{Treatment Response} \times \text{Sex}) + r_{4\text{i}}$$

$$\pi_{4\text{i}}\,(\text{Slope}) = \beta_{40} + \beta_{43}\,(\text{Treatment Response}) + \beta_{44}\,(\text{Sex}) + \beta_{44}\,(\text{Treatment Response} \times \text{Sex}) + r_{4\text{i}}$$

As seen in Table 3, results indicate that the cortisol reactivity measure at T1 was significantly and negatively associated with TPTO scores at the end of treatment (PRV=25.00). In other words, higher levels of cortisol reactivity at the onset of treatment were related to poorer treatment response as rated by therapists. In addition, and as hypothesized, the slope of cortisol reactivity across the course of treatment was significantly and negatively related to treatment response as measured by the TPTO (PRV=9.23). This suggests that in cases where cortisol reactivity increased across treatment, youth demonstrated less favorable TPTO ratings. Cortisol proxies of emotion regulation were not significantly associated with the Case Discharge Summary measure of treatment success or arrest (see Table 3). (Table 3 about here)

As can be seen in Table 4, the interaction term between sex and the treatment response variable of arrest (entered at Level 2) significantly predicted both the cortisol reactivity intercept and the slope of cortisol reactivity over time. To further understand the pattern of these sex interaction effects, the effect of sex on the results was examined using the Aiken and West (1991) method of probing interactions. Specifically, HLM models that tested for associations between cortisol intercept and slope and the treatment response variable of arrest were repeated, first with sex dummy coded as 0=male, 1=female and next with 0=female, 1=male. As noted in Table 4, both a higher level of cortisol reactivity at treatment onset (intercept; PRV=11.76), and reductions in cortisol reactivity across the course of treatment (slope; PRV=3.2) were related to a greater likelihood of post-treatment arrest for males. Opposite, but nonsignificant associations between cortisol reactivity and arrest were observed for females. (Table 4 about here)

Behavioral reactivity and treatment response—In the next set of analyses, behavioral reactivity was tested at Level 1 with time included as a predictor in the Level 1 equation and youth level confounds included as predictors at Level 2. Associations between treatment response measures (tested separately at Level 2) and the behavioral index of emotion dysregulation over time (Level 1) were assessed with the following equations:

Level 1 Equation:

$$\text{Behavioral Index}_{ti} = \pi_{0i} + \pi_{1i}\,(\text{time}) + e_{ti}$$

Level 2 Equations:

$$\pi_{0i}\,(\text{Intercept}) = \beta_{00} + \beta_{01}\,(\text{SES}_i) + \beta_{02}\,(\text{Caucasian Ethnicity}_i) + \beta_{03}\,(\text{Treatment Response}) + \beta_{04}\,(\text{Sex}) + \beta_{05}\,(\text{Treatment Response} \times \text{Sex}) + r_{0i}$$

$$\pi_{1i}\,(\text{Slope}) = \beta_{10} + \beta_{12}\,(\text{Caucasian Ethnicity}_i) + \beta_{13}\,(\text{Treatment Response}) + \beta_{14}\,(\text{Sex}) + \beta_{15}\,(\text{Treatment Response} \times \text{Sex}) + r_{1i}$$

As can be seen in Table 5, and consistent with the hypotheses, the slope of behavior reactivity over the course of treatment was significantly and negatively related to therapy response as measured by the Case Discharge Summary (PRV=20.80) and post-treatment arrest (PRV=22.94). Specifically, if a youth's behavior became less reactive over the course of treatment (thus reflecting less emotion dysregulation), clinicians indicated more successful termination and the participant was less likely to be arrested. (Table 5 about here)

As noted in Table 6, a significant interaction was also observed between sex and TPTO measure at Level 2 in predicting the slope of behavioral dysregulation over time. To further understand the pattern of this interaction, the overall sample was again examined using the Aiken and West (1991) method of testing interactions. As can be seen in Table 6 and consistent with the present hypothesis, reductions in behavioral indicators of emotion dysregulation across the course of treatment (slope) were related to significantly more favorable TPTO scores in females (PRV=27.70). This pattern was not observed in males. (Table 6 about here).

To rule out the possibility that the specific effect observed for behavior dysregulation in females was not better accounted for by a reduction in internalizing symptoms over the course of treatment, post-hoc analyses were performed in which the CBCL internalizing scale from T1, T2, and T4 was substituted for the CBCL behavior dysregulation scales from the same time points, and associations between internalizing problems and the TPTO scale were examined. The results were nonsignificant for both intercept and slope equations.

Discussion

This study adds to the existing aggression and delinquency literature (Allwood, Handwerger, Kivlighan, Granger, & Stroud, 2011; Popma et al., 2006; Sondeijker et al., 2007) by providing empirical evidence that changes in cortisol and behavioral reactivity are associated with treatment nonresponsiveness in a clinical sample of youth with externalizing behavior problems, thereby explicitly identifying specific factors that may relate to treatment effectiveness of an empirically validated EBT (Carr, 2009). The participants in the present study were referred for treatment, and thus represent extreme ("clinical") cases of aggressive and delinquent behavior, which is important because past studies have found the relationship between biological markers and delinquency to be stronger among clinical than community samples (Dietrich et al., 2013). Our study further demonstrates that both emotion

dysregulation at the onset of treatment and changes in emotion dysregulation over the course of treatment, as measured by both physiological and behavioral proxy variables, predict treatment nonresponsiveness. As discussed earlier, the lack of response coherence between the physiological and behavioral measures was not unexpected, as previous studies have demonstrated that the relationship between different constructs assessing emotion dysregulation is mixed at best (Mauss et al., 2005).

Many previous studies exploring the relationship between HPA axis functioning and delinquency have excluded females from analyses, often due to low sample sizes. The inclusion of females is a methodological strength of this study. Importantly, for females, results for the behavioral index of emotion dysregulation were stronger, and results for both physiological and behavioral indices of emotion dysregulation were consistently in the predicted direction.

Although most hypotheses were supported in the direction predicted, the finding that decreases in cortisol reactivity over the course of treatment predicted more arrests for males during a six-month follow up was unexpected. There are several possible explanations for this result. First, we performed multiple tests of our hypotheses with several different measures of treatment success, and therefore it is possible that this result is due to Type 1 error (and indeed the effect size for this finding was much lower than others noted in the study). Second, previous research has noted that sex moderates the direction of the association between cortisol and externalizing behaviors such that high levels of cortisol are associated with externalizing behaviors in girls, and low levels of cortisol are associated with externalizing behaviors in boys; our finding is consistent with this sex-moderated pattern (Marsman et al., 2009). Third, there may be subtypes of delinquent youth for whom different patterns of cortisol reactivity predict differently to treatment responsiveness. For example, previous research has shown that individuals with callous-unemotional traits are likely to have lower levels of cortisol (Cima, Smeets, & Jelicic, 2008; Honk, Schutter, Hermans, & Putman, 2003), so it is possible that these individuals in particular may show a poor response to MST treatment. Overall, these results expand on the available literature (e.g., Dorn et al., 2011), and suggest that future studies should more explicitly explore physiological markers associated with externalizing behavior problems

Overall, the results of the current study lend support to suggestions by researchers like Herts et al. (2012), who argued that clinicians should include emotion regulatory strategies in treatment protocols with delinquent youth, and should monitor changes in emotion dysregulation over the course of treatment in order to identify particular delinquent youth who may not be meeting therapeutic goals outlined at the onset of treatment. Secondly, findings raise the intriguing possibility that family-oriented interventions may have positive impacts on youth emotion regulation deficits without directly addressing them. Similar findings have been shown in a recent study evaluating the effectiveness of a family-based intervention in promoting self-regulation skills in young children (Chang, Shaw, Dishion, Gardner, & Wilson, 2014). Finally, the results presented here suggest that differential treatment predictors exist for males and females, and that females in particular may benefit from an emotion regulation focus in therapy.

Limitations and Future Directions

The findings of the present study should be interpreted in light of several limitations. Results may or may not generalize beyond the MST treatment model, to youth problems other than antisocial behavior, and to younger children. In addition, power limitations restricted tests of moderators to sex (i.e., 3 way interactions). Given that this study found ethnic differences in measures of dysregulation at treatment entry and in improvements in behavioral dysregulation, future researchers should consider exploring patterns of change among different ethnic groups. A further limitation of the present study is the lack of a comparison or control group. However, since the purpose of this study was to evaluate MST responders versus non-responders in an effectiveness context, not treatment efficacy per se, the lack of a control group does not necessarily minimize the importance of the study.

These preliminary findings are correlational, and do not establish a cause-effect relationship between improvements in emotion regulation and therapy responsiveness, and thus claims of including an emotion regulatory component in future iterations of these treatments need to be substantiated through additional carefully designed randomized control studies of ESTs for delinquency (e.g., MST). In addition our cortisol and behavioral reactivity measures are two of many possible proxies for emotion dysregulation. Replication of our findings with additional measures of emotion regulation are needed. Examples include heart rate variability measures, social vignettes (e.g., Dodge & Somberg, 1987) or the use of virtual reality to create stressful situations that the youth must maneuver. Finally, as is typical in most psychological research, our effect sizes were generally small in magnitude, suggesting that additional factors beyond emotion dysregulation influence MST treatment outcomes. Our findings should be considered in the context of the existing literature concerning moderators of MST treatment response.

In summary, results of this study confirmed that both cortisol and behavior proxies of emotion dysregulation early in treatment and over the course of therapy were associated with treatment responsiveness. Furthermore, this relationship was moderated by sex. As predicted, females were more likely to evidence a pattern of increasing emotion regulation prior to successful therapy response. In contrast, a decrease in cortisol reactivity across treatment was associated with increased risk of arrest for males. These findings suggest that researchers may want to explore modified treatment strategies for males and females who evidence problems with emotion regulation.

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Table 1

Descriptive Statistics for Predictor, Response, and Control Variables

Variable	*n*	Mean/%	Standard Deviation
Cortisol Reactivity			
Time 1	164	−0.05	0.18
Time 2	136	−0.03	0.22
Time 4	147	0.01	0.25
Behavioral Dysregulation			
Time 1	180	8.27	5.12
Time 2	146	5.73	4.60
Time 4	164	5.30	4.59
Clinician Rated Treatment Success	166	0.36	0.48
TPTO	157	46.12	13.21
Arrested Post Treatment	180	26%	—
Height (cm)	176	66.59	3.96
Minutes after Awakening			
Time 1	178	405.96	166.33
Time 2	146	377.17	162.47
Time 4	153	389.39	173.39

Table 2

Intercorrelations for Study Variables

Variable	Cortisol	Behavioral Dysregulation	Case Discharge	TPTO	Arrest
Level 1 Variables:					
Cortisol Reactivity	-	−0.02	−0.12 *	−0.15 **	0.08
Behavioral Dysregulation		-	−0.15 **	−0.12 *	−0.02
Level 2 Variables:					
Case Discharge Summary			-	0.53 **	−0.06
TPTO				-	−0.02
Arrested					-

* $p < .05$.

** $p < .01$.

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Table 3

Main Effects: MST Treatment Response as Related to Cortisol Reactivity Over Time

Treatment Response Predictor Variable	Coefficient	*p*-value	*S.E.*	*t*
Case Discharge				
Summary				
Intercept	−0.03	0.11	0.02	−1.63
Slope	−0.01	0.19	0.01	−1.57
TPTO				
Intercept	−0.02	**0.03**	0.01	−2.18
Slope	−0.01	**0.004**	0.01	−2.98
Arrest				
Intercept	0.04	0.12	0.03	1.57
Slope	−0.02	0.15	0.01	−1.44

Note. Bold-faced effects are statistically significant, $p < .05$

Table 4

Interactions: MST Treatment Response as Related to Cortisol Reactivity Over Time

Treatment Response Predictor Variable	Coefficient	*p*-value	*S.E.*	*t*
Case Discharge by Sex				
Interaction				
Intercept	0.03	0.41	0.04	0.83
Slope	0.01	0.52	0.02	−0.64
TPTO by Sex				
Interaction				
Intercept	0.001	0.98	0.03	0.03
Slope	−0.02	0.11	0.01	−1.59
Arrest by Sex				
Interaction				
Intercept	−0.11	**0.04**	0.05	−2.08
Slope	0.06	**0.04**	0.03	2.11
Males-Arrest				
Intercept	0.07	**0.03**	0.03	2.23
Slope	−0.03	**0.01**	0.01	−2.67
Females-Arrest				
Intercept	−0.04	0.33	0.05	−0.99
Slope	0.03	0.27	0.03	1.11

Note. Bold-faced effects are statistically significant, $p < .05$

Table 5

Main Effects: Behavioral Regulation Over Time and MST Treatment Response

Treatment Response Predictor Variable	Coefficient	*p*-value	*S.E.*	*t*
Case Discharge Summary				
Intercept	−0.68	0.35	0.72	−0.95
Slope	−0.42	**0.01**	0.15	−2.81
TPTO				
Intercept	−0.51	0.22	0.41	−1.23
Slope	−0.10	0.23	0.08	−1.20
Arrest				
Intercept	−0.97	0.20	0.76	−1.28
Slope	0.41	**0.03**	0.19	2.22

Note. Bold-faced effects are statistically significant, $p < .05$

Table 6

Interactions: Behavioral Regulation Over Time and MST Treatment Response

Treatment Response Predictor Variable	Coefficient	*p*-value	*S.E.*	*t*
Case Discharge by Sex				
Interaction				
Intercept	−0.69	0.65	1.55	−0.45
Slope	−0.48	0.11	0.30	−1.60
TPTO by Sex				
Interaction				
Intercept	0.74	0.42	0.92	0.81
Slope	−0.33	**0.01**	0.18	−2.49
Males- TPTO				
Slope	0.04	0.66	0.10	0.44
Females-TPTO				
Slope	−0.40	0.01	0.15	−2.71
Arrest by Sex				
Interaction				
Intercept	1.21	0.54	1.96	0.62
Slope	−0.10	0.80	0.39	−0.25

Note. Bold-faced effects are statistically significant, $p < .05$