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Biosocial Processes Predicting Multisystemic Therapy Treatment Response

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Abstract

This study examined biological (testosterone) and social (deviant peer affiliation) factors early in treatment as predictors of treatment outcome among adolescent boys receiving Multisystemic Therapy (MST) in community settings. Outcome variables included changes in youth aggression and delinquency as reported by the primary caregiver. Testosterone and deviant peer affiliation were assessed at treatment onset; and outcome variables (aggression and delinquency) were assessed at treatment onset, mid-treatment and end-of-treatment. Participants were 112 adolescent boys (M age = 15.42, SD = 1.31) and their caregivers. Growth curve analyses revealed that the combination of high testosterone and high deviant peer affiliation early in treatment were significantly associated with less of a decline in aggression and delinquency over the course of treatment. Results provide novel evidence for the role of testosterone in the prediction of future externalizing behaviors. Clinical and theoretical implications are discussed.

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Disclosure Statement

Phillippe B. Cunningham is a board member of Evidence Based Services, which has a licensing agreement with Multisystemic Services, LLC for the dissemination of Multisystemic therapy.

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Keywords

testosterone; aggression; MST; deviant peers; treatment outcome

The recalcitrance of serious externalizing behavior problems (e.g., delinquency, substance abuse) among adolescents is a major public health concern among many parents, community leaders, clinicians, and researchers. And while research has documented the efficacy and effectiveness of comprehensive family- and ecologically-based interventions (Curtis, Ronan, & Borduin, 2004; Waldron & Turner, 2008), even under the most favorable conditions, however, the large effect sizes shown in clinical trials dissipate to small effects when therapists deliver evidence-based treatments (EBTs) in real-world practice settings (Curtis et al., 2004). Eyberg, Nelson, and Boggs (2008) recently concluded that the field of treatment research would be greatly advanced if investigators began to answer the questions “*for whom, how, and when*” do EBTs work (Eyberg et al., 2008). One variable that is often targeted by family-based EBTs for youth externalizing behaviors is association with deviant peers. A focus on environmental factors, however, fails to illuminate the individual-level drivers for *why* some youth are unresponsive to EBTs. Examining individual-level factors that may moderate the impact of environmental therapeutic targets could enrich the theoretical foundation of evidence-based therapies that aim to ameliorate externalizing behavior problems.

One promising individual-level biological factor to examine in treatment outcome research with adolescents exhibiting serious externalizing disorders is baseline hormone levels, particularly testosterone. Current theoretical models of the reciprocal influence of testosterone and psychosocial processes complement the models of change that underlie evidence-based treatments for externalizing behavior problems (Fisher, Stoolmiller, Gunnar, & Burraston, 2007; Mazur & Booth, 1998). To begin integrating the role of hormones into our understanding of treatment mechanisms, it is necessary to first identify whether levels of hormones predict treatment outcome. The premise of the current study is that the additional examination of how biological factors interact with deviant peer affiliation to predict changes in externalizing behavior problems in youth might further help clarify the potential effectiveness of EBTs at an individual level. Specifically, our study examines whether youth testosterone levels interact with deviant peer affiliations to predict aggressive and delinquent behavior changes in the context of an effectiveness study of Multisystemic Therapy (MST; Henggeler, Schoenwald, Borduin, Rowland, & Cunningham, 2009).

Testosterone and Aggression

The consistent finding that boys are significantly more aggressive than girls and that boys have higher levels of testosterone than girls provided the empirical rationale for initial investigations of the association between testosterone and aggression. Whereas animal studies have consistently linked testosterone to aggression, research examining the direct association between testosterone and aggression in humans has been mixed (Archer, 2006; Mazur & Booth, 1998; Ramirez, 2003). For example, Dabbs and colleagues (1995, 1990) found a positive relationship between testosterone and violent behavior among prison inmates. A positive relationship between testosterone and aggression/delinquency was also observed by Maras et al. (2003) in a pre-adolescent sample. On the other hand, Dorn and colleagues (2009) found a negative relationship and others (e.g., Constantino et al., 1993) have found no relationship between testosterone and aggression. One possible explanation for these discrepant findings is that testosterone may not have a simple main effect on aggression (or delinquency). Instead, testosterone may be associated with aggression and or delinquency, but only under certain environmental circumstances (e.g., social interactions

with deviant peers). Consistent with this biosocial explanation, recent cross-sectional investigations have examined the interactive role of testosterone and social systems (e.g., Booth, Johnson, Granger, Crouter, & McHale, 2003; Dorn et al., 2009; Foshee et al., 2007; Rowe, Maughan, Worthman, Costello, & Angold, 2004; Ryan, 2009; Udry, 1990; Updegraff, Booth, & Thayer, 2006; Vermeersch, T'Sjoen, Kaufman, & Vincke, 2008). Particular social systems of interest have included deviant peer affiliations.

Testosterone and Deviant Peer Affiliation

To date, only two studies have directly examined the interaction between testosterone and deviant peer affiliation as a predictor of aggressive behavior (Dorn et al., 2009; Rowe et al., 2004). Neither study found evidence for a significant interaction. Methodological challenges, however, may have affected the results. For example, Rowe et al. (2004) used a single item scale to measure deviant peer affiliation. And Dorn et al. (2009) examined the moderating role of deviant peer affiliation in a group of pre-pubescent children. As noted by the authors, additional factors may have masked testosterone levels, making it difficult to observe effects.

Although others have not specifically tested the interaction between testosterone levels and deviant peer affiliation as a predictor of aggression per se, other findings suggest this interaction is related to risk behaviors in adolescence. For example, Vermeersch et al. (2008) found that boys with high testosterone were more prone to risk-taking behavior because they were also more likely to associate with boys who also engaged in risk-taking behavior. In another study, researchers examined the interaction between testosterone and deviant peer affiliation in the prediction of adolescent cigarette and alcohol involvement and found a significant positive relationship between testosterone and involvement with these substances for boys who affiliated with peers who used the respective substance (Foshee et al., 2007). Similarly, Bauman, Foshee, and Haley (1992) found a stronger relationship between testosterone and smoking behavior for adolescents who had friends who smoked than for adolescents who did not have friends who smoked.

Testosterone and Treatment Outcome

To date, only one study has examined pre-treatment levels of testosterone as a predictor of treatment outcome (Shenk et al., 2011). These researchers found that testosterone levels significantly predicted membership into a low treatment response trajectory and that testosterone levels were related to poorer long-term outcomes in pre-pubescent children. The current study extended this research by examining the relationship between testosterone and treatment outcome in a sample of adolescents and by examining a modifiable treatment target (deviant peer affiliation) as a moderator of this relationship. The literature reviewed above suggests that the consideration of social context is important for better understanding the testosterone-aggression/delinquency link. Because family-based EBTs such as MST often result in a "changed social environment," examination of the testosterone-aggression and testosterone-delinquency relationship in the context of an effectiveness study may be particularly informative. Furthermore, the interaction between testosterone and deviant peer affiliation in a treatment context is also directly relevant to determining the specific conditions under which EBTs are more or less effective in reducing adolescent externalizing behavior problems.

This study examined the testosterone-aggression and testosterone-delinquency relationship in the context of Multisystemic Therapy (Henggeler et al., 2009). MST is a home-based therapeutic approach aimed at targeting the systems that are known correlates of antisocial behavior: child characteristics, family characteristics, peer relationships, school factors, and neighborhood and community characteristics (Henggeler, Schoenwald, Borduin, Rowland,

& Cunningham, 1998). This home-based therapeutic approach is highly individualized and intensive. Therapists are on call at all times and visit the home 1–2 times a week, depending on individual need. The core principles of this therapeutic approach are strength focused and involve skills building (Henggeler et al., 1998). Therapists individualize treatments with a focus on behavioral principles, integrating additional evidence-based interventions (e.g., behavioral, cognitive behavioral interventions, parent management training, etc.) where appropriate. Interventions often include parent-child interventions (e.g., changing discipline practices, challenging parental beliefs about childrearing, child behaviors and child characteristics, implementing effective rule making, punishments, and rewards), familial intervention (e.g., conflict between caretakers that interferes with caring for the youth), school based interventions (e.g., establishing home-school communication and contingencies), and interventions at the peer level (e.g., decreasing association with deviant peers and increasing association with pro-social peers, and implementing unpleasant consequences). With over 30 years of research and 20 randomized clinical trials (six conducted by independent investigators), MST is considered by many to be a strongly empirically supported treatment for youth with serious externalizing problems (e.g., aggression, delinquency, substance use).

Our purpose in the current study was to examine the association between levels of testosterone, deviant peer affiliation, and aggressive and delinquent behavior changes in the context of an effectiveness study of MST. We hypothesized that basal levels of testosterone, measured early in treatment, would interact with deviant peer affiliation, measured early in treatment, to predict changes in caregiver report of aggression and delinquency over the course of MST. Because deviant peer association is an established risk factor for aggressive and delinquent behavior, we predicted that youth with high levels of testosterone and more deviant peers at treatment onset would evidence a poorer treatment response. The findings reported here include data that were part of a larger longitudinal multi-site study of the dissemination of MST in community settings. This larger study was designed to examine a transactional model of treatment response and non-response that focused on the interaction of individual-level (biological), familial, and environmental factors that are likely to influence treatment outcome.

Method

Participants

Participants for this study were 112 adolescent males and their caregivers who completed assessments early in treatment. At the time of the first assessment (Time 1; early in treatment), adolescent age ranged from 12 to 17 years ($M = 15.42$, $SD = 1.31$). Over 46.3% of the participants self-identified as Caucasian, 22.3% as African-American, 26.4% as Latino, and 5.0% of the participants identified as “other.” The majority of youth lived with their mother (71.9%) at the Time 1 assessment, 10.7% lived with their father, 11.6% lived with their grandmother, and 5.8% lived with another family member or a non-related adult. Average educational attainment for primary caregivers was 13 years. The primary caregivers’ mean age was 44.92 years ($SD = 9.95$). Forty percent of families reported they received financial assistance.

Families were referred by social service agencies and the juvenile justice system to one of four licensed MST provider organizations in a large metropolitan area. Inclusion criteria included a family with a child between the ages of 12 and 17 years who (a) was referred for MST services from social service agencies or juvenile justice courts for crimes against another person, property offenses, substance abuse, or other externalizing behavior problems; (b) was living in the caregiver’s home for at least a month prior to treatment onset, with no immediate plans for placement elsewhere; and (c) had at least one caregiver

willing to participate in MST. Only boys in the sample were included in the present study due to the major gender differences in testosterone levels during adolescence.

Procedure

Participants were identified by their therapist as eligible for the study, and then recruited by research staff. Assessments were completed in the families' homes on laptop computers. Time 1 assessments (treatment onset) took place an average of 23 days after intake appointments. Mid-treatment (Time 2 and Time 3) and post-treatment (Time 4) assessments took place an average of 45, 88, and 134 days after Time 1 assessments, respectively. Caregivers provided written consent, and youth provided assents. The Human Subjects Institutional Review Board at all of the participating research sites approved this study.

Predictor Measures

Salivary Testosterone Collection and Assay—At treatment onset, one “baseline” saliva sample was collected during the home visit by having youth passively drool directly into a specimen tube to the level of 1 cc for purposes of assaying testosterone. Participants' vials were then frozen and stored at -20°C for an average of three months before being shipped overnight in dry ice, in batches to the Endocrine Core Laboratory at the Yerkes National Primate Research Center at Emory University. Upon arrival at Yerkes, samples were stored at -20°C . On the day of assay, samples were thawed and vortexed, then centrifuged to remove particulate matter. A modified version of the Diagnostic Systems Laboratories (DSL, Webster, TX) double antibody radioimmunoassay (RIA; test kit catalogue number DSL-4100) of total testosterone in serum protocol was used for the testosterone assay. This assay procedure has an analytical sensitivity of 2.0 and uses 200 μl of saliva. The intra- and inter-assay coefficient of variation was 3.41 and 16.4%, respectively. Each sample was assayed in duplicate. Duplicate tests with an error more than 20% were retested. Duplicate test results were averaged and this value was used in analyses. Total levels of basal testosterone at Time 1 (treatment onset) provided a continuous measure of testosterone in the analyses.

Peer Delinquency Scale (PDS)—The PDS Loeber, Farrington, Stouthamerloeber-Loeber, & Van Kammen, 1994) was administered to youth at Time 1 to assess delinquent peer affiliation. The PDS provides a measure of the number of friends who engage in delinquent acts and/or substance use. Good concurrent and predictive validity has been reported (Keenan, Loeber, Zhang, Stouthamerloeber, & Vankammen, 1995). The original scale contains 15 items for which youth are asked to report on behavior during the past 30 days. In the current study, one additional substance use item was added: “During the past 30 days, how many of your friends have smoked cigarettes?” This item was added due to the strong overlap between tobacco and substance use (Biglan, Brennan, Foster, & Holder, 2003). Thus, the PDS for the current study contained 16 items. The alpha coefficient for this scale was 0.93. Scores were calculated by summing youth responses to all items to create a continuous scale of peer delinquency. Higher scores indicated more peer delinquency.

Outcome Measure

Child Behavior Checklist – Ages 6–18 (CBCL)—Caregiver reports of aggressive and delinquent behavior were assessed using ratings on the CBCL (Achenbach, 1991), which is one of the most well-validated measures of child behavioral functioning (Achenbach, 1991; Achenbach & Rescorla, 2001). This measure was administered to caregivers at Time 1, Time 2, Time 3, and Time 4. The CBCL consists of 113 behavior items in which caregivers rate as 0 (*not true*), 1 (*somewhat true*), or 2 (*very true*). This study used the aggression (20 items) and delinquency (13 items) subscales. Caregivers were asked to describe behaviors of

the youth that occurred at the time of assessment and within the past 30 days. Raw scores on the aggression and delinquency subscales were converted to *T*-scores and used as a continuous dependent measure of delinquency and aggression. In this study, the aggressive subscale had a coefficient alpha of .93 for Time 1, of .92 for Time 2, of .93 for Time 3, and of .93 for Time 4. The delinquency subscale had a coefficient alpha of .85 for Time 1, of .83 for Time 2, of .86 for Time 3, and of .87 for Time 4.

Control Variables

Several variables have been shown to influence testosterone, including health information such as sleeping, eating, and medication schedule, as well as pubertal development and body mass index (Booth, Johnson, & Granger, 1999; Granger, Shirtcliff, Booth, Kivlighan, & Schwartz, 2004; Hibel, Granger, Cicchetti, & Rogosch, 2007). Thus, each of these variables was examined as possible control variables in the current study. In addition, age and race were examined as influential variables on each outcome variable, as age and race are commonly related to aggression and delinquency outcomes (results are reported in Table 2).

Health Questionnaire—Consistent with previous research, eating, sleeping (i.e., how long the youth had been awake), and medication schedules were examined in relation to testosterone (Granger et al., 2004; Hibel et al., 2007). At T1, youth and their caregiver were asked about the youth's eating (e.g., *what time did you last eat something and have you had caffeine today*), sleeping, and medication schedules (e.g., *have you taken any steroid, antihistamines, cold/flu, or allergy medications today*) in the 24 hours before the assessment. Results did not reveal any significant relationships between eating patterns or medication schedules and testosterone: (eating: $r = .08$, $p = .39$; caffeine intake: $r = .22$, $p = .16$; steroids: $r = .12$, $p = .21$; antihistamines: $r = -.07$, $p = .47$; cold/flu: $r = .03$, $p = .74$; allergy medications: $r = -.04$, $p = .69$). Time of day was not significantly correlated with testosterone levels, $r = -.15$, $p = .13$; however, hours since awakening, $r = -.19$, $p = .04$, was significantly associated with testosterone levels. Analyses were performed twice—once controlling for time since awakening and once without controlling for time since awakening. Results were highly similar. Thus, results using the simpler model (i.e., results without this variable as a control) are presented (Raudenbush & Bryk, 2002).

Pubertal Development—The Petersen Pubertal Development Scale (PPDS; Petersen, Crockett, Richards, & Boxer, 1988) was administered to adolescents at T1. This scale contains 5 items and response choices range from “1 – not yet started” to “4 – seems completed.” Youth were asked about their growth spurt, body hair, changes in skin, and changes in their voice. This measure has been widely used in the examination of pubertal development/status (e.g., Granger et al., 2004) and has shown to be reliable and valid measure of pubertal status (Petersen et al., 1988). Consistent with other research, we assessed the association between testosterone and total puberty score, as well as testosterone and individual puberty items (Booth et al., 2003; Granger et al., 2004). The total puberty score was calculated by summing the items and dividing by 5, to maintain the original metric.

In this study, age and puberty were not as highly correlated as reported in previous studies, $r = .45$, $p < .001$, suggesting that age and puberty may account for unique variance when accounting for development (Booth et al. 2003). Testosterone was significantly positively associated with age, $r = .27$, $p = .01$. There were only trend results for the association between basal testosterone and puberty, $r = .17$, $p = .09$. When controlling for age, the trend results for the association between testosterone and puberty disappeared, $r = -.08$, $p = .44$. When controlling for puberty, the significant results for the association between testosterone and age remained significant, $r = .29$, $p = .003$. These results suggest that, for this study, age

is a potential confound, rather than self-reported puberty status. Age, therefore, was controlled in all analyses.

Height and Weight—For the purposes of this study, participant's body mass index (participant's body weight divided by the square of their height) was calculated (Booth et al., 1999; Mazur, 1995; Tremblay et al., 1998). The association between testosterone, height, weight, and BMI was assessed. Results revealed no significant associations with testosterone (Height: $r = .01$, $p = .95$; Weight: $r = .04$, $p = .68$; BMI: $r = .08$, $p = .39$). Therefore, height and weight were not included as control variables in the analyses.

Missing data

Our initial sample size was 121. At Time 1, seven adolescents did not provide a level of saliva that would support testosterone assay so these seven adolescents were removed before completing data analyses. Two additional adolescents provided data on the deviant peer measure that revealed a distribution of scores that suggested these youth may have provided inaccurate responses, so their data were deemed unusable for calculating deviant peer involvement. Thus, the sample size in the final analyses included 112 adolescents and their families.

Of the 112 caregivers, half (52.7%) of the families completed the aggression and delinquency assessments at all four time points, 23.2% of the caregivers completed assessments at three time points only, 17.9% of the caregivers completed assessments at two time points only, and 6.3% of the caregivers completed assessments at Time 1 only. There were several reasons why some youth and families failed to complete each of the planned assessments that attest to the severity of problem behaviors of the current sample, including (1) difficulty locating the youth and or the youth's family, (2) youth and/or caregiver refused to participate, (3) youth placed out-of-home and in a facility that would not allow the protocol to be completed (e.g., detention, wilderness program, residential treatment), and (4) youth ran away from home. Notably, those who completed Time 1 assessment only were not significantly different from those who completed the Time 4 assessment on demographic variables or on the dependent variables used in this study. Therefore data were considered to be missing at random (MAR) for the purposes of analyses and maximum likelihood estimation of hierarchical models was used. This procedure allows for efficient use of all available time points to estimate HLM models (Raudenbush & Bryk, 2002).

Results

Descriptive Analyses

Table 1 presents means and standard deviations for the independent and dependent variables used in the analyses at each time point. Table 2 presents the correlations between all repeated measures and the two control variables (i.e., race and age). As shown in Table 2, race ("1" Minority, "0" Non-minority) was correlated with CBCL aggression at all 4 time points; and with CBCL delinquency at three of the four time points. Of note, testosterone levels correlated positively with affiliation with deviant peer groups at treatment onset. In addition, deviant peer affiliation correlated positively with CBCL aggression scores at Time 1 and Time 4; and with CBCL delinquency scores at three of the four time points. Testosterone was not directly associated with any of the outcome measures of aggression or delinquency.

Data Analytic Approach

Analyses were conducted using growth curve modeling techniques (GCM; Raudenbush & Bryk, 2001) and the HLM 6 computer software (Raudenbush, Bryk, & Congdon, 2004).

Data analyses using these techniques has several advantages over other approaches to examine longitudinal data. First, these techniques allow for the retention of the entire sample, despite missing data across repeated measures. Second, similar to repeated measures ANOVA, HLM provides information as to whether there is significant change over time, on average, and the direction of that change. In addition to this, HLM allows for the examination of individual changes over time and between-subject differences in these changes. Third, HLM accounts for variation in time between data points across subjects which results in a more precise estimation of change.

Analyses included three-level models, with four repeated measurements of youth aggressive and delinquent behavior (Level 1) nested within 112 youth (Level 2) who were nested within 44 therapists (many therapists saw more than one family; Level 3). Although therapist level characteristics were not examined in the current study, youth were nested within therapist to account for interdependence among participants who met with the same therapist (Raudenbush et al., 2004). Change over time was modeled with linear terms. The linear term was computed as months between assessment periods of interest. More specifically, Time was calculated as the number of months for each participant between the first and subsequent assessments.

In each HLM model, all error terms were free to vary across adolescents and across therapists. Results were evaluated using final estimation of fixed effects with robust standard errors. Evaluation of results using these statistics has shown to provide accurate significance tests under conditions of non-normality and in the presence of outliers and model misspecification (Raudenbush & Bryk, 2002). Interaction terms were created by calculating the product of centered Time 1 predictors. For model specification, the *Time* variable was entered at Level 1, un-centered. All continuous variables at Level 2 were grand-mean centered; and the dichotomous control variable (minority status) at Level 2 was un-centered. Significant interactions were interpreted using simple slope analytic methods suggested by Preacher, Curran, & Bauer (2003) and Curran, Bauer, & Willoughby (2006).

Baseline Models—In the current study, caregiver report of aggressive, $t(43) = -7.47, p < .001$, and delinquent behaviors, $t(43) = -5.76, p < .001$, demonstrated a systematic linear decline over time, on average. When compared, a linear model was a better fit than a mean-and-variance model and a quadratic model for each outcome. Accordingly, a linear baseline model was specified for all analyses.

To examine moderation, the following model was specified with testosterone, the moderator, and the corresponding interaction term entered at Level 2:

$$\begin{aligned} \text{(Level 1)} \quad Y_{it} \text{ (aggression or delinquency)} &= \pi_{0i}(\text{Intercept}) + \pi_{1i}(\text{Time}) + e_{it} \\ \text{(Level 2)} \quad \pi_{0i}(\text{Intercept}) &= \beta_{00} + \beta_{01}(\text{testosterone}) + \beta_{02}(\text{moderator}) + \beta_{03}(\text{interaction}) + r_{0i} \\ \pi_{1i}(\text{Time}) &= \beta_{10} + \beta_{11}(\text{testosterone}) + \beta_{12}(\text{moderator}) + \beta_{13}(\text{interaction}) + r_{1i} \end{aligned}$$

For the hypotheses, the significance of the coefficient for the interaction term β_{13} was of interest.

Aggression Outcome—Results showed no significant main effects for T1 testosterone, $t(43) = 0.28, p = 0.78$, or T1 deviant peer affiliation, $t(43) = -0.69, p = .49$, on changes in aggression over the course of MST. The interaction between T1 testosterone levels and T1 deviant peer affiliation was a significant predictor of changes in caregiver report of CBCL aggression scores, $t(43) = 4.12, p < .001$. That is, the effect of T1 testosterone on changes in

caregiver report of aggression depended on the degree of T1 deviant peer affiliation. To examine this interaction further, Preacher, Curran, and Bauer's (2003) software for identifying simple effects was used to interpret the relationship between testosterone and the aggression at different values of deviant peer affiliations. For the moderator "deviant peer affiliations," simple effects were examined at zero, at the mean, and at 1 SD above the mean level of deviant peer affiliations as these values are clinically relevant and therefore provide more valuable information about simple effects.

Post hoc simple slope analyses of this significant interaction revealed that for participants with no deviant peer affiliation at T1, T1 testosterone was not significantly associated with changes in caregiver report of CBCL aggression, $b = 0.00$, $p = 0.84$. At the average level of deviant peer affiliation for the sample, testosterone levels were significantly associated with changes in CBCL aggression scores, $b = .09$, $p < .001$. That is, higher levels of testosterone were associated with a slower rate of decline in CBCL aggression over the course of treatment. At the value of one standard deviation above the sample mean of deviant peer affiliation, higher testosterone was more strongly associated with a slower rate of decline in CBCL aggression, $b = .19$, $p < .001$, over the course of MST treatment.

Delinquency Outcome—Similar results were found when CBCL delinquency was examined as the outcome variable. Specifically, the main effects for T1 testosterone, $t(43) = -0.23$, $p = .82$, and T1 deviant peer affiliation, $t(43) = -1.68$, $p = .10$, were not significant predictors of changes in delinquency over the course of MST. Results were significant for the interaction between T1 testosterone and T1 deviant peer affiliation as a predictor of changes in CBCL delinquency, $t(43) = 2.59$, $p = 0.01$. Post hoc simple slope analyses showed that Time 1 testosterone was not significantly associated with changes in caregiver report of CBCL delinquency for adolescents with no deviant peer affiliations, $b = 0.00$, $p = 0.87$. At the average level of deviant peer affiliation for the sample, testosterone levels were significantly associated with changes in CBCL delinquency scores, $b = .11$, $p = .02$. That is, higher levels of testosterone were associated with a slower rate of decline in CBCL delinquency over the course of treatment. At the value of one standard deviation above the sample mean of deviant peer affiliation, higher testosterone was more strongly associated with a slower rate of decline in CBCL delinquency, $b = .23$, $p = .03$, over the course of MST treatment.

Discussion

The purpose of this study was to examine the association between testosterone and changes in aggression and delinquency within the context of an intervention study carried out in an effectiveness context, while taking into account the moderating influences of deviant peer affiliations. Results suggest that among male adolescents with deviant peer associations, higher levels of testosterone predict poorer response to treatment. Our findings are consistent with previous research (Shenk et al., 2011) and affirm the value of incorporating both biological and social factors in predicting aggression and delinquent behavior (Bauman et al., 1992; Booth et al., 2003; Rowe et al., 2004). Our findings show how changes over time may be better explained by examining biosocial interactions than by looking at each factor in isolation. The results of the current study also suggest that testosterone may be an important biological risk factor to consider when predicting treatment response in boys.

As predicted, youth with initially high levels of testosterone and more deviant peer affiliations evidenced less decline in aggressive and delinquent behavior over the course of MST. Previous research suggests that high levels of testosterone are related to sensation seeking and risk taking behavior (Vermeersch et al., 2008). An increased propensity for sensation seeking and risk taking behaviors may promote some individuals to seek out

deviant peers, as evidenced by the correlation between testosterone levels and deviant peer affiliation at Time 1 in the present study. As such, these results would suggest that high-testosterone individuals may be at particular risk for deviancy training. Deviancy training increases the opportunity to learn and to be socialized into new problem behaviors (Snyder, 2002). An individual with a propensity for sensation seeking and risk taking behavior, such as high-testosterone individuals, may be more apt to participate and actively listen to deviant peer talk (e.g., describing previous violent or criminal behavior). Deviancy training, plus a compromised ability to appreciate negative consequences that result from negative behaviors, may exacerbate aggressive and delinquent behavior (Snyder, 2002; Terburg, Morgan, & van Honk, 2009). Furthermore, because these youth showed fewer behavioral changes during treatment, results suggest that youth with high testosterone may be especially reluctant to give up their deviant peer affiliations – a frequent target of MST and other parenting interventions. The reluctance to give up deviant peer affiliations may be related to the underlying vulnerability for a social circle that fulfills a drive for dominance and status (Mazur & Booth, 1998; Mehta, Jones, & Josephs, 2008; Sapolsky, 1993).

Clinical Implications for Evidence-Based Treatments

These results have several clinical implications for those treating or conducting treatment outcome studies of aggressive and delinquent adolescent males. Males with a combination of high testosterone and deviant peer affiliations showed less of a decline in their standardized CBCL scores over time. In contrast, youth with lower levels of testosterone and deviant peer affiliations in the sample evidenced a decrease in T-scores at a magnitude of approximately one half of a standard deviation. This magnitude of change typically resulted in a shift of CBCL T-scores from the clinical range to the borderline clinical range or from the borderline clinical range to the normal range of functioning over the course of MST. It appears then that males with high testosterone and deviant peer affiliations did not evidence the clinically significant problem behavior changes otherwise associated with MST. It may be important to consider the potential role of testosterone and other biological variables in predicting treatment non-response to evidence-based treatments in which the treatment targets deviant peer affiliations, especially when the adolescent has large numbers of deviant peers. A heavy emphasis on intervening in adolescent peer relationships may be necessary among biologically at-risk youth. Adolescent boys with this profile may be more vulnerable for sensation seeking and to have a stronger attraction (because of higher levels of testosterone) to deviant peer groups – making additional strategies above and beyond what current evidence-based treatments have to offer potentially necessary.

Strengths and Limitations

This study has some notable limitations. First, the current study did not include a comparison group/control group. The absence of a comparison/control group makes it impossible to attribute changes in aggression and delinquency over time to MST treatment, specifically. Such factors as maturation and passage of time may account for the observed declines in both aggression and delinquency. However, the use of an established empirically validated treatment for adolescents with chronic aggression and delinquency, administered with standardized procedures to enhance adherence, strengthens the confidence that participants received MST as it is typically delivered in real-world settings, where randomized controlled trials have shown significant improvements in past studies (see for example Borduin et al., 1995; Henggeler et al., 1993). Second, although it is a strength that an assessment of adolescent use of medications was included, it was assessed over a 24 hour period. It is possible that our assessment time frame was too short to reveal variability necessary to show a relationship with testosterone. Given our current knowledge of the impact of medication on testosterone level (Hibel et al., 2007), previous research has assessed medication use over longer periods of time (e.g., Dorn et al., 2009). Third, given

current conceptualizations of testosterone and dominance (e.g., Mazur & Booth, 1998), the inclusion of a measure of dominance behavior would have aided in the interpretation of our results. Fourth, we included aggression and delinquency as separate outcome variables because some biological variables (and testosterone in particular) have been differentially related to aggression and delinquency in past research (van Bokhoven et al., 2006). These variables were highly correlated in our sample, so it is not surprising that they share some common predictors. This may reflect common pathways within clinical samples. Alternately, it could be due to shared informant variance in CBCL subscale ratings. Future research might disentangle these more fully by including multiple informant measures of externalizing behavior. Finally, whether results generalize to other evidence- and family-based treatments for aggression and delinquency is an empirical question.

The current study has notable strengths as well. First, this study provides novel evidence for possible mechanisms underlying non-responsiveness to MST. Very few intervention studies have considered biological factors as predictors of treatment non-response. Second, the use of a clinical population allows for a clear examination of change over time. With a less severely delinquent sample, the nuances of behavior change may have been more difficult to detect. Third, the use of an established empirically validated treatment for adolescents with chronic aggression and delinquency strengthens the confidence in the current results. Fourth, the sample in this study is more demographically heterogeneous than those examined in previous studies of the relationship between testosterone and problem behavior. Finally, the data were collected in real-world settings in which MST was provided in the community, enhancing external validity.

Summary

In summary, this is the second study to examine testosterone as a predictor of treatment outcome and the first to examine this question in a sample of adolescents with significant behavior problems. Results of this study provide preliminary evidence for the role of testosterone and deviant peer affiliations as predictors of non-response to MST treatment. In addition our results suggest that complex biosocial risk processes may be important to consider in tailoring treatments for aggressive and delinquent youth.

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Highlights

- Examined the interaction between testosterone & deviant peers on treatment response.
- Boys with high testosterone and deviant peers showed poor treatment response.
- Novel insight into the impact of testosterone on treatment response.

Table 1
Cross-sectional descriptive statistics for the independent and dependent variables used in analyses

Variable	Time 1		Time 2		Time 3		Time 4	
	M	SD	M	SD	M	SD	M	SD
Basal Testosterone	69.34	62.03						
Deviant Peer Affiliation	9.60	10.59						
CBCL Aggression ^a	61.18	10.81	56.66	8.52	57.55	8.51	56.65	9.26
CBCL Delinquency ^a	67.07	9.94	62.75	8.79	61.38	9.26	62.63	10.06

Note: CBCL = Child Behavior Checklist 6–18 (Achenbach, 1991).

^a Scores represent T-scores.

Table 2

Correlations between study variables

	1	2	3	4	5	6	7	8	9	10	11	12
1. Age	—											
2. Race	.06	—										
3. Testosterone	.27*	.08	—									
4. Deviant Peer Affiliation	.16	.15	.21*	—								
5. CBCL Aggression T1	-.19*	.29**	.02	.23*	—							
6. CBCL Aggression T2	-.30**	.22*	-.04	.14	.71***	—						
7. CBCL Aggression T3	-.15	.28*	-.13	.22	.71***	.73***	—					
8. CBCL Aggression T4	-.12	.24*	.09	.30**	.68***	.73***	.74***	—				
9. CBCL Delinquency T1	-.10	.27**	.11	.24**	.69***	.55***	.48***	.54***	—			
10. CBCL Delinquency T2	-.17	.21*	-.05	.18	.54***	.73***	.57***	.63***	.64***	—		
11. CBCL Delinquency T3	.01	.19	.01	.28*	.46***	.46***	.73***	.51***	.56***	.64***	—	
12. CBCL Delinquency T4	-.08	.21*	.05	.28**	.40***	.51***	.63***	.69***	.57***	.72***	.68***	—

Note: T = time. CBCL = Child Behavior Checklist 6–18 (Achenbach, 1991).

^aRace: 1 = minority; 0 = non-minority.*
 $p < .05$.**
 $p < .01$.***
 $p < .001$. Italicized text represents trend result at $p < .10$.