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Stacy R. Ryan Date

Examining Testosterone and Aggression in a Biosocial Framework

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Examining Testosterone and Aggression in a Biosocial Framework

By

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B.A., Emory University, 2004
M.A., Emory University, 2006

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An abstract of
A dissertation submitted to the Faculty of the
James T. Laney School of Graduate Studies of Emory University
in partial fulfillment of the requirements for the degree of
Doctor of Philosophy in Clinical Psychology
2010

Abstract

Examining Testosterone and Aggression in a Biosocial Framework
By Stacy R. Ryan, M.A.

Recent investigations suggest that examining the moderating role of social context variables may be important for understanding the link between testosterone and adolescent aggression and delinquency. The present research extends previous work by examining the interaction between testosterone and social context within a cross-sectional study (Study One) and within a longitudinal treatment outcome study (Study Two). Study One examined adolescent boys between the ages of 12 and 17 who were participating in a larger longitudinal investigation of Multisystemic Therapy in community settings. Data were gathered in the context of pre-treatment assessments, and were analyzed to test the hypotheses that the relationship between testosterone and aggression and delinquency depends on (a) deviant peer group affiliation, (b) parenting style, and/or (c) basal cortisol levels. Results did not reveal significant interactions between testosterone and cortisol or between testosterone and social context (i.e., parenting style and deviant peer affiliation) as predictors of aggressive or delinquent outcomes. Study Two extended the investigation of these variables to a treatment context by examining the relationship between testosterone and social context and testosterone and cortisol on the growth trajectories of caregiver report of therapist adherence, youth aggression, and youth delinquency. Study Two also examined the co-variation between testosterone and youth aggression and delinquency over the course of MST treatment. Results revealed a lower rate of positive change in caregiver report of therapist adherence for the youth in the sample who had both high basal cortisol levels and high testosterone levels at pre-treatment. Additionally, the combination of high testosterone and high deviant peer affiliation as well as the combination of high testosterone and high quality parenting at pre-treatment were associated with less of a decline in aggression and delinquency over the course of treatment. Last, results of Study Two revealed a positive association between testosterone and delinquency across time in the MST treatment context. Results of Study One add to the mixed literature on the association between testosterone and aggression and delinquency. Results of Study Two 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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Acknowledgments

I would like to express my gratitude to my advisor, Dr. Patricia Brennan, for all her support and guidance in helping me create this work and for contributing so greatly to my professional and personal growth throughout my tenure at Emory University.

I would also like to take this opportunity to express my deepest appreciation to my fiancé, Amati Pettes, for his support throughout the years. Without his support, this journey would not have been possible. Amati has been my rock of stability through the day-to-day challenges of my professional journey. Likewise, thank you Aiyana for coming into my life and helping me to find my center, which helped me to create a healthy balance during the final years of such an arduous process.

Mom, thank you for always listening, patiently, to me vent and rant and for always believing in me. To my future mother-in-law, Ms. Delores Pettes, thank you for your encouragement and the many trips to Atlanta and Houston to help care for our family unit. Your efforts helped to insure stability during major milestones along the way.

Last, but certainly not least, I would like to thank my committee members, as well as Phillippe Cunningham, Ph.D., at MUSC and Sharon Foster, Ph.D., at Alliant International University for their support and guidance, which helped shape the final product of this project.

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EXAMINING TESTOSTERONE AND AGGRESSION IN A BIOSOCIAL FRAMEWORK

Cumulative evidence suggests that the relationship between testosterone and aggression and delinquency is more complex than originally hypothesized. Researchers have studied this association in pre-pubescent, pubescent, and post-pubescent children and adolescents: examining testosterone and aggression (Azumerdi et. al, 2006; Chance et al., 2000; Constantino, Grosz, Saenger, Chandler, Nandi, & Earls, 1993; Granger, Shirtcliff, Zahn-Waxler, Usher, Klimes-Dougan, & Hastings, 2003; Mattsson, Schalling, Olweus, Low, & Svensson, 1980; Owleus, Mattson, Schalling, & Low, 1980; Scerbo & Kolko, 1994; Susman, Inoff-Germain, Nottleman, Loriaux, Cutler, & Chrousos, 1987), the hormonal precursors of testosterone and aggression (Constantino et al., 1993; Susman et al., 1987; van Goozen, Matthys, Cohen-Kettenis, Thijssen, & van Egeland, 1998; van Goozen, van den Ban, Matthys, Cohen-Kettenis, Thijssen, & van Engeland, 2000), and the ratio of testosterone to other hormones and aggression (Nottelman, Susman, Inoff-Germain, Cutler, Loraux, & Chrousos, 1987). Across all types of studies, results are mixed.

More specifically, researchers have examined (1) whether testosterone levels correlate with conduct disorder or behavior scale ratings of aggression and delinquency; and (2) whether youth with an aggressive disposition have higher levels of testosterone than youth without an aggressive disposition.

Panel design studies have included the examination of testosterone as a determinant of aggressive behavior; and studies employing regression techniques have examined bidirectional relationships between testosterone and aggressive behavior over time (for a review see Archer, 1991; Book, Starztk, & Quinsey, 2001; Ramirez, 2003). Again, findings from these studies have been mixed.

Importantly, the majority of studies of the association between testosterone and aggression and delinquency have been cross-sectional, with only three studies providing prospective data on this relationship (Maras, Laucht, Gerdes, Wilhelm, Lewicka, Haack, et al., 2003; Schaal, Tremblay, Soussignan, & Susman, 1996; van Bokhoven, van Goozen, Engeland, Schaal, Arseneault, Seguin, et al., 2006) and only one study reporting a retrospective association (Dabbs & Morris, 1990). Cross-sectional studies have produced highly variable results (see Archer, 1991 for a review), whereas longitudinal studies provide more consistent findings concerning the relationship between these factors.

Some researchers have suggested that the reason for the inconsistent results in the cross sectional literature may be the wide variations in methodology: varying sample size, varying sample population characteristics, varying sampling strategies, varying types of co-occurring mental illnesses, and varying methods of measurement and scoring of aggression and delinquency (Archer, 1991; Ramirez, 2003). Another potential reason for the mixed results in this area is that the social context or other hormonal factors may moderate the testosterone-aggression relationship; these potential moderators have not been consistently controlled or assessed in previous studies.

This dissertation is designed to specifically test for such moderating effects in an attempt to further elucidate the associations between testosterone and adolescent aggression and delinquency. This work is based on two premises: one is that the cross-sectional relationship between testosterone and aggression and delinquency is best captured within the context of a biosocial model, one that takes into account potential social or environmental moderators of the testosterone-aggression relationship (Booth, Johnson, Granger, Crouter, & McHale, 2003; Dorn, Kolko, Susman, Huang, Stein, Music, et al., 2009; Rowe, Maughan, Worthman, Costello & Angold, 2003; Vermeersch, T'Sjoen, Kaufman, & Vincke, 2008a). And two, that the relationship between testosterone and aggression and delinquency can be experimentally tested within the context of a longitudinal design in which the social context is intentionally altered, such as a treatment study (Mazur & Booth, 1998).

There are several theoretical models that have been used to conceptualize the relationship between testosterone and aggression and delinquency including Social Control Theory (Udry, 1990), Sutherland's Differential Association Theory (Vermeersch et al., 2008a), and Dodge and Pettit's (2003) biopsychosocial model of conduct problems (Dorn et al., 2009). Mazur et al. (1998) provide two additional models: the basal model and reciprocal model. The basal model assumes that testosterone levels are stable over time with negligible fluctuations around a genetically determined level. The reciprocal model assumes that there is a reciprocal relationship between testosterone and status competitions such that there is a positive feedback loop, whereby testosterone influences status competitions and the outcome of status competitions, in turn, influences testosterone levels.

In general, cross-sectional research designs assume a basal model of the relationship between testosterone and aggression and delinquency. It is important, however, to note that a longitudinal research design does not automatically imply the investigation of a reciprocal model. In fact, to date, longitudinal designs of the association between testosterone and aggression and delinquency have largely adhered to the basal model as well. It is argued that exploring the testosterone-aggression relationship within a longitudinal design, whereby a reciprocal model may be applied, should be the next step in this area of research.

The major aim of this dissertation was to extend the literature on testosterone and youth aggression by examining this relationship in a biosocial context.
First, a cross-sectional study design was used to investigate a basal-biosocial model of the relationship between testosterone and social context variables as well as testosterone and cortisol as predictors of aggression and delinquency. Second, a longitudinal treatment outcome study was used in the investigation of the interplay between testosterone and social context variables and testosterone and cortisol in the prediction of changes in aggression over time, whereby a reciprocal-biosocial model was applied to the results. One major strength of this two- study design is that the youth who served as participants in the cross-sectional study also served as participants in the longitudinal study, using the same social context variables. This allows for the direct comparison of the basal model and the reciprocal model. Such a comparison could be fruitful for future theoretical developments in this area of research.

Study 1: Cross Sectional Study

The consistent finding that boys are significantly more aggressive than girls and the fact that boys have higher levels of testosterone than girls provided the initial rationale for the investigation of the testosterone–aggression link. Reviews of the literature, however, reveal that the association between testosterone and aggression and delinquency is mixed (Archer, 2006; Muzar et al., 1998; Ramirez, 2003). Some studies find that testosterone and aggression are positively related, some find a negative relationship, and others find that there is no relationship between these factors (e.g., Constantino et al., 1993; Dorn et al., 2009; Maras et al., 2003; van Bokhoven et al., 2006).

In the last decade, researchers have shifted from the examination of the correlational and linear relationship between testosterone and aggression to the examination of testosterone as part of a dynamic process, where mutually influential and interrelated phenomenon are entered into prediction models to account for the interactive role of testosterone and social systems (Booth et al., 2003; Foshee, Ennett, Bauman, Granger, Benefield, Suchindran, et al., 2007; Rowe et al., 2004; Updegraff, Booth, & Thayer, 2006) and the interactive role of testosterone and other hormonal processes (Popma, Vermeiren, Geluk, Rinne, van den Brink, Knol, et al., 2007) on human behavior. This shift has taken place in the child and adolescent literature as well as the adult literature (Booth et al., 2003; Cohan, Booth, & Granger, 2003; Popma et al., 2007; Updegraff et al., 2006). In line with this shift in the literature, the current study examines social systems and cortisol as moderators of the relationship between testosterone and aggressive and delinquent behavior in adolescent males.

In this investigation, it is argued that the interaction between testosterone and social factors (i.e., delinquent peer affiliation and poor parenting quality) and the interaction between testosterone and cortisol are important in explaining individual differences in aggressive and delinquent behavior.

Theoretical Background

Terri Moffitt's (1993) life-course persistent offender model of the development of aggression and delinquency is one theoretical model that may be used to help conceptualize the role of testosterone in relation to aggression and delinquency. Moffitt (1993) proposed that life-course persistent offenders begin offending early in childhood and continue their antisocial behavior throughout development and into adulthood. Moffitt proposed that life-course persistent offenders have neuropsychological deficits that leave them vulnerable to develop a difficult temperament; and that this difficult temperament predisposes them to behavior problems if it occurs in conjunction with a high-risk social environment.

Although Moffitt focused on executive functioning deficits in her theory, other biological risks including hormonal differences are consistent with the life-course persistent offender model. In fact, previous research suggests that high testosterone levels influence brain areas associated with emotion and executive functioning such that individuals with high testosterone levels have a cognitive bias for attention to negative stimuli (Hermans, Ramsey, & van Honk, 2008; van Honk, Tuiten, van den Hout, Koppeschaar, Thijssen, de Haan, et al., 2000) and may be more withdrawn and difficult as children (Strong & Dabbs, 2000). Moffitt's theory has received impressive support in the literature (e.g., Frost, Moffitt, & McGee, 1989; Henry, Moffitt, & Silva, 1992;

Moffitt, 1990a, 1990b; Moffitt, 1993; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Moffitt & Henry, 1989; Moffitt & Silva, 1988a, 1988b, 1989; White, Moffitt, Earls, Robins, & Silva, 1990; White, Moffitt, & Silva, 1989, 1992). Consistent with Moffitt (1993), it is hypothesized that there will be a stronger relationship between high levels of testosterone and aggression and delinquency among those with at-risk social environments (e.g., poor parenting style and affiliation with deviant peers) than among those with high levels of testosterone and non-at-risk social environments.

Mazur and Booth's (1998) basal model of testosterone can also be used to guide the measurement and conceptualization of the testosterone-aggression relationship. This model assumes that only one measurement of testosterone is necessary because there is little fluctuation of testosterone over time and across situations. Consistent with this model, it is hypothesized that baseline levels of testosterone, measured at a single time point, will be related to behavior at that same time point. Incorporating Moffitt's theory, it is hypothesized that the testosterone aggression relationship at any single time point in development will be strongest among youth who are exposed to at-risk social environments.

Parenting as a Moderator of Testosterone and Aggression and Delinquency

Following a thorough review of the literature, Moffitt (1993) proposed that a difficult temperament that results from biological vulnerabilities makes it hard for a parent to respond to their child in a nurturing manner. In fact, she describes early interactions as negative, which later leads to parental distance and a lack of parental involvement.

Moffitt (1993) purports that the interaction between biological vulnerabilities and a high-risk environment such as the presence of poor-parenting is a major stepping-stone to a life-course path of offending. Additional research by Patterson and colleagues at the Oregon Social Learning Center support this hypothesis (Patterson, 1982).

To date, one study has specifically examined parenting as a moderator of the relationship between testosterone and aggression. In a sample of nine year old boys enrolled in a treatment outcome study for disruptive behavior disorder (DBD), Dorn et al. (2009) failed to find significant results for the interaction between testosterone and parenting and testosterone and parent-child relationship quality as predictors of DBD. The authors encouraged future research using samples of children with more representative testosterone levels for their ages. Of note, the authors reported lower levels of testosterone among the group of children with disruptive behavior disorder as compared to children without disruptive behavior disorder.

In a less direct measure of parenting, Urdy (1990) examined the moderating role of family factors consistent with social control on the testosterone-aggression link. More specifically, he examined social order variables including attachment to household rules (e.g., completion of chores), affiliation with community activities (i.e., church attendance, involvement in sports), and commitment to education (e.g., value of grades, value of high education) in a community sample of boys in grades 8 through 10. Urdy (1990) found that a combined scale of affiliation with church and attachment to household rules moderated the relationship between testosterone and problem behavior. He also found involvement in school (measured as the value of one's grades) to moderate the relationship between testosterone and problem behavior.

In each of these cases a more positive social environment dampened the association between testosterone and behavior problems.

Other studies have examined parenting or parent-child relationship quality as a moderator of testosterone and risk-taking behaviors. For example, in a community sample of 4th and 5th graders and 9th and 10th graders, Booth et al. (2003) examined parent-child relationship quality as a moderator of the relationship between testosterone and risky behavior. The authors found that for boys, mother-son relationship quality and father-son relationship quality moderated the relationship between testosterone and risk taking: boys with higher levels of testosterone participated in less risk behaviors if relationship quality with either parent was high. Testosterone alone did not predict risk-taking behavior.

Peer Affiliation as a Moderator of Testosterone and Aggression and Delinquency

Adolescence is a period of development marked by less time spent with parents and increased affiliation with peers. Research suggests that deviancy training among peers contributes to increasing levels of aggression and delinquency (Snyder, 2002). Because the sample for the current study is drawn from an adolescent population, it was important to also examine the role of testosterone and peer-affiliation in predicting aggressive behavior.

To date, only two studies have examined the interaction between testosterone and deviant peer affiliation to predict aggressive outcomes (Dorn et al., 2009; Rowe et al., 2004).

Using a subsample from the Great Smoky Mountain Study, Rowe et al. (2004) found that for youth with deviant peer affiliations, high testosterone was related to nonaggressive conduct disorder; whereas for youth without deviant peer affiliations, high testosterone was related to leadership. The researchers did not find testosterone to be related to physical aggression in this study, but methodological weaknesses may have impacted the results. For example, the researchers used single item scales to measure both peer deviancy and peer leadership.

Within the context of a comparative study of children with a diagnosis of DBD versus healthy controls, Dorn et al. (2009) examined peer delinquency as a moderator of the relationship between testosterone and aggression and the relationship between hormonal precursors of testosterone (i.e., DHEA and androstenedione) and aggression. They found a group (DBD versus healthy controls) by delinquent peer group interaction for the prediction of hormone levels, such that androstenedione was higher in healthy controls who associated with a delinquent peer group. This finding was surprising given that the DBD group had higher levels of androstenedione than the healthy control group.

Although no other study has examined peer relationships as a moderator between testosterone and aggression, several researchers have examined the testosterone-peer relationship interaction as it predicts other deviant behaviors. For example, Vermeersche et al. (2008a) found that older 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 cigarette involvement for boys who engaged with peers who used cigarettes (Foshee et al., 2007). Similar results were found for alcohol involvement.

In another study conducted by Bauman, Foshee, & Haley (1992), researchers examined the interaction between testosterone and peer smoking and its effect on adolescent smoking behavior. They found a stronger relationship between testosterone and smoking behavior for participants who had friends who smoked than for participants who did not have friends who smoked.

The above studies were conducted to test the hypothesis that affiliation with deviant peers increases the existing hormone related predisposition toward aggressive and other problem behaviors (Rowe et al., 2004; Vermeersch et al., 2008a). Given the limited empirical literature in this area, the current study can make a significant contribution. Specifically, one aim of this study was to examine whether social environment factors (i.e., parenting and peer associations) moderate the testosterone-aggression relationship in a treatment sample of adjudicated adolescent boys with a history of persistent aggression and delinquency.

Cortisol and Aggression

It has been theorized that children with low cortisol levels have reduced autonomic activity, resulting in fearlessness or sensation-seeking; and that fearlessness or sensation seeking leaves a child vulnerable to aggressive behavior (Raine, 1993).

The fearlessness interpretation of low arousal suggests that if a child has impaired physiological responding, then punishment or threat of punishment will be ineffective in socializing that child. The sensation-seeking interpretation of low arousal is complementary to the fearlessness hypothesis and purports that, through acts of risky behavior, children with low arousal are motivated to seek stimulation from the environment in an effort to decrease the unpleasant feelings of under-arousal.

Although there are solid conceptualizations of the relationship between low basal cortisol levels and aggressive behavior in children, currently research on this relationship is mixed (see van Goozen, Fairchild, Snoek, & Harold, 2007 for a review). A recent meta-analysis of this literature found a moderate effect size for the relationship between low basal cortisol and problem behavior (d = -0.40) when methodological variations were controlled (van Goozen et al., 2007).

In addition to the methodological limitations in this area, it is possible that findings concerning the relationship between low basal cortisol and problem behavior have been inconsistent because researchers have failed to account for additional hormonal factors that regulate and are regulated by cortisol. In a review of the hypothalamic-pituitary-adrenal axis (HPA) and hypothalamic-pituitary-gonadal (HPG) axis literatures, Viau (2002) concluded that there is sufficient support for the hypothesis that testosterone down regulates the HPA axis. Related to the above argument, Viau (2002) discussed evidence that HPA activity varies as a function of status in which subordinates show high cortisol and decreased testosterone under stress, whereas social dominants exhibit decreased cortisol and increased testosterone in response to stress. Terburg, Morgan, & van Honk (2009) reviewed the literature and found similar results.

Terburg et al. (2009) added a hypothesis for how a high testosterone, low cortisol profile may be related to aggression and delinquency. They argued that high levels of testosterone down regulate the HPA axis, leaving one vulnerable to missed opportunities for punishment cues, and demonstrated that a high level of testosterone is related to a hostile attribution bias as well as poor impulse control. This combination of factors increases the risk for aggressive and delinquent behavior.

To date, two studies have examined the interaction between testosterone and cortisol in relation to aggressive outcomes in children and adolescents. Specifically, Popma et al. (2007) found that in a sample of adolescent boys between the ages of 12 and 14 who were referred to a delinquency diversion program, cortisol significantly moderated the relationship between testosterone and aggressive behavior, such that there was a significant positive relationship between testosterone and aggressive behavior for children with cortisol levels one standard deviation below the mean of the sample. This relationship did not exist for adolescents with cortisol one standard deviation above the mean value of the sample.

In contrast, in a sample primarily consisting of boys between the ages of 7 and 14 who were arriving at a 7-week treatment program for children with disruptive disorders, Scerbo et al. (1994) found that the relationship between testosterone and teacher-reported ADHD symptoms was stronger when children had higher levels of cortisol. No interaction effects were found for aggressive behavior. More research is needed to better understand these mixed results. Of note, Scerbo et al. (1994) collected saliva samples for hormone assay in the morning, while Popma et al. (2007) collected saliva samples in the afternoon.

Some research seems to indicate that testosterone levels are more variable in the morning (Dabbs, 1990). Additionally, Scerbo et al. (1994) reported significant comorbidity in their sample, whereas Pompa et al. (2007) did not report comorbid diagnoses.

Rationale for the Current Study

The research literature on the relationship between testosterone and child and adolescent aggression and delinquency is inconclusive (Archer, 1991; Book et al., 2001). Growing evidence suggests that the application of biosocial models may help to elucidate these findings (e.g., Booth et al., 2003; Fisher, Stoolmiller, Gunnar, & Burraston, 2007; Rowe et al., 2004; Sapolsky, 1993; van de Wiel, van Goozen, Matthys, Snoek, & England, 2004). More specifically, recent research on child development suggests that environmental and family factors influence physiology and risk for poor outcome (Booth et al., 2003; Rowe et al., 2004). Thus, this study investigated the role of peer-affiliation and parenting style as moderators of the relationship between testosterone and aggressive and delinquent behavior. Research also suggests that the simultaneous investigation of testosterone and other hormones that influence, or are influenced by, testosterone may add valuable information to the study of the relationship between testosterone and poor outcome (Popma et al., 2007; Terburg et al., 2009). Thus, this study also investigated the role of low cortisol levels as a moderator of the relationship between testosterone and aggressive and delinquent behavior. The specific hypotheses were:

1) Baseline measures of testosterone will be more strongly associated with aggression and delinquency among youth with deviant peer affiliations;

2) Baseline measures of testosterone will be more strongly associated with aggression and delinquency among youth with a poor parenting style; and

3) Baseline measures of testosterone will be more strongly associated with aggression and delinquency among youth with low baseline cortisol levels.

In all cases, the association between testosterone and aggression and delinquency was hypothesized to be positive in the "at-risk" groups.

Method

Overview

The data for this study were collected as part of Time 1 (pre-treatment) procedures for a collaborative five-year longitudinal treatment outcome study of differential response to Multisystemic Therapy (MST), conducted in Denver, Colorado. This study was approved by the Human Subjects Institutional Review Board at the University of Colorado, the Medical University of South Carolina, and Emory University.

Participants

Participants for this study included 121 adolescent boys (M =15.44, SD = 1.28) and their caregivers. Fifty one percent of the participants were European American, 26% of the participants were Latino American, 20% of the participants were African American, and 3% of the participants identified as "other." Median family income was $27,000. Sixty percent of families reported additional income in the form of financial assistance (e.g., Food Stamps, Aid to Families with Dependent Children, Section Eight Housing, other Housing Assistance, Women Infants and Children (WIC), Supplemental Security Income). Average educational attainment for primary caregivers was 13 years.

Sample Selection

Families were referred by social service agencies and the juvenile justice system to one of four MST provider organizations in the Denver metropolitan area: Savio House (26.4%), University of Colorado Hospital Outpatient Community-Based Services (5.8%), Jefferson Center for Mental Health (11.6%), or Synergy Center for Mental Health (56.2%). Mean level of testosterone for participants from each provider organization is provided in Table 1; these measures did not differ significantly across the four organizations. These values are similar to those reported by other researchers (e.g., Granger et al., 2004; van Bokhoven et al., 2006).

Inclusion criteria included a family with a son between the ages of 12 and 17 years who (1) was referred for MST services from social service agencies or juvenile justice courts for substance abuse, property offenses or crimes against another person; (2) was living in the caregivers home for at least a month prior to treatment onset, with no immediate plans for placement elsewhere; and (3) had at least one caregiver willing to participate in MST.

Eligibility to participate in this study was determined at treatment onset. After being identified, therapists' provided families with a cursory overview of the study and with the option to receive additional information. Youth and families who were interested in receiving more information were contacted by research assistants and were provided with detailed information about the study, the time frame, and compensation. For interested families, the first assessment appointment was scheduled in the families' home.

Procedures

All assessments were completed within the home. Families were assessed, on average, within 23 days of referral to the project. Following an explanation of the study and written caretaker consent (as well as assent by the youth), the caretaker and youth completed measures of adolescent aggression, adolescent delinquency, and parenting practices. The youth completed additional questionnaires of deviant peer affiliation. All questionnaire assessments were completed on a laptop computer. Following these measures, a saliva sample was obtained from the youth. Next, the youth completed additional questionnaires including those that provided health and pubertal status information. After completing the procedures, families were debriefed and given 75 dollars to compensate them for their time.

Testosterone and Cortisol Collection

A saliva sample was collected for purposes of assaying testosterone and cortisol. Although the concentration levels of testosterone and cortisol are lower than those found in serum (Malamud & Tabak, 1993; Ohzeki, Manella, Gubelin-DeCampo, & Zachman, 1991), methods have been advanced to increase immunoassay sensitivity (Granger, Schwartz, Booth, & Arentz, 1999) and several studies shed light on procedures that maintain reliability and validity of salivary testosterone and cortisol levels (e.g., Dabbs, 1990; Dabbs, 1991; Granger et al., 1999; Granger, Shirtcliff, Booth, Kivlighan, & Schwartz, 2004; Hibel, Granger, Cicchetti, & Rogosch, 2007; Schwartz, Granger, Susman, Gunnar, & Laird, 1998; Shirtcliff, Granger, & Likos, 2002).

Additionally, researchers have found that salivary levels of testosterone and cortisol are highly correlated with serum levels of testosterone and cortisol in children and adolescents (Sannikka, Terho, Suominen, & Santti, 1983).

Testosterone and cortisol follow a similar diurnal pattern – levels are highest in the early morning, levels begin to decrease but are still high before noon, and demonstrate a steep decline after noon – allowing for the collection of one saliva sample at the same time of day for purposes of assaying both testosterone and cortisol (Dabbs, 1990; Granger et al., 2003; Hibel et al., 2007; Kahn, Rubinow, Davis, Kling, & Post, 1988; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001; Stansbury & Gunnar, 1994).

Saliva was collected from the youth by having them passively drool directly into a specimen tube to the level of 1cc. Neither cotton swabs nor polyester swabs were used to collect saliva because these methods have been related to artificially high levels of testosterone and cortisol (Dabbs, 1991; Granger et al., 1999; Granger et al., 2004; Schwartz et al., 1998; Shirtcliff et al., 2001). Following procedures outlined by Shirtcliff et al. (2002), if a participant reported that they might have a difficult time expectorating the desired level of saliva, they were allowed to drink some water at the start of the assessment, more than 5 minutes before they provided their first saliva sample.

Samples were stored in an adult size lunch box with one ice pack for the duration of the in home assessment and then transported to the research lab. The ice pack creates refrigeration temperatures. Saliva can be refrigerated for up to 1 week before bacteria growth disturbs testosterone levels (Granger et al., 2004).

Immediately upon arriving to the research lab, participants' vials were frozen and stored at -20°C for an average of three months before being mailed to the Yerkes Endocrine Core Laboratory for assay. Research evidence suggests that PM saliva samples for the purpose of assaying testosterone may be stored at -20°C (Granger et al., 1999; Granger et al., 2004). Saliva storage at -20°C is customary for the purpose of assaying cortisol (e.g., Granger, Serbin, Schwartzman, Lehoux, Cooperman, & Ikeda, 1998). The Center for Disease Control guidelines were followed for the transport of biological specimens, and samples were sent overnight delivery on dry ice in batches to the Endocrine Core Laboratory at the Yerkes National Primate Research Center of Emory University.

Testosterone and Cortisol Immunoassay Protocols

Upon arriving to Yerkes, saliva samples were stored at -20 °C. On the day of assay, samples were thawed and vortexed, then centrifuged to remove particulate matter. Salivary cortisol was assayed using an enzyme immunoassay kit (DSL, Webster, Texas), catalogue number DSL-10-67100. This assay procedure has an analytical sensitivity of 0.10 mg/dl, using 25 ml of saliva. The intra- and inter-assay coefficient of variation is 4.1 and 7.2%, respectively. 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 had an analytical sensitivity of 2.0 and used 200 ul of saliva. The intra- and inter-assay coefficient of variation was 3.41 and 19.16%, 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 (for cortisol and testosterone) was used in analyses.

Ideally, testosterone is assayed before cortisol because of testosterone degradation due to repeated freeze thaw. Due to a change in study protocol during the study, this order of assay was not consistently maintained. To examine batch-to-batch variation in testosterone assay values, one-way ANOVA analyses were conducted whereby participant assay number was entered as the independent variable and testosterone was entered as the dependent variable. Results revealed a significant difference among assay number on their levels of testosterone, $F(7, 95) = 5.69$, $p < .01$. Assay number (dummy coded) was therefore entered as a statistical control in all analyses.

Measures

Biological Measures

Testosterone Determination

Total levels of basal testosterone provided a continuous scale of measurement and served as the independent variable in all analyses. Research assistants noted conditions previously found by other researchers to have an influence on testosterone levels: eating food with high acidity or drinking beverages with caffeine immediately before saliva collection, noticeable oral injury, poor oral hygiene, teeth brushing, smoking, or gum chewing. All of these factors have been shown to artificially inflate testosterone levels. Thus, participants were excluded if they were noted as having one of these conditions (N=9). Samples with testosterone levels equal to or greater than three standard deviations from the mean were considered outlying values and were excluded from the current analyses (N=2).

Cortisol Determination

Total levels of basal cortisol provided a continuous scale of measurement and served as a moderator of the relationship between basal testosterone and aggressive and delinquent behavior. Samples with cortisol levels equal to or greater than three standard deviations from the mean were considered outlying values and were excluded from the current analyses (N =3).

Social Moderators

Peer Delinquency Scale (PDS)

In this study, the PDS (Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1994) was used to assess delinquent peer affiliation. The PDS provides a measure of the ratio of the number of the participants' friends who engage in delinquent acts and/or substance use to the number of their friends who do not. The original scale contains 15 items for which youth are asked to report on behavior during the past 30 days. Response choices for each question include: "0-None of them," "1-Few of them," "2-Half of them," "3 Most of them," and "4-All of them." For purposes of the current study, one additional substance use item was added: "During the past 30 days, how many of your friends have smoked cigarettes?" Thus, the PDS for the current study contained 16 items. The alpha coefficient for this scale was 0.93. Youth delinquent peer affiliation scores as measured on the PDS was calculated by summing youth response to all items, providing a continuous scale of measurement. A higher score indicated that participants' friends engaged in more delinquent activity. Please see Appendix A for a copy of this measure.

Loeber et al. (1994) reported a Cronbach's alpha of .84 with a sample of 500 11-year-old boys. In a separate study, Blackson and Tarter (1994) reported a Cronbach's alpha of .76 with a sample of 130 10, 11, and 12 year old boys. Content validity is suggested, as the PDS was developed from corresponding items of the Self-Reported Delinquency Scale (SRD) (Elliott et al., 2005). Good concurrent and predictive validity has also been reported (Keenan, Loeber, Zhang, Stouthamer-Loeber, and Van Kammen, 1995).

Alabama Parenting Questionnaire (APQ)

A modified form of the Alabama Parenting Questionnaire (Loeber, Stouthamer-Loeber, van Kammen, & Farrington, 1991) was used to assess parenting. In its original format, the APQ is a 42 item, paper and pencil measure that examines parental involvement (10 items, e.g., you ask your child about his/her day at school), positive parenting (6 items, e.g., you compliment your child when he/she does something well), poor monitoring / supervision (10 items, e.g., your child is out with friends you don't know), inconsistent discipline (6 items, e.g., you threaten to punish your child and then do not actually punish him/her), corporal punishment (3 items, e.g., you slap your child when he/she does something wrong), and other parenting practices (7 items, e.g., you give your child extra chores when he/she is misbehaving). Our modified format excluded corporal punishment items, used rewording to focus on the primary caregiver rather than both parents, and was administered on a laptop rather than paper and pencil format. Spanish translations were provided for the participant caregivers who reported Spanish as their first language.

Both child report of parenting style and caregiver report of parenting style were used in this study. These scales were scored by calculating a total score for each subscale and then combining the subscales, providing one continuous measure of quality of parenting. The coefficient alphas were 0.91 for the youth report and 0.66 for the caregiver report of quality of parenting. Similar reliability statistics have been reported by others (Dadds, Maujean, & Fraser, 2003; Shelton, Frick, &Wootton, 1996).

Aggressive and Delinquent Outcome

Child Behavior Checklist – Ages 6-18 (CBCL)

Aggressive and delinquent symptoms were assessed by caregiver report using ratings on the CBCL (Achenbach, 1991), which is one of the most well-validated measures of child behavior functioning (Achenbach, 1991; Achenbach & Edelbrock, 1983; Achenbach & Rescorla, 2001). The CBCL consists of 113 behavior problem items. Broadly, this rating scale taps symptoms of Internalizing Problems (e.g., depression and anxiety), Externalizing Problems (e.g., aggression, conduct problems, attention-deficit/hyperactivity symptoms, and delinquency), and Total Behavior Problems (i.e., total scores on internalizing and externalizing problems). This study used the Aggression and Delinquency subscales. Responses to items ranged from zero to two: "0-Not True", "1-Sometimes True," and "2-Very True or Often True." Caregivers were asked to describe behaviors of the youth that were occurring at the time of the study or 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 aggressive and delinquent behavior. *T*- scores higher than 60 were considered clinically significant levels of aggressive or delinquent behavior.

Forty-six percent of the sample received a *T*-score of 60 or above on the measure of CBCL Aggression; and 77% of the sample received a *T*-score of 60 or above on the measure of CBCL Delinquency. In this study, the aggressive subscale had a coefficient alpha of .93 and the delinquency subscale had a coefficient alpha of .85.

Self-report Delinquency (SRD)

The SRD (Elliot, 1994; Elliot, Ageton, Huizinga, Knowles, & Canter, 1983; Elliot, Ageton, Hunter, & Knowles, 1976) is a 47-item scale that measures antisocial behavior and includes subscales that pertain to violent offending (e.g., attacking someone with the intention of hurting them, forcing another person to have sex), general delinquency (i.e., covert and overt delinquency and violent crimes), property offenses (e.g., purposely damaging or destroying property belonging to parents, other family members, or school), and status offenses –defined as an offense that is illegal for a youth, but would otherwise be legal if the youth was an adult (e.g. alcohol use, not attending school, staying out late, and running away). In its original format, youth are asked to report on these behaviors over the last year.

For the purposes of the current study, several modifications were made to this measure. First, the general delinquency total score was the only measure of aggression and delinquency used. Second, youth were asked to report on their behavior over the last 30 days (e.g., "How many times in the past 30 days have you:"). Third, seven items related to drug use were excluded. Fourth, frequency variations for item response choices differed. The original measure measured behaviors in ranges of 0-24 times, 25-49 times, 50-199 times, and 200 plus times. In this study delinquent behaviors were reported in exact counts in the range of 0-99, or as the category of 100 plus.

Scores were derived by calculating the total number of youth problem behaviors reported in each category of offense. High scores indicated more involvement in general delinquency.

Researchers have found test-retest reliability for this measure to range from .85 to .99, using intervals ranging from less than one hour to over two months (Patterson & Loebber, 1982; Hindelang, Hirschi, & Weis, 1982). Huizinga & Elliott (1986) found that there were no significant differences across sex, race, class, place of residence, or delinquency levels for estimates of test-retest reliability. Huizinga et al. (1986) also found that the majority of individuals who have been arrested self-report their delinquent behavior, and the majority of offenses they commit are also reported.

For the current study, the general delinquency total score was positively skewed. To approximate a normal distribution of scores, outliers, defined as equal to or greater than three standard deviations above the mean, were removed (N=2) and a log transformation was performed (after determining that there were no curvilinear relationships to the independent variables). Research has shown that the SRD scale is the best validated of the self-report delinquency scales (Henggeler, 1989). Please see Appendix B for a copy of this measure. For this study, the general delinquency scale had a coefficient alpha of 0.90.

Control Variables

Health Questionnaire

First, the youth and their caregiver were asked about the youth's physical health and typical eating and sleeping patterns in the 24 hours before the assessment.

Consistent with previous research, eating, sleeping (i.e., how long the youth had been awake), and medication schedules were examined in relation to salivary testosterone and basal cortisol levels (Granger et al., 2004; Hibel et al., 2007). Preliminary analyses revealed significant results for the association between cortisol levels and the time of day that cortisol was collected, $r = -.21$, $p < .05$ and between cortisol levels and the number of hours that participants were awake before collection, $r = -.26$, $p < .01$. Time of day was no longer a significant predictor of cortisol when number of hours awake was controlled. Analyses examining cortisol therefore controlled for number of hours the participant was awake when they provided the sample. Time of day ($r = .04$, $p = .71$) and number of hours since awakening ($r = .01$, $p = .92$) were not associated with testosterone levels in this study, and all other associations between health, eating, and sleeping and youth hormone levels were non-significant.

Peterson Pubertal Development Scale (PPDS)

The PPDS 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). For purposes of the current study, a total puberty score was calculated by summing the items and dividing by five, to maintain the original metric.

In this study, age and puberty were not as highly correlated as reported in previous studies ($r = .40$, $p < .00$), 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, $r = .36$, $p < .00$.

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, $r = -.01$, $p = .90$. When controlling for puberty, the significant results for the association between testosterone and age remained significant, $r = .31$, $p < .01$. 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. Please see Appendix C for the combined *Health Questionnaire* and *PPDS.*

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; Muzar, 1995; Tremblay, Schaal, Boulerice, Arseneault, Soussignan, Paquette, et al., 1998). The association between testosterone, height, weight, and BMI was assessed. As shown in Table 2, results revealed no significant associations with testosterone.

Analytic Procedures

Overview

Multiple regression analyses were used to test the hypotheses in this study. For main effect analyses, covariates and confounding variables were entered in Step One and testosterone was entered in Step Two. For interaction analyses, Step One included the main effect terms (centered), the covariates, and the confounding variables. Step Two included the two-way interaction between testosterone and moderating terms (i.e., the product of the centered linear terms). For significant interactions, a median split of the moderating term was performed and separate regression analyses were completed for those above and below the median value.

Results

Descriptive Statistics and Bivariate Relationships Between Variables Used in the Analyses

Table 3 presents descriptive statistics for the sample used in the analyses. Table 4 presents the Pearson correlation matrix between the independent and dependent measures used in the analyses. None of the inter-relationships between testosterone and the moderating variables (i.e., basal cortisol, deviant peer affiliation, and parenting) were statistically significant. The correlation between testosterone and CBCL aggression is significant and negative, however, this is due to the confounding effect of age; younger males have lower testosterone and higher scores on CBCL aggression as noted below.

The inter-relationships among the moderator and dependent variables, as well as the inter-relationships among the dependent variables were in the expected direction –as the number of deviant peer affiliations increased, the quality of parenting as reported by the caregiver and youth decreased. There was a significant positive association between youth and caregiver report of parenting style.

Also, as the number of deviant peer affiliations increased, the level of self-reported aggression and delinquency (i.e., SRD general delinquency scores) and the level of caregiver report of aggression and delinquency increased. As the quality of youth-reported parenting style decreased the level of self-report and caregiver report of aggression and delinquency increased. Quality of parenting as reported by the caregiver was significantly negatively associated with caregiver and youth report of aggression and delinquency, as well as youth report of delinquency.

As expected, the outcome variables were positively associated with one another. As caregiver report of aggression increased, caregiver report of delinquency increased. These two variables were expected to be highly correlated given they are from the same scale. In addition, youth report of aggressive and delinquent behavior was significantly correlated with caregiver report of aggressive and delinquent behavior.

Table 5 presents the Pearson correlation matrix between continuous demographic variables (i.e., age, puberty) and dependent variables used in analyses. Table 6 presents the Spearman correlation matrix between the categorical demographic variable (i.e., ethnicity) and the dependent variables (including the one categorical dependent variable) used in the analyses. These correlation matrices were split into two tables for ease of reading. Puberty was not significantly correlated with any of the dependent variables. Age was significantly negatively correlated with CBCL aggression: the younger the youth, the higher the caregiver scored the youth on aggressive behavior. As a result, age was controlled in all analyses. Ethnicity was significantly correlated with caregiver report of aggressive and delinquent behavior: European American youth were rated as more aggressive and delinquent by their caregiver. Hence, ethnicity was statistically controlled in analyses of caregiver report of aggressive and delinquent outcome.

Outlier Values

As with the determination of the testosterone and cortisol variables, one dependent variable required the removal of two influential outlier values, with an outlier defined as an individual value more than three standard deviations above the mean value SRD general delinquency (N=2).

Cases were also excluded where participants did not report on variables of interest (N varies depending on the measure of interest). Thus, sample size varies by analyses and is noted throughout.

Direct Effects of Testosterone on Aggressive and Delinquent Outcome

There were no statistically significant results for basal testosterone predicting aggressive or delinquent symptoms: testosterone did not predict SRD general delinquency scores, $\Delta F(1, 97) = 1.23$, $\Delta R^2 = .01$, $\beta = .00$, $p = .27$ (see Table 7), CBCL aggression, $\Delta F(1, 96) = 2.44$, $\Delta R^2 = .02$, $\beta = -.04$, $p = .12$ (see Table 8), or CBCL delinquency, $\Delta F(1, 91) = 0.72$, $\Delta R^2 = .01$, $\beta = -0.02$, $p = .40$ (see Table 9).

Moderating Effects of Basal Cortisol

The hypothesis that baseline measures of testosterone would be more associated with aggression and delinquency among youth with low baseline cortisol levels was not supported. The interaction between testosterone and cortisol did not significantly predict SRD general delinquency, $\Delta F(1, 90) = 0.31$, $\Delta R^2 = .00$, $\beta = -.00$, $p = .58$, CBCL aggression, $\Delta F(1, 89) = 2.96$, $\Delta R^2 = .03$, $\beta = .18$, $p = .09$, or CBCL delinquency, $\Delta F(1, 84) = 1.79$, $\Delta R^2 = .02$, $\beta = .13$, $p = .18$).

Moderating Effects of Peer Affiliation

The hypothesis that high basal testosterone would be more associated with aggressive and delinquent outcome among individuals with deviant peer affiliations was not supported. The interaction between deviant peer affiliation and basal testosterone was not significant in predicting SRD general delinquency, $\Delta F(1, 94) = 0.20$, $\Delta R^2 = .00$, $\beta = 0.00$, $p = 0.66$, CBCL aggression, $\Delta F(1, 93) = 0.76$, $\Delta R^2 = .01$, $\beta = -0.00$, $p = .34$, or CBCL delinquency, $\Delta F(1, 88) = 0.10$, $\Delta R^2 = .00$, $\beta = -0.00$, $p = 0.75$).

Moderating Effects of Parenting

The hypothesis that youth with high basal testosterone would be more associated with aggressive and delinquent outcomes among youth with low-quality parenting was not supported. Testosterone did not significantly interact with either child report or caregiver report of parenting in predicting SRD general delinquency [($\Delta F(1, 94) = 0.20$, $\Delta R^2 = .00$, $\beta = 0.00$, $p = .87$; $\Delta F(1, 94) = 1.78$, $\Delta R^2 = .02$, $\beta = 0.00$, $p = .19$; respectively)], CBCL aggression [($\Delta F(1, 93) = 0.92$, $\Delta R^2 = .01$, $\beta = 0.00$, $p = .34$; $\Delta F(1, 94) = 0.01$, $\Delta R^2 = .00$, $\beta = 0.00$, $p = .94$; respectively)], or CBCL delinquency, [($\Delta F(1, 88) = 0.00$, $\Delta R^2 = .00$, $\beta = -0.00$, $p = .95$; $\Delta F(1, 89) = 0.00$, $\Delta R^2 = .00$, $\beta = 0.00$, $p = .99$; respectively)].

Discussion

Previous investigations of the relationship between testosterone and aggression and delinquency have produced mixed results. The primary purpose of this study was to contribute to, and attempt to clarify, this literature by using a biosocial model to investigate the interactive role of testosterone and social context as predictors of adolescent aggression and delinquency in males. Consistent with Moffitt's (1993) biosocial model of life-course persistent offending, it was expected that there would be a significant association between high levels of testosterone and aggressive and delinquent behavior among adolescent boys in at-risk social environments. The results of this study do not support this biosocial model, and are inconsistent with studies that have found that parent-child relationship (e.g., Booth et al., 2003) and deviant peer affiliation (Rowe et al., 2004; Dorn et al., 2009) moderate the relationship between testosterone and youth problem behaviors.

Results also showed cortisol did not moderate the relationship between testosterone and aggression/delinquency. This too is inconsistent with previous investigations (Pompa et al., 2007).

Competing theories of the role of testosterone within social contexts (for a review see Archer, 2006; Mazur et al., 1998), suggest that information about the micro-level organization of social contexts is necessary for understanding the role of testosterone in predicting behavior. For example, research suggests that status within a peer group influences testosterone levels (Vaillancourt, deCatanzaro, Duku, & Muir, 2009). Parallel research suggests that levels of testosterone influence social group dynamics (Oyegbile et al., 2005, 2006) and when peer groups are unstable, the high testosterone –aggression link emerges, however, under stable conditions, the high testosterone – aggression link is not evident (Schaal et al., 1996). Studies that have narrowly defined the peer group context have noted more consistent findings for the testosterone-behavior link, in the predicted direction (Bauman et al., 1992; Foshee et al., 2007). Our measure of deviant peer affiliation may not have sufficiently captured the social context that is most relevant to a testosterone-aggression relationship. Future researchers should incorporate a measurement of the micro-level organization of deviant peer groups when investigating the role of peer affiliation as a moderator of the relationship between testosterone and aggression and delinquency.

Considering micro-level factors within parent-child relationships may also be important for understanding the moderating role of parenting.

One possible explanation for inconsistent results for the relationship between testosterone and aggression/delinquency is that testosterone may not be related to aggression, but rather dominance and sensation seeking (e.g., Metha et al., in press; Muzar et al., 1998). Extrapolating from the Challenge Hypothesis (for a review see Archer, 2006), Social Dominance hypothesis (Mazur et al., 1998), and Coercion theory (Patterson, 1982), one might expect youth with high levels of baseline testosterone to be more likely to engage in a process that challenges the dominance hierarchy of a parental relationship, and further, that high levels of baseline testosterone would be related to maintaining dominance when a parent attempts to challenge the dominance status of the youth. This suggests that the relationship between testosterone and aggression may only appear when the parent-child relationship is disrupted and the child's status as the dominant figure is challenged. It is possible, therefore, that a third factor such as parental compliance to therapy might be an important consideration in our biosocial model.

The current study also examined the interactive role of testosterone and cortisol as predictors of youth aggression and delinquency. In keeping with the current conceptualization of the role of cortisol and aggression (Susman, 2006), as well as theory and research on the interplay between testosterone and cortisol (Terburg et al., 2009; Viau, 2002), it was hypothesized that there would be a significant relationship between testosterone and aggression and delinquency for individuals with low levels of cortisol. This hypothesis was not supported.

These findings are consistent with research by Scerbo et al. (1994); however, these findings are inconsistent with Popma et al. (2007) who, with a similar sample, found a significant interaction in the predicted direction.

Popma et al. (2007) examined subtypes of aggressive behavior (i.e., overt aggression and covert aggression), as measured by the Buss-Durkee Hostility Inventory. The Buss-Durkee Hostility Inventory measures more of the attitudinal or cognitive components of aggression (i.e., questions about what happens when one gets upset). Scerbo et al. (1994) and the current study measured reports of overt aggressive behaviors. One possible interpretation of the difference between these studies is that the interaction between testosterone and cortisol may be more indicative of an aggressive attitude as opposed to aggressive behaviors per se (Terburg et al., 2009).

The current study failed to show a significant correlation between testosterone and basal cortisol. Though there is an increasing amount of interest in the relationship between testosterone and cortisol, our understanding about this relationship is incomplete. Terburg et al. (2009) hypothesized that the profile of high testosterone and low cortisol paralleled the negative feedback loop in the Behavior Inhibition System and Behavior Activation Systems. This suggests that changes in the environment may have a reciprocal relationship on the autonomic nervous system. Future research should incorporate factors that are capable of capturing changes in the environment; this would include moving away from cross-sectional studies toward longitudinal studies.

Limitations

Although this paper has many strengths, there are also several limitations that are important to note. First, it is possible that a restriction of range among the aggression and delinquency measures obscured additional findings. More specifically, given that this sample consisted of adjudicated adolescent males, levels of aggression and delinquency were restricted at the low end.

Relatedly, this study did not include a non-aggressive / non-delinquent comparison group. Third, the current study was a cross-sectional investigation of the relationship between testosterone and aggression. Although cross-sectional studies make theoretical assumptions about the role of testosterone on behavior, such a design limits the ability to examine reciprocal models of the relationship between testosterone and aggression. Fourth, testosterone levels were significantly different among three assay batches. Though this variability was statistically controlled in all analyses, the lack of significant findings may have been due to the possible unreliability of the testosterone measure.

Clinical Implications and Future Directions

Overall, the results of this study suggest that testosterone may not be a useful biological marker of aggressive behavior in the context of at-risk environments. It would be important, however, to more fully examine the biosocial model. It might be the case that research should be steered in a direction other than basal, cross-sectional designs, toward studies that are capable of testing new theoretical models. Mazur et al. (1998), for example, have proposed a reciprocal model for the role of testosterone on behavior which seems promising; in this model testosterone influences social context and social context, in turn, influences testosterone.

Study 2: A Longitudinal Analysis

The association between testosterone and aggression (in humans) appears to be mixed (Archer, 2006; Mazur et al., 1998; Ramirez, 2003). Some cross-sectional studies find that testosterone and aggression are positively related, some find they are negatively related, and others find no relationship between these factors (e.g., Constantino et al., 1993; Dorn et al., 2009; Maras et al., 2003; Ryan, 2009; van Bokhoven et al., 2006).

There are at least two possible explanations for the inconsistent findings in the literature to date. First, many of the existing studies vary widely in the methods used to recruit participants and the methods used to measure aggression and delinquency (Ramirez, 2003); only a subset of these studies may have produced valid findings. A second possible explanation is that there may not be a one-to-one association between gonadal hormones and behavior, whereby testosterone would simply have direct, concurrent activational effects on aggression and/or delinquency. Instead, testosterone may only be associated with aggression and/or delinquency under certain social or biosocial circumstances. In line with this explanation, researchers examining testosterone and aggression and delinquency have begun to examine the interactive role of testosterone and social systems (Booth et al., 2003; Dorn et al., 2009; Foshee et al., 2007; Rowe et al., 2004; Ryan, 2009; Updegraff et al., 2006; Urdy, 1990; Vermeersch et al., 2008a) and the interactive role of testosterone and other hormonal processes (Popma et al., 2007; Ryan, 2009) on human behavior. This shift has taken place in the child and adolescent literature, as well as the adult literature (Booth et al., 2003; Cohan, Booth, & Granger, 2003; Popma et al., 2007; Updegraff et al., 2006). Collectively, these studies suggest that the consideration of social context may be important for better understanding the testosterone-aggression/delinquency link.

Only a handful of longitudinal studies have examined the direct relationship between testosterone and aggression and/or delinquency. Longitudinal designs more reliably suggest that individuals with persistent aggression and/or delinquency evidence higher levels of testosterone.

Three studies have prospectively examined the relationship between testosterone and aggression (Maras et al., 2003; Schaal et al., 1996; van Bokhoven et al., 2006) and one has examined this link retrospectively (Dabbs et al., 1990). No study to date, however, has examined the interaction between testosterone and social factors or other hormonal processes within the context of a longitudinal design.

In addition to the longitudinal analysis of social context variables as moderators of the relationship between testosterone and aggression, it may be worthwhile to examine testosterone and aggression in the context of an intervention study. A parallel set of intervention and prevention studies have focused on cortisol in association with treatment outcome and/or child behavior problems. In these treatment studies, researchers have found that therapeutic services significantly alter cortisol levels and/or diurnal patterns to the degree that they then match levels and patterns of children not at risk for problem behaviors (Bakermans-Kranenburg, van Ijzendoorn, Pijlman, Mesman, & Juffer, 2008; Brotman, Gouley, Huang, Kamboukos, Fratto, & Pine, 2007; Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008; Dozier, Peloso, Lindhiem, Gordon, Manni, & Sepulveda, et al., 2006; Fisher et al., 2007; van de Wiel et al., 2004). Perhaps because of the more mixed literature on testosterone and aggression, no previous studies have examined changes in testosterone in the context of behavioral interventions.

To fill the existing gaps in the literature, the current study investigated whether testosterone in conjunction with social factors (i.e., delinquent peer affiliation and parenting) may be important in explaining individual differences in behavioral treatment response. The investigation of these biosocial relationships in the context of a therapy outcome study has several advantages.

For example, with a treatment outcome study, it is possible to investigate the relationship between a changing social context (brought on by the intervention) and the aggressive propensities of individuals with high levels of testosterone. Improving one's social context may protect a biologically high-risk individual against aggressive outcomes (Booth, Johnson, & Granger, 1999). Another advantage to testing youth over the course of treatment is that changes in youth behavior and changes in social context, as a result of therapy, may have a reciprocal and measureable influence on hormone levels that can be assessed systematically over time (Booth, Carver, & Granger, 2000). It is also possible to test whether particular testosterone –social context profiles might be predictive of poor treatment response to MST.

Thus, by using the same social context variables (i.e., deviant peer affiliation and parenting) as used in Study 1, the current study will extend the previous investigation of the biosocial model of the relationship between testosterone and social context to a longitudinal, treatment outcome study. In addition to the aforementioned rationale (as presented in Study 1) for examining deviant peer affiliation and parenting in the context of a biosocial model with testosterone, parenting and deviant peer association, are targeted mechanisms of MST – making this the ideal extension study.

Multisystemic Therapy

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 and visit the home 1-2 times a week, depending on individual need. The core principle of this therapeutic approach is strength focused skills building (Henggeler et al., 1998). Therapists individualize treatments with a focus on behavioral principles, while integrating additional forms of therapy 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., respect for hierarchy), and interventions at the peer level (e.g., decreasing association with deviant peers and increasing association with pro-social peers).

Biosocial Theory

The Challenge Hypothesis (Archer, 2006), the Social Dominance hypothesis (Mazur et al., 1998), and Patterson's (1982) coercion model may be combined to provide a biosocial explanation as to why individuals with high testosterone in at-risk environments may evidence a differential response to MST treatment. The goal of treatment focused on parent management skills (i.e., parenting) is to redistribute the power and control in the family system away from the youth and toward the parent by increasing effective parenting skills, therefore altering the parent-child relationship (Patterson, 1982). Parental attempts to gain control create a competitive interaction where the child's status as the dominant figure is challenged.

In keeping with the Challenge Hypothesis and the Social Dominance hypothesis, it may be the case that individuals with high testosterone are more motivated to maintain a dominance status when challenged (Mazur et al., 1998; Oyegbile et al., 2005, 2006), making it difficult for parents to be successful in changing the landscape of their interactions with their child. Parents' lack of success in implementing intervention techniques may be related to compliance in treatment, which in turn may be related to caregiver report of therapist adherence to treatment and treatment success (Oyegbile et al., 2005, 2006).

In addition, previous research suggests that those with high testosterone levels may have a bias in favor of negative stimuli such as sensation seeking and attention to negative verbal and non-verbal information during social interactions (Hermans et al., 2008; van Honk et al., 2000; Vermeersch, et al., 2008a; Wirth & Schultheiss, 2007). This vulnerability combined with deviant peers or poor parenting may make treatment more difficult; that is, deviant peers reinforce a youth's propensity for sensation seeking (Snyder, 2002), and youth with poor parenting may not recognize positive changes in a family therapy context, thereby influencing the potential effectiveness of treatment.

Cortisol as a Moderator of Testosterone and Changes in Aggression and Delinquency

In addition to examining a biosocial model of the relationship between testosterone and social context as predictors of changes in aggression and delinquency over the course of treatment, the current study also extends the cross-sectional investigation of the interaction between testosterone and cortisol (Popma et al., 2007; Scerbo et al., 1994; Study 1), by examining the interaction between testosterone and

cortisol and their relationship with changes in aggression and delinquency over the course of treatment. To date, no study has examined the interaction between testosterone and cortisol in the context of a longitudinal, treatment outcome study. A theoretical model presented by Terburg et al. (2009) suggests that the interaction between testosterone and cortisol may be related response to treatment.

Following a review of the literature HPG and HPA axises literatures, Terburg et al. (2009) theorize that high levels of testosterone down regulate the HPA axis, leaving one vulnerable to missed opportunities for punishment cues, and that a high level of testosterone is related to a hostile attribution bias, as well as poor impulse control. This combination of hormonal factors increases the risk for aggressive and delinquent behavior. In the context of treatment, a vulnerability to miss behavior-consequence associations and to have a bias for hostile cues could create a climate in which youth with high levels of testosterone and low basal cortisol are difficult to treat, leading to less therapeutic implementation success on the part of the therapist and the parent.

Rationale for the Current Study

Cross-sectional research on the relationship between testosterone and child and adolescent aggression and delinquency is inconclusive (Archer, 1991; Book et al., 2001). Longitudinal designs more reliably suggest that individuals with persistent aggression and/or delinquency evidence higher levels of testosterone (Maras et al., 2003; Schaal et al., 1996; van Bokhoven et al., 2006; Dabbs et al., 1990). Growing evidence suggests that the application of biosocial models may help to elucidate these findings (e.g., Booth et al., 2003; Fisher, Stoolmiller, Gunnar, & Burraston, 2007; Rowe et al., 2004; Sapolsky, 1993; van de Wiel, van Goozen, Matthys, Snoek, & England, 2004).

Current theoretical models for the relationship between testosterone and behavior suggest that longitudinal designs that incorporate a biosocial model in which the social environment is undergoing change may be particularly important (Mazur et al., 1998). Thus, the primary goal of this study was to assess the interaction between testosterone and social context (i.e., deviant peer affiliation and parenting) in relation to caregiver report of treatment adherence and behavioral response (i.e., changes in aggression and delinquency) in the context of MST. A secondary goal of this study was to examine how ecological and behavior change that occurs in the context of MST may be related to changes in youth testosterone levels.

Research also suggests that the simultaneous investigation of testosterone and other hormones that influence, or are influenced by, testosterone may add valuable information to the study of the relationship between testosterone and poor outcome (Popma et al., 2007; Terburg et al., 2009). Thus, this study also examined whether low cortisol levels moderated the relationship between testosterone and changes in aggressive and delinquent behavior. The specific hypotheses were:

Primary Hypotheses

1) Basal levels of testosterone measured at pre-treatment will interact with parenting quality, peer affiliation, and basal cortisol levels to predict mid-treatment and end of treatment caregiver report of therapist adherence to MST treatment.
2) Basal levels of testosterone will interact with pre-treatment levels of parenting quality, peer affiliation, and basal cortisol levels to predict changes in aggression and delinquency over the course of treatment.

3) The relationship between testosterone and aggressive and delinquent behavior over the course of MST treatment will be explored.

For all interaction hypotheses, the association between testosterone and changes in aggression and delinquency was hypothesized to be positive in the "at-risk" groups (i.e., those with deviant peers, poor parenting, low basal cortisol).

Method

Overview

The data for the current study were collected as part of a collaborative five-year longitudinal investigation on differential response to Multisystemic Therapy (MST), conducted in Denver, Colorado. This study was approved by the Human Subjects Institutional Review Board at the University of Colorado, the Medical University of South Carolina, and Emory University.

Participants

Participants for this study included 114 adolescent boys and their caregivers who completed a pre-treatment assessment and at least one other assessment (i.e., at mid treatment or end of treatment). Fifty percent of the participants were European American, 26% of the participants were Latino American, 19% of the participants were African American, and 4% of the participants identified as "other." Average educational attainment for primary caregivers was 13 years. At the time of the first assessment, adolescents' ages ranged from 12 to 17 years (M = 15.44, *SD* = 1.30). The primary caregivers' mean age was 44.43 years (*SD* = 9.66). Median family income was $27,000, with 60% of families reporting additional income in the form of financial assistance.

Sample Selection

Families were referred by social service agencies and the juvenile justice system to one of four MST provider organizations in the Denver metropolitan area: Savio House (26.3%), University of Colorado Hospital Outpatient Community-Based Services (6.1%), Jefferson Center for Mental Health (12.3%), or Synergy Center for Mental Health (55.3%). Inclusion criteria included a family with a son between the ages of 12 and 17 years who (1) was referred for MST services from social service agencies or juvenile justice courts for substance abuse, property offenses or crimes against another person; (2) was living in the caregivers home for at least a month prior to treatment onset, with no immediate plans for placement elsewhere; and (3) had at least one caregiver willing to participate in MST.

Procedure

Participants in this study were identified by their therapist as eligible for the study. Eligible families were contacted by phone by a research assistant and were provided with detailed information about the study, the time frame, and compensation. For those interested in participating, assessments were scheduled in the families' home.

The goal was to schedule all assessments on the same day, between the hours of 1 pm and 4 pm to control for the diurnal rhythm of testosterone and cortisol. Given the time commitment to participate in this study, however, research assistants were encouraged to be flexible when scheduling appointments with the families. All questionnaires were completed on a laptop computer. Youth completed measures of aggression, delinquency, deviant peer affiliation, and parenting. They also reported on their current pubertal status.

Caregivers completed measures of youth aggression and delinquency and parenting. Both the youth and caregiver provided health status information for the adolescent participants.

Pre-treatment (Time 1) assessment took place an average of 23 days after MST intake appointments, the first mid-treatment (Time 2) assessments took place an average of 44 days after Time 1 assessments, the second mid-treatment (Time 3) assessments took place an average of 87 days after Time 1 assessments, and end of treatment (Time 4) assessments took place an average of 135 days after Time 1 assessments. Reasons for nonparticipation in an assessment period included difficulty locating the youth and or the youth's family, youth and/or caregiver refusal to participate, youth in a placement or facility that would not allow the protocol to be completed (e.g., detention, wilderness program, incarceration), and youth run-away from home. Approximately half (51.2%) of the youth completed assessments at all four time points, 26.4% of the youth completed assessments at three time points only, 16.5% of the youth completed assessments at two time points only, and 5.8% of the youth completed assessments at Time 1 only. Youth who completed assessments at Time 1 only were dropped from analyses (N=7).

Measures

Salivary Testosterone and Cortisol Collection and Assay

At treatment onset, mid-treatment and post-treatment, 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 and cortisol.

Testosterone and cortisol follow a similar diurnal pattern, allowing for the collection of one saliva sample at the same time of day for purposes of assaying both testosterone and cortisol (Dabbs, 1990; Granger et al., 2003; Hibel et al., 2007; Klimes-Douga et al., 2001; Stansbury et al., 1994). And, although salivary concentration levels of testosterone and cortisol are lower than concentration levels found in serum (Malamud et al., 1993; Ohzeki et al., 1991), researchers have found that salivary levels of testosterone and cortisol are highly correlated with serum levels of testosterone and cortisol in children and adolescents (Sannikka et al., 1983).

This study followed the methods that have been advanced to increase immunoassay sensitivity of salivary testosterone (Granger et al., 1999), as well as suggested procedures for maintaining reliability and validity of salivary testosterone and cortisol levels (e.g., Granger, et al., 1999; Granger et al., 2004; Hibel et al., 2007; Schwart et al., 1998; Shirtcliff et al., 2002; Shirtcliff et al., 2001). Participants' vials were frozen and stored at -20°C for an average of three months before being mailed to the Yerkes Endocrine Core Laboratory for assay. The Center for Disease Control guidelines were followed for the transport of biological specimens, and samples were sent overnight delivery on 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. Salivary cortisol was assayed using an enzyme immunoassay kit (DSL, Webster, Texas), catalogue number DSL-10-67100. This assay procedure has an analytical sensitivity of 0.10 mg/dl, using 25 ml of saliva.

The intra- and inter-assay coefficient of variation are 4.1 and 7.2%, respectively. 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 ul 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 (for cortisol and testosterone) was used in analyses.

Ideally, testosterone is assayed from the saliva sample prior to cortisol because of testosterone degradation due to repeated freeze thaw. Due to a change in study protocol, this order of assay was not consistently maintained. To examine batch-to-batch variation in testosterone assay values, one-way ANOVA analyses were conducted whereby participant assay number was entered as the independent variable and testosterone was entered as the dependent variable. Results revealed a significant difference among assay number on their levels of testosterone for Time 1, $F(7, 97) = 3.34$, $p < .001$, for Time 2, $F(7, 86) = 4.00$, $p < .001$, and for Time 4, $F(8, 92) = 4.25$, $p < .001$. Assay number was therefore included as a statistical control in all analyses.

Testosterone Determination. Total levels of basal testosterone at Time 1 (pre-treatment), Time 2 (mid-treatment), and Time 4 (end of treatment) provided a continuous measure of testosterone in the analyses. Time of day, $r = -0.28$, $p < .01$, and number of hours since awakening, $r = -0.28$, $p < .01$, were significantly associated with testosterone levels at Time 2. Time of day was no longer a significant predictor of Time 2 testosterone when number of hours awake was controlled.

Analyses examining testosterone at Time 2, therefore, controlled for number of hours the participant was awake when they gave the saliva sample. To simplify the models, in cases where the results did not significantly change when this variable was left out as a control, results are presented without this variable as a control (i.e., Hypothesis 3).

Cortisol Determination. Total levels of basal cortisol levels at Time 1 provided a continuous scale of measurement and served as a moderator of the relationship between testosterone and aggressive and delinquent behavior (Hypothesis 2) and as a moderator for the relationship between testosterone and caregiver report of therapist adherence to MST principles (Hypothesis 1).

Preliminary analyses revealed significant results for the association between cortisol levels and the time of day that cortisol was collected, $r = -0.25$, $p < .01$, and between cortisol levels and the number of hours that participants were awake before collection, $r = -0.30$, $p < .01$. Time of day was no longer a significant predictor of cortisol when number of hours awake was controlled, $r = -0.08$, $p = .40$. Analyses examining cortisol therefore controlled for number of hours the participant was awake when they provided the saliva sample. To simplify the models, in cases where the results did not significantly change when this variable was left out as a control, results are presented without this variable as a control (i.e., Hypothesis 1).

Peer Delinquency Scale (PDS)

In this study, the PDS (Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1994) was administered at Time 1 and was used to assess delinquent peer affiliation. The PDS provides a measure of the ratio of the number of the participants' friends who engage in delinquent acts and/or substance use to the number of their friends who do not.

The original scale contains 15 items for which youth are asked to report on behavior during the past 30 days. Response choices for each question include: "0-None of them," "1-Few of them," "2-Half of them," "3 Most of them," and "4-All of them." For purposes of the current study, one additional substance use item was added: "During the past 30 days, how many of your friends have smoked cigarettes?" 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, providing a continuous scale of measurement. A higher score indicated that participants' friends engaged in more delinquent activity.

Loeber et al. (1994) reported a Cronbach's alpha of .84 with a sample of 500 11-year-old boys. In a separate study, Blackson and Tarter (1994) reported a Cronbach's alpha of .76 with a sample of 130 10, 11, and 12 year old boys. Content validity is suggested, as the PDS was developed from corresponding items of the Self-Reported Delinquency Scale (SRD) (Elliott et al., 2005). Good concurrent and predictive validity has also been reported (Keenan, Loeber, Zhang, Stouthamer-Loeber, and Van Kammen, 1995).

Alabama Parenting Questionnaire (APQ)

A modified form of the Alabama Parenting Questionnaire (Loeber et al., 1991) was administered at Time 1 and was used to assess parenting quality.

In its original format, the APQ is a 42 item, paper and pencil measure that examines parental involvement (10 items, e.g., you ask your child about his/her day at school), positive parenting (6 items, e.g., you compliment your child when he/she does something well), poor monitoring / supervision (10 items, e.g., your child is out with friends you don't know), inconsistent discipline (6 items, e.g., you threaten to punish your child and then do not actually punish him/her), corporal punishment (3 items, e.g., you slap your child when he/she does something wrong), and other parenting practices (7 items, e.g., you give your child extra chores when he/she is misbehaving). The modified format excluded corporal punishment items, used rewording to focus on the primary caregiver rather than both parents, and was administered on a laptop rather than in paper and pencil format. Spanish translations were provided for the participant caregivers who reported Spanish as their primary language.

Both child and caregiver reports of parenting were used in this study to account for potential problems with shared method variance (as both child and caregiver also reported on the child's levels of aggression and delinquency). Both child and caregiver reports on the APQ were scored by calculating a total score for each subscale and then combining the subscales, providing one continuous measure of parenting. Higher scores indicate better parenting quality. The coefficient alphas were .91 for the youth and .66 for the caregiver reports of parenting. Similar reliability statistics have been reported by others (Dadds et al., 2003; Shelton et al., 1996).

Outcome Measures

Therapist Adherence. The MST Therapist Adherence Measure - Revised (TAM-R; Henggeler, Borduin, Schoenwald, Huey, & Chapman, 2006) is a 28-item scale that assesses caregiver report of therapist adherence to the nine MST principles (Henggeler et al., 1998). For example, MST Principle 1, "the primary purpose of assessment is to understand the fit between the identified problems and their broader systemic context" (Henggeler et al., 1998) is assessed by the TAM-R item, "therapist tried to understand how the family's problems all fit together." TAM-R items were rated on a 5-point Likert scale, with response options ranging from 1 (not at all) to 5 (very much). Research has shown that therapist adherence is predictive of improved parenting skills and decreased youth problem behavior post- MST (Henggeler, Melton, Brondino, Sherer, & Hanley, 1997; Huey, Henggeler, Brondino, & Pickrel, 2000; Schoenwald, Sheidow, Letourneau, & Liao, 2003). This measure has been validated in several MST transportability studies (Schoenwald et al., 2003; Sholomskas, Syracuse-Siewert, Rounsaville, Ball, Nuro, & Carroll, 2005).

For purposes of this study, therapist adherence was measured at two mid-treatment time points (i.e., Time 2 and Time 3) and at post-treatment (i.e., Time 4). At each time point, caregivers were asked to report on adherence in the past week. Total scores were calculated with higher scores indicating greater therapist adherence. The alpha coefficient for this measure was 0.93 at Time 2, 0.94 at Time 3, and 0.97 at Time4.

Child Behavior Checklist – Ages 6-18 (CBCL). Aggressive and delinquent symptoms were assessed by caregiver report using ratings on the CBCL (Achenbach, 1991), which is one of the most well-validated measures of child behavioral functioning (Achenbach, 1991; Achenbach et al., 1983; Achenbach et al., 2001). This measure was administered to caregivers at Time 1, Time 2, Time 3, and Time 4. The CBCL consists of 113 behavior problem items. Broadly, this rating scale taps symptoms of Internalizing Problems (e.g., depression and anxiety), Externalizing Problems (e.g., aggression, conduct problems, attention-deficit/hyperactivity symptoms, and delinquency), and Total Behavior Problems (i.e., total scores on internalizing and externalizing problems). This study used the aggression and delinquency subscales. Responses to items range from zero to two: "0-Not True", "1-Sometimes True," and "2-Very True or Often True." 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 measures of aggressive and delinquent behavior. *T*-scores higher than 60 were considered clinically significant.

At Time 1, 46% of the sample received a *T*-score of 60 or greater on the CBCL Aggression subscale, while 77% of the sample received a *T*-score of 60 or greater on the CBCL Delinquency subscale. At Time 2, 21% of the sample received a *T*-score of 60 or greater on the CBCL Aggression subscale, while 48% of the sample received a *T*-score of 60 or greater on the CBCL Delinquency subscale. At Time 3, 19% of the sample received a *T*-score of 60 or greater on the CBCL Aggression subscale, while 31% of the sample received a *T*-score of 60 or greater on the CBCL Delinquency subscale.

Last, at Time 4, 26% of the sample received a *T*-score of 60 or greater on the CBCL Aggression subscale, while 53% of the sample received a *T*-score of 60 or greater on the CBCL Delinquency subscale.

In this study, the aggressive subscale had a coefficient alpha of 0.93 at Time 1, of 0.92 for Time 2, of 0.91 for Time 3, and of 0.94 for Time 4. The delinquency subscale had a coefficient alpha of 0.85 for Time 1, of 0.83 for Time 2, of 0.82 for Time 3, and of 0.87 for Time 4.

Self-report Delinquency Scale (SRD). The SRD (Elliot, 1994; Elliot et al., 1983; Elliot et al., 1976) was administered at Time 1, Time 2, Time 3, and Time 4 and is a self-report, 47-item scale that measures antisocial behavior and includes subscales that pertain to violent offending (e.g., attacking someone with the intention of hurting them, forcing another person to have sex), general delinquency (i.e., covert and overt delinquency and violent crimes), property offenses (e.g., purposely damaging or destroying property belonging to parents, other family members, or school), and status offenses (e.g. alcohol use, not attending school, staying out late, and running away). In its original format, youth are asked to report on these behaviors over the last year and in instances where the youth endorses 10 or more problem behaviors, they are asked if the acts occurred (1) once a month, (2) once every two to three weeks, (3) once a week, (4) two to three times a week, (5) once a day, or (6) two to three times a day.

For the purposes of the current study, several modifications were made to this measure. First, we utilized the general delinquency total score. Second, youth were asked to report on their behavior over the last 30 days (e.g., "How many times in the past 30 days have you:").

Third, seven items related to drug use were excluded. Fourth, frequency variations for item response choices differed: behaviors were reported in range of 0-99, or as the category of 100 plus. Scores were derived by calculating the total number of youth problem behaviors reported in each category of offense. High scores indicated more involvement in aggression and delinquency.

The SRD is the best validated of the self-report delinquency scales (Henggeler, 1989). Researchers have found test-retest reliability for this measure to range from .85 to .99, using intervals ranging from less than one hour to over two months (Patterson & Loebber, 1982; Hindelang, Hirschi, & Weis, 1982). Huizinga & Elliott (1986) found that there were no significant differences across sex, race, class, place of residence, or delinquency levels for estimates of test-retest reliability. Huizinga et al. (1986) also found that the majority of individuals who have been arrested self-report their delinquent behavior, and the majority of offenses they commit are also reported. In this study, the general delinquency scale had a coefficient alpha of 0.90 at Time 1. At Time 2 and Time 3, the general delinquency scale had a coefficient alpha of 0.47; at Time 4 it was 0.70.

Control Variables

Health Questionnaire. At Time 1, Time 2, and Time 4 youth and their caregiver were asked about the youth's physical health in the 24 hours before the assessment. Eating patterns and medication schedules were examined in relation to all independent measures of salivary testosterone and cortisol. Results did not reveal significant relationships for the association between the aforementioned variables and cortisol.

A significant correlation was found for the association between Time 2 and Time 4 testosterone levels and youth report of whether they smoked a cigarette on the day of assessment, $r = .28$, $p < .01$ and $r = .27$, $p < .01$. For analyses that included Time 2 and Time 4 testosterone levels, analyses were run with and without controls for this factor. Results were not significantly different. Thus, reported analyses did not include this statistical control.

Peterson Pubertal Development Scale (PPDS, Peterson et al., 1988*).* The PPDS was administered at Time 1 and Time 4. This measure 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, change in skin, and changes in their voice. Peterson et al. (1988) reported adequate reliability and validity statistics. Additionally, this measure has been widely used in the examination of pubertal development/status (e.g., Granger et al., 2004). For the purposes of the current study, a total puberty score was calculated by summing the items and dividing by 5, to maintain the original metric. There was a trend toward significant results for the association between puberty scores and testosterone levels at Time 1, $r = .17$, $p = .09$, and Time 4, $r = .20$, $p < .06$. Puberty status was not collected at Time 2.

Partial correlation analyses revealed that age was more closely associated with hormonal development than puberty. In this study, the relationship between testosterone and puberty, controlling for age was not significant at Time 1, $r = .01$, $p = .94$ or at Time 4, $r = .09$, $p = .39$. The relationship between testosterone and age, controlling for puberty, however, remained significant at Time 1, $r = .20$, $p < .05$, and at Time 4, $r = .27$, $p < .01$. Age was therefore controlled in all analyses.

Height and Weight. Height and weight were assessed at Time 1, Time 2, and Time 4. For the purposes of this study, the 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 and height, weight, and BMI was assessed. Results revealed no significant associations at Time 1, Time 2, or Time 4.

Analytic Procedures

Data Analyses and Statistical Modeling

Growth curve analyses were conducted using the HLM 6 computer program (Raudenbush, Bryk, & Congdon, 2004) in order to model growth curve trajectories of the following outcome variables over the course of treatment: (a) caregiver report of therapist adherence to MST protocol, (b) aggressive behavior, and (c) delinquent behavior. Growth Curve Modeling (GCM) allows for a simultaneous, two-stage process. The first stage (Level 1) estimates a trajectory of change (growth curve) for a variable (e.g., caregiver report of therapist adherence), described by two parameters: intercept and slope (rate of change in variable over time). Time was measured in multiple ways. For Hypothesis 1, Time was calculated (a) as days since Time 2 to model the intercept parameter as level of the outcome at Time 2, (b) as days since Time 2 to model the intercept as level of the outcome at Time 3, and (c) as days since Time 3 to model the intercept as level of the outcome at Time 4. For Hypotheses 2 and 3, Time was calculated as number of days since Time 1, at Time 1, Time 2, Time 3, and Time 4 assessments.

To identify the best fitting growth curve models, three types of models were compared for each of the outcome variables: a mean and variance model (i.e., modeling scores as varying randomly around a participant's mean level of a variable over time), a linear model (i.e., modeling systematic increase or decrease over time), and a quadratic model (i.e., modeling systematic curvilinear change over time). To model quadratic change over time, a quadratic variable was computed by squaring the time variable; both time and quadratic variables were included in the quadratic model (Singer & Willett, 2003). To examine the relative fit of these models, parameter estimates were examined (e.g., *t* test of intercept and slope parameters and χ^2 tests of between-subject variance). Deviance statistics of mean-and-variance, linear, and quadratic models were compared to determine which model was the best fit for the data using the hypothesis testing function in HLM 6.0.

For the present study, an additional (3^{rd}) level was added to each model, nesting participants within primary therapists in order to account for interdependence among participants who met with the same therapist[2]. For example, to test Hypothesis One, three repeated measures of caregiver report of therapist adherence (Level 1) were nested within adolescent boys (Level 2) who were nested within their primary therapists (Level 3).

In each HLM model, initial levels of the outcome variable at the start of treatment and the random effects modeled at Level 2 were allowed to vary across adolescents. 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 Time 1 predictors, centered. The linear counterparts were entered into HLM in raw score format. For model specification, the *Time* variable was entered at Level 1, un-centered. All continuous variables at Level 2 were grand-mean centered; and all dichotomous variables at Level 2 were un-centered.

With regard to moderation analyses, significant interactions were interpreted using methods suggested by Preacher, Curran, & Bauer (2003) and Curran, Bauer, & Willoughby (2006). For significant interactions, Preacher, et al.'s (2003) software for identifying simple effects was used to interpret the relationship between testosterone and the outcome variables at different values of the moderating term. For most significant interactions, simple effects were examined at (a) 1 standard deviation (SD) below the mean level of the moderator, (b) at the mean level of the moderator, and (c) at 1 SD above the mean level of the moderator. However, 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 because these values are clinically relevant and therefore provide more valuable information about simple effects. See Appendix D for interpreting simple slope analyses within the context of growth curve models.

Missing Data

HLM can handle missing data at Level 1 (repeated outcome measures). However, HLM cannot handle missing data at Level 2 which, in this study, included adolescent level variables such as testosterone, cortisol, deviant peer affiliation, parenting, age, race, etc.

As suggested by others (Schafer & Graham, 2002), missing data are best handled by estimating the values (i.e., data imputation). For purposes of this study, all missing data for predictor variables were estimated with the use of the *NORM* software developed by Schafer (1999). Five imputations were deemed appropriate given that only 1.8%-13.3% of data were missing.

Results

Descriptive Analyses

Table 10 presents the means and standard deviations for the independent and dependent variables used in the analyses at each time point. Table 11 presents the correlations between all repeated measures and two of the control (i.e., race and age) variables used in the analyses. As shown in the Table, race ("0" Minority, "1" European-American was correlated with CBCL aggression at all 4 time points, thus this measure was entered as a statistical control when CBCL aggression and CBCL delinquency (given the high correlation with CBCL aggression) served as the outcome variable. Of note, caregiver report of therapist adherence to treatment was not related to aggression or delinquency at Time 2, Time 3, or Time 4.

Hypothesis 1: Time 1 basal levels of testosterone will interact with Time 1 parenting, peer affiliation, and basal cortisol levels as predictors of Time 2, Time 3, and Time 4 caregiver report of therapist adherence to MST treatment.

Baseline Models. Baseline models of caregiver report of therapist adherence were examined using scores obtained at Times 2, 3, and 4.

First, the following linear model was specified:

$$Y_{ti}\,(\text{therapist adherence}) = \pi_{0i}\,(\text{Intercept}) + \pi_{1i}\,(\text{Time}) + e_{ti}$$

where Y_{ti} is caregiver report of therapist adherence for individual *i* at time *t*, π_{0i} is the intercept for individual *i* (i.e., caregiver report of therapist adherence at Time 2, Time 3, or Time 4), π_{1i} is the slope for individual *i* (the rate of linear change in caregiver report of therapist adherence for individual *i* over time), and e_{ti} which is the residual variance in repeated measures for individual *i*, which is assumed to be independent and normally distributed. On average, there was no systematic linear change in caregiver report of therapist adherence scores across time, $t(113) = 1.23$, $p = .23$; however, there was significant between-subject variability in the time parameter (π_{1i}), $\chi^2(100) = 77.88$, $p < .001$. The relative fit of the linear model was compared to a mean-and-variance model (omitting the time parameter): $Y_{ti}\,(\text{therapist adherence}) = \pi_{0i}\,(\text{Intercept}) + e_{ti..}$ The linear model was a better fit for the data, $\chi^2\,(5) = 546.25$, $p < .001$, and indicated a slight increase in caregiver report of therapist adherence over the course of treatment.

The relative fit of the linear model was then compared to that of a quadratic model:

$$Y_{ti}\,(\text{therapist adherence}) = \pi_{0i}\,(\text{Intercept}) + \pi_{1i}\,(\text{Time}) + \pi_{3i}\,(\text{Time}^2) + e_{ti}$$

where π_{3i} is the rate of curvilinear change in caregiver report of therapist adherence for individual *i* over time). On average, caregiver report of therapist adherence did not change in a curvilinear fashion over time, $t(113) = 0.71$, $p = .48$; however, there was significant between-subject variability for the quadratic parameter, $\chi^2(5) = 16.37$, $p < .01$, suggesting that curvilinear change was occurring for a subset of participants. The relative fit of the linear model was compared to that of the curvilinear model.

The linear model was a better fit for the data (adding the quadratic term did not significantly improve the fit of the model), $\chi^2(1) = 3.70$. $p = .07$. Thus, a linear model of caregiver report of therapist adherence was specified for all subsequent analyses[3].

Moderation Analyses. To examine moderation, the following model was specified with testosterone, each moderator, and the corresponding interaction term entered in each of the Level 2 equations:

(Level 1): Y_{ti} (therapist adherence) = π_{0i} (Intercept) + π_{1i} (Time) + e_{ti}

(Level 2): π_{0i} (Intercept) = β_{00} + β_{01} (testosterone) + β_{02} (moderator) + β_{03} (interaction) + r_{0i}

π_{1i} (Time) = β_{10} + β_{11} (testosterone) + β_{12} (moderator) + β_{13} (interaction) + r_{1i}

Control variables were also included in the analyses, but are omitted from the equations for ease of presentation. For this hypothesis, the significance of the coefficient for the interaction term β_{03} was of interest.

Testosterone and Cortisol. It was hypothesized that Time 1 basal testosterone and Time 1 basal cortisol levels would interact as predictors of caregiver report of therapist adherence at Time 2, Time 3, and Time 4. Results did not support this hypothesis. When controlling for the overall trajectory of change in caregiver report of therapist adherence over time, there were no significant interactions between testosterone and cortisol predicting Time 2 caregiver report of therapist adherence, $t(108) = -1.40$, $p = .17$, Time 3 caregiver report of therapist adherence, $t(108) = -0.48$, $p = .64$, or Time 4 caregiver report of therapist adherence, $t(108) = -1.05$, $p = .30$.

Of interest, however, is that the interaction between Time 1 basal testosterone and Time 1 basal cortisol *was* significant in predicting the rate of change in caregiver report of therapist adherence throughout the course of treatment, $t(108)= -2.07$, $p < 0.05$, (see Table 12). In other words, the effect of Time 1 testosterone on linear change in caregiver report of therapist adherence varied as a function of Time 1 cortisol levels. Post-hoc analyses of these significant interactions were conducted in order to assess simple effects of Time 1 testosterone on changes in caregiver report of therapist adherence at three distinct levels of Time 1 cortisol: at 1 SD below the sample mean, at the mean levels of cortisol for this sample ($M = 0.43$), and at one standard deviation above the sample mean of cortisol levels.

For adolescents with cortisol levels one standard deviation below the mean, Time 1 testosterone levels were significantly associated with changes in caregiver report of therapist adherence scores, $b = -0.001$, $p < .01$. At the average level of cortisol for the sample, testosterone levels were also significantly associated with changes in caregiver report of therapist adherence scores, $b = -0.003$, $p < .05$. In addition, at the value of one standard deviation above the sample mean of cortisol levels, testosterone was associated with changes in caregiver report of therapist adherence scores, $b = -0.006$, $p < .05$. Results of these post-hoc moderation analyses suggest that increases in Time 1 testosterone levels are more strongly associated with a linear decline in caregiver report of therapist adherence at higher levels of Time 1 cortisol (see Figure 1 and Appendix D).

Testosterone and Deviant Peer Affiliation. It was hypothesized that Time 1 basal testosterone and Time 1 deviant peer affiliation would interact as predictors of caregiver report of therapist adherence at Time 2, Time 3, and Time 4.

Results did not support this hypothesis. There were no significant interactions between testosterone and deviant peer affiliations in the prediction of Time 2 caregiver report of therapist adherence, $t(108) = 0.70$, $p = .49$, Time 3 caregiver report of therapist adherence, $t(108) = 0.87$, $p = .39$, or Time 4 caregiver report of therapist adherence, $t(108) = 1.11$, $p = .27$. Further, moderation was not significant for *changes* in caregiver report of therapist adherence over time, $t(108) = 1.14$, $p = .26$.

Testosterone and Parenting. It was hypothesized that Time 1 basal testosterone and Time 1 parenting quality would interact as predictors of caregiver report of therapist adherence at Time 2, Time 3, and Time 4. Results did not support this hypothesis. There were no significant interactions between testosterone and youth report or caregiver report of parenting quality in the prediction of Time 2 caregiver report of therapist adherence, (youth report: $t(108) = -0.27$, $p = .79$; caregiver report: $t(108) = -0.06$, $p = .95$), Time 3 caregiver report of therapist adherence (youth report: $t(108) = -0.14$, $p = .89$; caregiver report: $t(108) = 0.66$, $p = .51$), or Time 4 caregiver report of therapist adherence (youth report: $t\ (108) = -0.20$, $p = .85$; caregiver report: $t(108) = -0.51$, $p = .62$). Further, moderation was not significant for *changes* in caregiver report of therapist adherence over time, (youth report, $t(108) = 0.68$, $p = .50$, caregiver report, $t(108) = 0.78$, $p = .44$).

Hypothesis 2: Time 1 baseline levels of testosterone will interact with Time 1 parenting, peer affiliation, and basal cortisol levels to predict changes in aggression and delinquency over time.

Baseline Models. Existing treatment outcome studies of MST suggest that aggressive and delinquent behavior decline over the course of treatment.

In the current study there was a trend toward youth self-report of delinquency (i.e., SRD general delinquency) declining over the course of treatment, $t(113) = -1.85$, $p = .07$. Results also revealed that caregiver report of aggressive, $t(113) = -5.75$, $p < .001$, and delinquent behaviors, $t(113) = -3.18$, $p < .01$, demonstrated a systematic linear decline in scores over time, on average. A linear model was a better fit than a mean-and-variance model and a quadratic model for each of these outcomes. Accordingly, a linear baseline model was specified for subsequent analyses pertaining to this hypothesis.

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

(Level 1): Y_{ti} (aggression or delinquency) = π_{0i} (Intercept) + π_{1i} (Time) + e_{ti}

(Level 2): π_{0i} (Intercept) = β_{00} + β_{01} (testosterone) + β_{02} (moderator) + β_{03} (interaction) + r_{0i}

π_{1i} (Time) = β_{10} + β_{11} (testosterone) + β_{12} (moderator) + $\boldsymbol{\beta_{13}}$ **(interaction)** + r_{1i}

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

Testosterone and Cortisol. It was hypothesized that the interaction between Time 1 basal testosterone levels and Time 1 basal cortisol levels would predict *changes* in aggression and delinquency as measured by youth report (i.e., SRD General Delinquency) and changes in caregiver reports of aggressive and delinquent behavior. This hypothesis was not supported. In the context of this study, there were no significant interactions between testosterone and cortisol as predictors of the observed linear decline in SRD general delinquency, $t(107) = -0.78$, $p = .44$, CBCL Aggression, $t(106) = -1.18$, $p = 0.26$, or CBCL Delinquency, $t(106) = -1.24$, $p = .23$.

Testosterone and Deviant Peer Affiliations. It was hypothesized that Time 1 basal testosterone levels and Time 1 deviant peer affiliations would interact to predict changes in caregiver and youth report of aggressive and delinquent behavior in response to MST. Results revealed partial support for this hypothesis: two out of four interactions were significant.

The interaction between Time 1 testosterone levels and Time 1 deviant peer affiliation was significant for the outcomes of caregiver report of CBCL aggression, $t(107) = 2.54$, $p < 0.05$ and CBCL delinquency scores, $t(107) = 2.27$, $p < .05$, (see Table 13). That is, the effect of Time 1 testosterone on changes in caregiver report of aggression and delinquency depended on the degree of Time 1 deviant peer affiliation. Post hoc analyses of these significant interactions were conducted in order to assess the simple effects of Time 1 testosterone on changes in aggression and delinquency at three distinct levels of Time 1 deviant peer affiliation: at zero (no deviant peer affiliations), at the mean level of deviant peer affiliation for this sample ($M = 9.12$), and at one standard deviation above the sample mean of deviant peer affiliation.

For participants with no deviant peer affiliation at Time 1, Time 1 testosterone was not significantly associated with changes in caregiver reports of CBCL aggression scores, $b = 0.00$, $p = 0.49$. At the average level of deviant peer affiliation for the sample, testosterone levels were significantly associated with changes in CBCL aggression scores, $b = 0.07$, $p < .05$. Higher levels of testosterone were associated with a slower decline in CBCL aggression scores.

At the value of one standard deviation above the sample mean of deviant peer affiliation, higher testosterone was even more strongly associated with a smaller decline in CBCL aggression scores b =0.15, $p < .05$ over the course of MST treatment (see Figure 2 and Appendix D).

A similar pattern of moderation emerged for caregiver report of CBCL delinquency (see Figure 3). Specifically, Time 1 testosterone was not significantly associated with changes in CBCL delinquency for participants with zero deviant peer affiliation, b = -0.00, p = 0.46. However, testosterone was significantly associated with changes in CBCL delinquency at mean levels of deviant peer affiliation; that is, higher levels of testosterone at baseline were significantly associated with a lesser decline in delinquency over time, b= 0.11, $p < .05$. Post-hoc analyses also revealed that the association between testosterone and changes in delinquency was even stronger at one standard deviation above the mean of deviant peer affiliation, b = 0.22, $p < .05$.

There were no significant interactions between Time 1 testosterone and Time 1 deviant peer affiliation in predicting changes in youth report of aggression and delinquency (i.e., SRD general delinquency) throughout the course of MST treatment, $t(108) = -0.90$, $p = .37$.

Testosterone and Parenting, Youth Report. It was hypothesized that Time 1 testosterone levels and Time 1 youth report of parenting quality would interact to predict aggressive and delinquent behavior. This hypothesis was not supported.

There were no significant interactions between testosterone and youth report of parenting quality as predictors of changes in youth report of aggression and delinquency, $t(108) = -0.95$, $p = .35$, caregiver report of aggressive behavior, $t(107) = -0.11$, $p = .91$, or caregiver report of delinquent behavior, $t(107) = 0.25$, $p = .80$.

Testosterone and Parenting, Caregiver Report. It was hypothesized that Time 1 testosterone and Time 1 parenting quality would interact to predict changes in aggressive and delinquent behavior over time. Results revealed that for caregiver report of parenting quality, this hypothesis was partially supported; that is, one out of four interactions was significant.

The interaction between Time 1 testosterone levels and Time 1 parenting quality as reported by the caregiver was significant for the outcome of CBCL delinquency, $t(107) = 2.12$, $p < .05$ (see Table 14). The effect of Time 1 testosterone on changes in caregiver report of CBCL delinquency depended on the degree of Time 1 parenting quality. Post hoc analyses of this significant interaction were conducted in order to assess the simple effects of Time 1 testosterone on changes in delinquency at three distinct levels of Time 1 parenting quality: at one standard deviation below the sample mean of parenting quality ($b = 0.97$, $p < .05$), at the mean level of parenting quality for this sample ($M = 137.14$; $b = 1.09$, $p < .05$), and at one standard deviation above the sample mean of parenting quality ($b = 1.22$, $p < .05$). Results of post-hoc moderation analyses suggest that higher levels of testosterone are more strongly associated with lesser declines in delinquency over the course of treatment for those youth with a higher quality parenting (see Figure 4).

There were no significant interactions between Time 1 testosterone and Time 1 caregiver report of parenting as predictors of changes in youth report of aggression and delinquency (i.e., SRD general delinquency), $t(108) = 0.80$, $p = .43$, or caregiver report of aggressive behavior (i.e., CBCL aggression), $t(107) = -0.09$, $p = .93$.

Hypothesis 3: Changes in baseline levels of testosterone will be associated with changes in aggression and delinquent behavior across time.

This hypothesis was tested, first, with each aggression and delinquency variable as the outcome and, second, with testosterone as the outcome. Given that this hypothesis was concerned with the covariation of testosterone and aggression and delinquency over time, it does not matter which variable is used as the dependent variable: testosterone or aggression /delinquency. However, since "time" was included in the baseline equations, how we control for the effects of time is different for each outcome variable. When testosterone was entered as the dependent variable, the linear trajectory of testosterone was taken into account (i.e., systematic change in testosterone across time that is independent of the effects of aggression and delinquency), whereas when aggression and delinquency were entered as the dependent variables, the linear trajectories of aggression and delinquency were taken into account (e.g., systematic change in delinquency across time that is independent of the effects of testosterone).

Baseline models. As previously discussed, a linear model (i.e., on average there was a systematic linear decline in scores over time) was the best fit for aggression and delinquency data (see Hypothesis 1). The same method was applied to examine the best fitting baseline model of testosterone assessed at Time 1, Time 2, and Time 4.

Results did not reveal significant linear change (i.e., increase) in testosterone over time, t (112) = 1.25, p = .22. Results did reveal, however, significant between-subject variability in the time parameter for the linear model. When compared to a mean and variance model, the linear model was a better fit for the testosterone data, χ^2 (5) = 781.83, $p <$ .001. A quadratic model was not a better fit. Thus, testosterone was examined as a linear increase over time. For all analyses, the error terms for the intercepts were fixed to achieve model convergence.

Covariation. The following (Level 1) model was specified to examine covariation:

$$Y_{ti}\,(\text{Outcome}) = \pi_{0i}\,(\text{Intercept}) + \pi_{1i}\,(\text{Time}) + \pi_{2i}\,(\text{Covariate}) + e_{ti}$$

where π_{2i} represents the extent to which changes in the covariate are associated with changes in the outcome over time.

When aggression and delinquency were each entered as the outcome, testosterone (entered as the covariate) was not significantly associated with rates of change in aggression or delinquency during treatment: SRD general delinquency, t(108) = 1.25, p = .22, CBCL aggression, t(107)=-0.59, p =.56, and CBCL delinquency, t(107) = -0.17, p = .87. When testosterone was examined as the outcome and aggression and delinquency were each entered as covariates (in separate analyses), results revealed partial support for this hypothesis; that is, results showed that to the extent that testosterone increased over time, there was less linear decline in SRD general delinquency over time, t(108) = 2.18, p < .05. Alternatively stated, to the extent that delinquency decreases over time, there was steeper linear decline in testosterone over time as well.

Results were not significant for caregiver report of aggression and delinquency: CBCL aggression, $t(107) = -0.98$, $p = .33$, CBCL delinquency, $t(107) = -0.70$, $p = .49$.

Discussion

The goal of this study was to examine the association between testosterone and changes in aggression and delinquency within the context of an intervention study, while taking into account the moderating influences of deviant peer affiliations, parenting, and cortisol levels. The aim was to test for these associations by first examining the relationship between hormones and social context and its impact on caregiver report of therapist adherence. Results revealed that only intra-individual levels factors (testosterone and cortisol) interacted to predict changes in caregiver report of therapist adherence scores over time. Results concerning the moderating effect of social context on the relationship between testosterone and youth behavioral outcomes suggest that male adolescents with higher levels of testosterone were less responsive to MST, if they also had delinquent peer associations. Interestingly, higher testosterone also predicted a lesser decline in aggression and delinquency over the course of MST for those youth who evidenced high quality parenting (i.e., parenting monitoring, involvement, supervision, etc.). These findings affirm the value of incorporating both biological and social factors in predicting delinquent behavior (Bauma et al., 1992; Booth et al., 2003; Rowe et al., 2004). The results of the current study also suggest that testosterone may be an important biological risk factor to consider when predicting treatment outcome.

The findings concerning the interaction between testosterone and cortisol were not in the predicted direction.

Specifically, results revealed that the association between high testosterone and rate of linear decline in caregiver report of therapist adherence was stronger for individuals with relatively *high* levels of cortisol at Time 1. Additional information such as history of stressful life events may have been necessary to fully interpret these findings. Research suggests that duration and chronicity of stress is related to increased levels of basal cortisol (Alink et al., 2007). For the current study, Time 1 assessments took place shortly after an event which required the youth and his family to come in contact with family services or juvenile justice agencies. Individual difference in time since the stressor that led to seeking these services might be an important variable to account for. It is possible that individuals with high cortisol levels may have experienced more recent stress. This in conjunction with a propensity for dominance or sensation seeking may have led therapists to diverge from MST principles more often when working with these families.

A limitation of the aforementioned interpretations is that the current study only assessed caregiver report of therapist adherence to MST principles. Lower levels of caregiver report of therapist adherence do not imply poor therapeutic services, just services outside the scope of the MST principles. And our findings suggest that a combination of high cortisol and high testosterone did not differentially predict changes in youth externalizing behavior, only changes in caregiver report of therapist adherence to MST. Future research should include a broader assessment of therapeutic techniques in order to assess whether youth with this "high-risk" hormonal profile may pull for and be more responsive to alternative modes of treatment.

In a review of the literature concerning the relationship between HPA axis and antisocial behavior, Hawes, Brennan, and Dadds (2009) propose that the direction of the relationship between basal cortisol and aggression/delinquency depends on several other factors including the presence of callous-unemotional traits and early life adversity. To better understand the relationship among testosterone and cortisol and changes in caregiver report of therapist adherence, aggression and delinquency, additional third variables (such as personality traits and stress exposure) should be included in the analyses.

Research within the dominance literature, as well as the Challenge Hypothesis suggests that winning of dominance contests is associated with changes in cortisol levels (Mehta, Jones, and Josephs, 2008; Oyegbile et al., 2006). Research has also demonstrated that individuals with high levels of testosterone are more likely to seek and maintain a dominant status and that testosterone levels rise and fall depending on pre and post contest status (Mazur et al., 1998). Taken together, these literatures suggest a possible reciprocal relationship among testosterone, cortisol, and behavior over time (Archer, 2006; Mazur et al., 1998; Terburg et al., 2009). It is possible that by only examining testosterone and cortisol at Time 1, the current investigation was unable to identify a reciprocal relationship between testosterone and cortisol and the influence on changes in aggression and delinquency.

As predicted, youth with initially high levels of testosterone and deviant peer affiliations evidenced less of a 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., 2008a). An increased propensity for sensation seeking and risk taking behaviors, in addition to deviant peer affiliation, may make an individual particularly vulnerable to aggressive and delinquent behavior. It is possible that with increasing numbers of deviant peer affiliations, the opportunity for engaging in problem behaviors increases because of a greater exposure to 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 may be more apt to engage deviant peer talk. The vulnerability to engage in deviancy training plus a compromised ability to appreciate negative consequences that result from negative behaviors provides conditions for engaging in aggressive and delinquent behavior (Snyder, 2002; Terburg et al., 2009). In other words, adolescent males with high levels of testosterone may (1) appeal more to deviant peers because of the vulnerability for sensation seeking and risk taking behavior, (2) attract toward deviant peer groups because of the vulnerability for sensation seeking and risk taking behavior, and/or (3) a combination of numbers one and two.

In this study, parenting also interacted with youth testosterone levels at the outset of treatment to predict changes in delinquency over time. However, this finding was not in the predicted direction. Specifically, high testosterone was a better predictor of continued delinquency in cases where the quality of the parenting was *better* at the onset of treatment. It may be the case that in families with adequate parenting, MST was less effective (because there was less to work with in terms of existing problems), particularly in cases where the youth was at high biological risk for externalizing behavior problems.

Our findings are also consistent with the 'social push' hypothesis (Raine, 2002), which posits that, within healthy social environments, the relationship between biological factors and outcome prevails, whereas, in non-healthy environments, the contextual factors overshadow the relationship between biological factors and outcome.

Alternatively, caregiver report of parenting quality may have been vulnerable to impression management or caregiver's report of ideal parenting behaviors. As indicated above, internal consistency for this measure was relatively lower, 0.66. This value, when compared to .91 for youth report of parenting quality, suggests that caregivers may have responded differently to similar items, rendering the scale less valid. In addition to the youth report of parenting quality, a third objective measure of parenting quality may be important (i.e., therapist of observational measure). Future research should consider additional measures of parenting quality, especially at treatment on-set –a period where families are acclimating to their new treatment provider.

The results of the current study also revealed that to the extent that youth report of general delinquency scores decreased across time, there was a smaller rate of increase in testosterone levels. This finding provides promising initial results for the application of a reciprocal model between testosterone and social context. If delinquency is associated with increasing dominance that could lead to an increase in testosterone (Mazur et al., 1998; Oyegbile et al., 2005, 2006). It is important to note, however, that the association over time between testosterone and youth delinquency was significant for youth report of delinquency as opposed to caregiver report of aggression and delinquency, whereas the interaction between testosterone and social context predicted *caregiver* report of aggression and delinquency over time.

In addition, the SRD had lower reliability than the caregiver report of aggression and delinquency. Thus, results should be interpreted with caution. Future researchers may wish to apply different measures of aggressive and delinquent behavior in an attempt to replicate these findings.

Strengths and Limitations

The current study has at least five notable strengths. 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 behavior change. Second, the use of a clinical population allows for a clear examination of change over time. With a less severe population, 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, this study included repeated measurement of the independent and dependent variables, measured simultaneously, at each time point. Last, the population in this study is more demographically heterogeneous than those examined in previous studies of the relationship between testosterone and problem behavior.

This study also suffered from some notable limitations. First, the use of a clinical sample resulted in a restricted range of aggressive and delinquent behavior at the lower end. Second, the current study did not include a comparison group/control group. The absence of a comparison/control group makes it difficult to attribute changes in aggression and delinquency over time to MST treatment. Such factors as maturation and passage of time may also account for the observed decline in aggression and delinquency.

Third, given current conceptualizations of testosterone and dominance (e.g., Mazur et al., 1998), the inclusion of a measure of dominance behavior would have aided in the interpretation of our results. Fourth, this study did not include adolescent girls in the sample. Although this was intentional, due to the need to control for factors such as menstrual cycle and pregnancy, additional studies that examine the association between testosterone and aggression among women is necessary. Currently, only a handful of studies have examined this relationship in a sample of girls or women (e.g., Granger et. al, 2003; Vermeersch, et al., 2008b). Fifth, this study did not consider such co-occurring disorders as ADHD. Research suggests that there is a curvilinear relationship between testosterone and depression (Booth et al., 1999). It is possible that a positive, negative, or curvilinear relationship exists between testosterone and ADHD. Co-occurring ADHD may predict chronicity of aggression or delinquency (Frick & Ellis, 1999). Hence, co-occurring ADHD may influence the relationship between testosterone and changes in aggression or delinquency. Last, testosterone levels were significantly different among three assay batches at Time 1 and two assay batches at Time 2 and Time 4. Though this variability was statistically controlled in all analyses, results may have been impacted by the possible unreliability of the testosterone measure.

Clinical Implications

It may be important to consider the potential role of testosterone in predicting treatment non-response to MST, especially in cases where the adolescent lives in a home where the parent practices sound social control strategies (e.g., consistent discipline, supervision, etc.).

Current theoretical assumptions, as well as preliminary results presented here suggest that varying therapeutic approaches may be necessary for treating youth who respond poorly to treatments such as MST and who have high levels of testosterone.

The results of this study also suggest that a heavy emphasis on intervening in adolescent peer relationships may be necessary among youth who respond poorly to MST and who have high levels of testosterone. 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 MST has to offer potentially necessary. For example, therapist could implement immediate removal (with monitoring) from delinquent peer groups in conjunction with social skills training, instead of only relying on increasing parent's attempts at social control. It seems most appropriate; however, for this to occur in addition to teaching parents to implement a contingency-management program when their child does not follow the rules related to associating with delinquent peers.

General Discussion

Current research on the relationship between testosterone and aggression and delinquency is mixed. This mixed literature led to the hypothesis that social context factors might moderate the relationship between testosterone and aggression or delinquency. This hypothesis was tested in both a cross-sectional and a longitudinal study. The cross-sectional study failed to show a significant interaction between testosterone and social context as related to aggressive or delinquent behavior. The longitudinal study, on the other hand, suggests that social context variables moderate the relationship between testosterone and aggression and delinquency over time.

Independently, the results of these studies are consistent with the larger literature; that is, cross-sectional studies seem to yield more negative results, whereas, longitudinal studies seem to consistently reveal a significant relationship between testosterone and aggression or delinquency at a future time point during adolescence. Importantly, the current project demonstrates differential results based on study design using the same sample.
It may be the case that mixed findings from cross-sectional studies are lacking micro-level factors that are better captured in a longitudinal context, especially a treatment outcome study.

As shown in Study 2, results were significant for deviant peer affiliation and parenting as moderators of the relationship between testosterone and changes in aggression and delinquency. Results, also suggests, however, that specific measures of micro-level factors may be important for better understanding the interaction between testosterone and parenting. The dominance literature suggests that youth with high testosterone levels are motivated to maintain dominant status in social interactions and this propensity for dominant behavior may lead to less of a decrease in aggression and delinquency in a family treatment context.

The current studies provide an opportunity to evaluate available theoretical models for the relationship between testosterone and aggression and delinquency. Mazur and Booth (1998) provide two models: basal model and reciprocal model. The basal model assumes relatively stable levels of testosterone across time. The reciprocal model assumes that testosterone fluctuates in response to changes in social environments. Results of the co-variation between testosterone and delinquency over time support the application of the reciprocal model.

Continued investigations of the relationship between testosterone and changes in aggression and delinquency within the context of treatment outcome may serve to increase our understanding of differential treatment response. A better understanding of biological mechanisms may serve to increase both intervention and prevention efficiency. Preliminary research suggests that intervention models have a therapeutic effect on hormones (e.g., Bakermans-Kranenburg et al., 2008; Brotman et al., 2007; Dozier et al., 2008). A next step in intervention research may be to therapeutically target biological risk factors so that intervention specialists can simultaneously treat both the social risk and the biological risk factors that lead to aggressive and delinquent behavior.

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Appendix A

PEER DELINQUENCY SCALE
Youth Report

The following questions will be about your friends. Think of the friends you usually have played with or hung out with during the past 30 days. Please remember, your answers are strictly confidential.

During the past 30 days how many of your friends have...

1. Skipped school without an excuse?
   0 None of them
   1 Few of them
   2 Half of them
   3 Most of them
   4 All of them

2. Lied, disobeyed, or talked back to adults such as parents, teachers, or others?
   0 None of them
   1 Few of them
   2 Half of them
   3 Most of them
   4 All of them

3. Purposely damaged or destroyed property that did not belong to them?
   0 None of them
   1 Few of them
   2 Half of them
   3 Most of them
   4 All of them

4. Stolen something worth less than $5?
   0 None of them
   1 Few of them
   2 Half of them
   3 Most of them
   4 All of them

5. Stolen something worth more than $5 but less than $100?
   0 None of them
   1 Few of them
   2 Half of them
   3 Most of them
   4 All of them

During the past 30 days how many of your friends have...

6. Stolen something worth more than $100?

0 None of them
1 Few of them
2 Half of them
3 Most of them
4 All of them

7. Gone into or tried to go into a building to steal something?

0 None of them
1 Few of them
2 Half of them
3 Most of them
4 All of them

8. Gone joyriding, that is, taken a car or motorcycle for a ride or drive without the owner's permission?

0 None of them
1 Few of them
2 Half of them
3 Most of them
4 All of them

9. Hit someone with the idea of hurting that person?

0 None of them
1 Few of them
2 Half of them
3 Most of them
4 All of them

10. Attacked someone with a weapon or with the idea of seriously hurting that person?

0 None of them
1 Few of them
2 Half of them
3 Most of them
4 All of them

11. Used a weapon or force to get money or things from people?

0 None of them
1 Few of them
2 Half of them
3 Most of them
4 All of them

During the past 30 days how many of your friends have...

12. Sold hard drugs such as heroin, cocaine, LSD, crystal or ecstasy?
    0 None of them
    1 Few of them
    2 Half of them
    3 Most of them
    4 All of them

13. Used alcohol?
    0 None of them
    1 Few of them
    2 Half of them
    3 Most of them
    4 All of them

14. Used marijuana or hashish?
    0 None of them
    1 Few of them
    2 Half of them
    3 Most of them
    4 All of them

15. Used hard drugs such as heroin, cocaine, LSD, crystal, or ecstasy?
    0 None of them
    1 Few of them
    2 Half of them
    3 Most of them
    4 All of them

16. Smoked cigarettes?
    0 None of them
    1 Few of them
    2 Half of them
    3 Most of them
    4 All of them

17. Have you ever been a gang member?
    0 No
    1 Yes

18. Are you now a gang member?
    0 No
    1 Yes

19. Are/were any of your CLOSE friends members of a gang?
    0 No
    1 Yes

Appendix B

SELF-REPORT DELINQUENCY SCALE
Youth Report

Directions: This questionnaire deals with your own behavior. All your answers are confidential. Please give your best estimate of the exact number of times you've done each of these in the last 30 days. Please refer to the calendar we made together. Write the correct number in the space under the statement.

For example, let's say you were given the item:

How many times in the past 30 days have you:

Been in a fight at school?

If you have been in 10 fights over the last 30 days, then you would write the number "10" in the space. If you have done something more than 100 times write "99".

How many times in the past 30 days have you:

1. Purposely damaged or destroyed property belonging to your parents or other family members.

2. Purposely damaged or destroyed property belonging to a school.

3. Purposely damaged or destroyed other property that did not belong to you (not counting family or school property).

4. Stolen (or tried to steal) a motor vehicle, such as a car or motorcycle.

5. Stolen (or tried to steal) something worth more than $50.

6. Knowingly bought, sold or held stolen goods (or tried to do any of these things).

7. Thrown objects (such as rocks or bottles) at cars or people.

8. Run away from home.

9. Lied about your age to gain entrance or to purchase something; for example, lying about your age to buy liquor or get into a movie.

10. Carried a hidden weapon other than a plain pocket knife.

11. Stolen (or tried to steal) things worth $5 or less.

12. Attacked someone with the idea of seriously hurting or killing him or her.

13. Been paid for having sexual relations with someone.
How many times in the past 30 days have you:

14. Had sexual intercourse with a person of the opposite sex other than your steady boyfriend/girlfriend.

15. Been involved in gang fights.

16. Sold marijuana or hashish ("pot", "grass", "hash").

17. Cheated on school tests.

18. Hitchhiked where it was illegal to do so.

19. Stolen money or other things from your parents or other members of your family.
20. Hit (or threatened to hit) a teacher or other adult at school.

21. Hit (or threatened to hit) one of your caregivers.

22. Hit (or threatened to hit) other students.

23. Been loud, rowdy, or unruly in a public place (disorderly conduct).

24. Sold hard drugs such as heroin, cocaine, and LSD.

25. Taken a vehicle for a ride (drive) without the owner's permission.

26. Bought or provided liquor for a minor.

27. Had (or tried to have) sexual relations with someone against their will.

28. Used force (strong-arm methods) to get money or things from other students.

29. Used force (strong-arm methods) to get money or things from a teacher or other adult at school.

30. Used force (strong-arm methods) to get money or things from other people (not students or teachers).

31. Avoided paying for such things as movies, bus rides and food.

32. Been drunk in a public place.

33. Stolen (or tried to steal) things worth between $5 and $50.

How many times in the past 30 days have you:

34. Stolen (or tried to steal) something at school such as someone's coat from a classroom, locker, cafeteria or a book from the library.

35. Broken into a building or vehicle (or tried to break in)to steal something or just to look around.

36. Begged for money or things from strangers.

37. Skipped classes without an excuse.

38. Failed to return extra change that a cashier gave you by mistake.

39. Been suspended from school.

40. Made obscene telephone calls, such as calling someone and saying dirty things.

Appendix C

HEALTH QUESTIONNAIRE

Now we are going to ask you some questions about your health and about medicines you might have taken today.

1. What time did you wake up today? _____ (24 Hour Clock/Military time)

2. How many hours of sleep did you get last night? _____hours

3. How many hours of sleep do you usually get? _____hours

4. Did you smoke a cigarette today?
0 No
1 Yes

5. Did you chew tobacco today?
0 No
1 Yes

The RA will help you with the next few questions. These questions are about medicines you take. Please let the RA know that it is time to do the medicine questions.

6. Did you take any prescription medications today?
0 No (skip to question 7)
1 Yes

Which prescription medication did you take today?
_____

7. Did you take any stimulants today to get high?
Some common stimulants are Meth (methamphetamine), Cocaine/Crack, Ecstasy, Amphetamines, Diet Pills/Slimming Tablets.
0 No
1 Yes

8. Did you use any marijuana today?
0 No
1 Yes

9. Did you take antihistamines today?
Some common antihistamines are Diphenhydramine (Benadryl),Chlorpheniramine (Chlor-Trimeton), Clemastine (Tavist),Loratadine (Claritin), Fexofenadine (Allegra), Cetirizine (Zyrtec),and Azelastine (Astelin).
0 No 1 Yes

10. Did you take steroids today?
Some common steroids are Prednisone, Prednisolone, Methylprednisolone, Betamethasone, Triamcinolone, and Hydrocortisone.
0 No
1 Yes

11. Did you take any medications to help you sleep last night?
0 No
1 Yes

12. Did you take allergy medications today?
0 No
1 Yes

13. Did you take cold/flu medication today?
0 No
1 Yes

14. Do you currently have a cold or flu?
0 No
1 Yes

15. Have you had any caffeine today (coffee, soft drinks, chocolate)?
0 No
1 Yes

When (time and day) did you last have caffeine (coffee, soft drinks, chocolate)? _____ (24 Hour Clock/Military Time)

16. What time did you last eat something? _____ (24 Hour Clock/Military Time)

17. Have you done any aerobic activity (running, brisk walking, bike riding, etc.) in the past two hours?
0 No
1 Yes

Instructions:
All boys change and develop physically, mentally, and emotionally in the process of "growing up." The growth and development of the body is an especially important part of this process. It is normal for different boys to go through this development at different ages. While answering these questions about your development, it is important to remember that no one will see these answers other than the researchers doing the study. Please be as honest as possible; honest answers will help us learn about boys your age.

To answer each question, please circle the number in front of the answer that best describes your development.

18a. Would you say your growth spurt in height spurt means more growth than usual)...
1 Has not begun
2 Has barely begun
3 Is definitely underway
4 Seems completed

18b. And how about the growth of your body hair e.g., underarm or pubic hair)? Would you say that yours...
1 Has not begun
2 Has barely begun
3 Is definitely underway
4 Seems completed

18c. Have you noticed any skin changes, especially pimples?
1 No changes
2 Change have barely begun
3 Changes are definitely underway
4 Changes seem completed

18d. Have you noticed a deepening of your voice?
1 Has not yet started changing
2 Has barely started changing
3 Change is definitely underway
4 Change seems completed

18e. Have you begun to grow hair on your face?
1 Have not yet started growing hair
2 Have barely started growing hair
3 Facial hair growth is definitely underway
4 Facial hair growth seems completed

Appendix D

Interpretation of betas for simple effects depends on the direction of the growth trajectories for the baseline models. It is important to understand that a significant beta indicates that at a given level of a moderator term, the independent variable (IV) is significantly associated with the slope parameter (i.e., the coefficient representing the association between time and the dependent variable). With this in mind, interpretation of betas for simple effects is as follows: for a dependent variable (DV) that increases over time, a positive beta suggests that higher values of the IV are associated with a greater rate of change in the DV; and a negative beta suggests that higher values of the IV are associated with a smaller rate of change in the DV. On the other hand, for a DV that decreases over time, a positive beta suggests that higher values of the IV are associated with a smaller rate of change in the DV; and a negative beta suggests that higher values of the IV are associated with a greater rate of change in the DV.

The reverse of these is true and the t-ratio is used when interpreting the co-variation between two variables at Level 1 in HLM analyses (i.e., Hypothesis 3). More specifically, for a DV that increases over time, a positive t-ratio for the IV indicates that a greater decline in the IV across time points is associated with smaller rate of positive change in the DV; and a negative t-ratio for the IV indicates that a greater decline in the IV across time points is associated with a greater rate of positive change in the DV.

On the other hand, for a DV that decreases over time, a positive t-ratio for the IV indicates that a greater decline in the IV across time points is associated with a greater rate of negative change in the DV; and a negative t-ratio for the IV indicates a smaller rate of negative change in the DV.

To the extent that the covariate increases or decreases across time points, the DV will be that much higher or lower at the time point, causing deviation from the average trajectory of change. In terms of simple effects, the value of the beta indicates the direction of the moderating term, which is interpreted within the context of the t-ratio.

Footnote

[1] In addition to basal testosterone levels, several measures of testosterone were considered for each hypothesis: a measure of testosterone following a stressor task, the difference between testosterone levels before and after a stressor task, the ratio of testosterone and cortisol before a stressor task, a ratio of testosterone and cortisol after a stressor task, and area under the curve. Results revealed largely non-significant results, thus in line with the existing literature, basal testosterone was chosen as the independent measure of testosterone for this study.

[2]Natural transitions prevented some youth from remaining with the same therapist throughout the study. For one subject, the therapist declined participation in the study after intake. Given that this study was not interested in Level 3 (therapist variables) effects, a unique therapist identification number was assigned to this youth for purposes of nesting within the HLM models. For eight of the families, their therapist switched during treatment. To account for differences in nesting within therapist across time, two Level 3 therapist identification variables were created. One variable represented the therapist that families met with the greatest number of times. Presumably that therapist would have the biggest impact given more frequent contact. The second variable represented the subsequent therapist that families met with during treatment. Analyses were first conducted with adolescents nested within the therapist that their family was treated by the greatest number of times. All analyses were re-run to examine whether results changed when nesting these adolescents within their other therapist. All results remained the same, thus the nesting of adolescents within therapist that families saw the greatest number of times is reported throughout.

[3] A similar approach to determining the best fitting baseline model was taken for all subsequent analyses.

Table 1

Mean Testosterone Levels of Participants for Each Provider Agency

	Basal Testosterone	
Agency	Mean (SD)	Sample Size
Synergy	62.95 (38.89)	58
Savio	43.32 (36.82)	26
JCMH	71.81 (39.29)	12
UCH	45.05 (38.20)	7

Table 2

Pearson Product Moment Inter-correlations Between Testosterone Levels, Height, Weight, and Body Mass Index

Measure	1	2	3	4
1. Testosterone	----			
2. Height	.11	----		
3. Weight	.04	**.52****	----	
4. Body Mass Index	.01	-.01	**.85****	----

* $p < .05$. **$p < .01$.

Table 3

Descriptive Statistics

	M	%	SD	Minimum	Maximum
Age	15.44		1.28	12	17
Younger Youth (12-14) (N=27)		22.3			
Older Youth (15-17) (N=93)		76.9			
Puberty	2.83		0.62	1.00	4.00
Race					
Minority (African-American, Latino, "Other") (N= 58)		47.9			
European-American (N= 62)		51.2			
Basal Testosterone	57.82		39.15	4.27	188.12
Basal Cortisol	0.41		0.27	0.10	1.49
Deviant Peer Affiliation	9.27		10.40	0	48.00
Parenting, Youth Report	122.98		19.29	76.00	173.00
Parenting, Caregiver Report	136.72		15.38	98.00	170.00
SRD General Delinquency	24.03		48.29	0	362.00
CBCL Aggression (T-score)	61.26		10.96	50.00	87.00
CBCL Delinquency (T-score)	66.69		9.89	50.00	94.00

Table 4

Pearson Product Moment Inter-correlations Between Independent and Dependent Variables Used in the Analyses

Measure	1	2	3	4	5	6	7	8
1. Basal Testosterone	----							
2. Basal Cortisol	.07	----						
3. Deviant Peer Affiliation	-.05	-0.03	----					
4. Parenting , Youth Report	.07	0.05	**-.38****	----				
5. Parenting, Caregiver Report	-.07	0.07	**-.25****	**.32****	----			
6. Log Transformation of SRD General Delinquency	.05	-0.10	**.51****	**-.30****	-.11	-----		
7. CBCL Aggression	**-.20***	-0.06	**.23***	**-.30****	**-.21***	**.26****	----	
8. CBCL Delinquency	-.10	-.10	**.24***	**-.36****	**-.34****	**.35****	**.70****	-----

* $p < .05$. **$p < .01$.

Table 5

Pearson Product Moment Intercorrelations Between Demographic Variables and Dependent Variables Used in the Analyses

Measure	1	2	3	4	5
1. Age	----				
2. Puberty	**.40****	----			
3. Log Transformation of SRD General Delinquency	-.06	.03	----		
4. CBCL Aggression	**-.18***	-.15	**.26****	----	
5. CBCL Delinquency	.00	.02	**.35****	**.70****	----

* $p < .05$. **$p < .01$.

Table 6

Spearman Intercorrelations Between Categorical Demographic Variables and Dependent Variables Used in the Analyses

Measure	1	2	3	4
1. Race[a]	----			
2. Log Transformation of SRD General Delinquency	.14	----		
3. CBCL Aggression	**.29****	.27**	----	
4. CBCL Delinquency	**.25***	.39**	.78**	----

Note. [a]The race variables was coded as "0" Minority (African-American, Latino & "Other") and "1" European-American.

* $p < .05$. ** $p < .01$.

Table 7

Summary of Direct Effects Using Multiple Regression for Basal Testosterone Predicting SRD General Delinquency (N=101)

	Model 1			Model 2		
Predictor	*B*	*SE B*	β	*B*	*SE B*	β
Age	-0.02	0.05	-0.04	-0.05	0.06	-0.08
T Assay Number	0.07	0.05	0.15	0.08	0.05	0.18*
Basal T				0.00	0.00	0.12
ΔR^2		0.03			0.01	
F for change in R^2		1.26			1.23	

* $p < .10$. ** $p < .05$. ***$p < .01$.

Table 8

Summary of Direct Effects Using Multiple Regression for Basal Testosterone Predicting CBCL Aggression (N=101)

	Model 1			Model 2		
Predictor	*B*	*SE B*	β	*B*	*SE B*	β
Age	-1.78	0.77	-0.22**	-1.29	0.83	-0.16
Race[a]	5.22	2.04	0.25***	5.15	2.03	0.24***
T Assay Number	-0.45	0.64	-0.07	-0.71	0.66	-0.11
Basal T				-0.04	0.03	-0.16
ΔR^2		.11			.02	
F for change in R^2		4.17***			2.44	

Note: [a]The race variables was coded as "0" Minority and "1" European-American

* $p < .10$. ** $p < .05$. ***$p < .01$.

Table 9

Summary of Direct Effects Using Multiple Regression for Basal Testosterone Predicting CBCL Delinquency (N=96)

	Model 1			Model 2		
Predictor	*B*	*SE B*	β	*B*	*SE B*	β
Age	-0.37	0.72	-0.05	-0.12	0.78	-0.02
Race	2.87	1.89	0.16	2.90	1.90	0.16
T Assay Number	0.21	0.59	0.04	0.10	0.60	0.02
Basal T				-0.02	0.03	-0.10
ΔR^2		.03			.01	
F for change in R^2		0.92			0.72	

Note: [a]The race variables was coded as "0" Minority and "1" European-American

* $p < .10$. ** $p < .05$. ***$p < .01$.

Table 10

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	61.08	45.44	75.60	58.27			70.57	75.80
Basal Cortisol	0.43	0.34						
Deviant Peer Affiliation	9.12	9.97						
Parenting, Youth Report	124.35	18.8						
Parenting, Caregiver Report	137.14	15.56						
Caregiver Report of Therapist Adherence			13.63	8.51	12.07	8.95	12.76	9.74
SRD General Delinquency	32.16	103.93	8.94	17.05	8.72	19.89	11.05	29.59
CBCL Aggression	60.86	10.89	56.42	8.39	56.98	8.20	56.30	8.70
CBCL Delinquency	66.26	9.51	62.29	8.59	61.48	8.90	62.16	10.20

Table 11

Pearson Product Moment Intercorrelations Among Study Variables

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Time 1, T	-----															
2. Time 2, T	**.38****	-----														
3. Time 4, T	.11	**.53****	-----													
4. Cortisol	.05	-.03	-.03	-----												
5. PDS	-.05	**.22***	**.20***	-.03	-----											
6. Parenting, Youth	.01	-.14	.00	-.05	**-.38****	-----										
7. Parenting, Cg	-.02	-.11	-.01	.00	**-.20***	**.31****	-----									
8. Time 2 TAM-R	-.12	-.17	-.07	**-.23***	**-.30****	**.30****	**.43****	-----								
9. Time 3 TAM-R	-.18	-.14	-.19	-.17	**-.32****	.20	**.36****	**.71****	-----							
10. Time 4 TAM-R	-.17	-.20	**-.23***	-.16	**-.20***	.10	**.27****	**.58****	**.84****	-----						
11. Time 1 SRD	.09	.06	-.13	-.08	**.20***	-.08	-.13	-.13	.04	.07	-----					
12. Time 2 SRD	.09	.02	-.08	.02	**.25***	**-.22***	**-.27***	-.13	-.10	-.17	.20	-----				
13. Time 3 SRD	-.15	-.09	**.38****	-.07	**.36****	-.23	**-.26***	-.13	-.19	-.18	.01	.17	-----			
14. Time 4 SRD	-.11	.17	-.03	.16	.10	**-.22***	**-.20***	-.19	-.08	-.08	.14	**.26***	.15	-----		
15. Time 1 CBCL Agg.	-.18	.12	-.04	-.06	**.23***	**-.30****	-.19	-.03	.11	-.13	-.02	.16	.01	.13	-----	
16. Time 2 CBCL Agg.	**-.26***	-.01	-.03	-.10	.16	**-.27****	-.18	.01	.15	-.05	-.01	.20	.14	.16	**.69****	-----
17. Time 3 CBCL Agg.	-.16	-.10	-.05	-.20	.23	**-.25***	-.17	.15	.03	-.09	.05	.20	.04	**.26***	**.64****	**.72****
18. Time 4 CBCL Agg.	-.17	.17	.04	.04	**.31****	**-.27****	-.11	-.07	.12	-.17	.03	.18	.01	**.29****	**.65****	**.74****
19. Time 1 CBCL Del.	-.11	.16	.07	-.03	**.23***	**-.36****	**-.30****	-.14	.04	-.05	-.02	**.27***	.05	**.27****	**.69****	**.52****
20. Time 2 CBCL Del.	**-.25***	-.05	-.06	-.05	.19	**-.28****	**-.30****	-.12	.00	-.03	-.04	**.31****	.12	**.22***	**.50****	**.73****
21. Time 3 CBCL Del.	.00	-.18	.12	-.04	.20	**-.25***	**-.33***	-.07	-.18	-.24	-.06	**.33****	.05	**.42****	**.44****	**.56****
22. Time 4 CBCL Del.	-.17	-.04	-.09	-.05	**.25***	**-.23***	-.19	-.11	-.14	-.12	.07	**.35****	.10	**.35****	**.31****	**.51****
23. Age	**.24***	**.24***	**.33****	.06	.16	-.00	-.12	-.11	**-.25***	**-.19***	-.05	.14	.09	-.04	-.18	**-.27****
24. Race	-.04	.14	.06	-.07	.14	**-.22***	-.02	-.13	-.10	-.09	.15	.06	.04	.13	**.29****	**.21***

Note. T= testosterone. PDS= Peer Delinquency Scale. Youth= Youth Report. Cg= Caregiver report. Race = "0" Minority, "1" European-American

* $p < .05$. **$p < .01$.

17	18	19	20	21	22	23	24

.71**	-----						
.41**	**.50****	-----					
.57**	**.62****	**.59****	-----				
.66**	**.48****	**.56****	**.71****	-----			
.59**	**.68****	**.46****	**.72****	**.63****	-----		
-.11	-.12	.00	-.15	.08	-.10	-----	
.27*	**.22***	**.26****	.17	.16	.18	.04	-----

Table 12

Testosterone X Cortisol Predicting Caregiver Report of Therapist Adherence

	Caregiver Report of Therapist Adherence at Time 2				
Effect	Coefficient	SE	*t (108)*	*effect size r*	*p*
Intercept (Adherence at T2)					
Age	-0.65	0.50	-1.30	0.12	0.20
Assay Number	-0.13	0.50	-0.26	0.03	0.80
Testosterone	-0.02	0.02	-0.95	0.09	0.35
Cortisol	-10.26	2.26	-4.55	0.40	**0.00**
Testosterone X Cortisol	-0.93	0.67	-1.40	0.13	0.17
Slope (Changes in Adherence)					
Age	-0.00	0.00	-0.69	0.07	0.49
Assay Number	0.00	0.00	0.69	0.07	0.49
Testosterone	-0.00	0.00	-0.62	0.06	0.54
Cortisol	-0.03	0.03	-1.06	0.10	0.30
Testosterone X Cortisol	-0.01	0.01	-2.07	0.20	**0.05**

Table 13

Testosterone X Deviant Peer Affiliation Predicting CBCL Aggression and CBCL Delinquency

	CBCL Aggression					CBCL Delinquency				
Effect	Coefficient	SE	*t (107)*	*effect size r*	*p*	Coefficient	SE	*t(107)*	*effect size r*	*p*
Intercept										
Age	-1.8	0.66	-2.71	0.25	**0.01**	-0.43	0.50	-0.86	0.08	0.40
Race[a]	5.38	1.91	2.82	0.26	**0.01**	3.82	1.68	2.27	0.21	**0.03**
Assay Number	-0.67	0.52	-1.3	0.12	0.20	0.14	0.53	0.28	0.03	0.78
Testosterone	-0.03	0.02	-1.49	0.14	0.14	-0.02	0.02	-1.02	0.10	0.31
Deviant Peer Affiliation	0.22	0.07	2.95	0.27	**0.00**	0.09	0.09	1.01	0.10	0.32
Testosterone X Deviant Peer	-0.79	1.05	-0.75	0.07	0.46	-0.20	0.93	-0.22	0.02	0.83
Slope										
Age	0.00	0.00	1.07	0.10	0.29	0.00	0.01	0.50	0.05	0.62
Race[a]	-0.02	0.01	-1.80	0.17	***0.08***	0.00	0.01	0.33	0.03	0.74
Assay Number	-0.00	0.00	-0.08	0.01	0.93	-0.01	0.00	-1.64	0.16	0.11
Testosterone	0.00	0.00	0.68	0.07	0.50	-0.00	0.00	-0.75	0.07	0.46
Deviant Peer Affiliation	-0.00	0.00	-0.21	0.02	0.84	0.00	0.00	2.66	0.25	**0.01**
Testosterone X Deviant Peer	0.01	0.00	2.54	0.24	**0.02**	0.01	0.01	2.27	0.21	**0.03**

Note: [a]The race variables was coded as "0" Minority and "1" European-American

Table 14

Testosterone X Caregiver Report of Parenting Predicting CBCL Delinquency

	CBCL Delinquency				
Effect	Coefficient	SE	*t(107)*	*effect size r*	*p*
Intercept					
Age	-0.62	0.49	-1.26	0.12	0.21
Race[a]	4.09	1.58	2.59	0.24	**0.01**
Assay Number	0.23	0.54	0.44	0.04	0.66
Testosterone	-0.02	0.02	-1.22	0.12	0.23
Parenting	-0.20	0.05	-3.76	0.34	**0.00**
Testosterone X Parenting	0.12	0.65	0.18	0.02	0.86
Slope					
Age	0.00	0.01	0.77	0.07	0.45
Race[a]	-0.01	0.01	-1.31	0.13	0.20
Assay Number	-0.00	0.00	-1.43	0.14	0.16
Testosterone	-0.00	0.00	-0.66	0.06	0.51
Parenting	-0.00	0.00	-0.49	0.05	0.63
Testosterone X Parenting	0.01	0.00	2.12	0.20	**0.04**

Note: [a]The race variables was coded as "0" Minority and "1" European-American

Figure 1

Simple slope plot illustrating the interaction of Time 1 testosterone and Time 1 cortisol on changes in caregiver report of therapist adherence over the course of treatment.

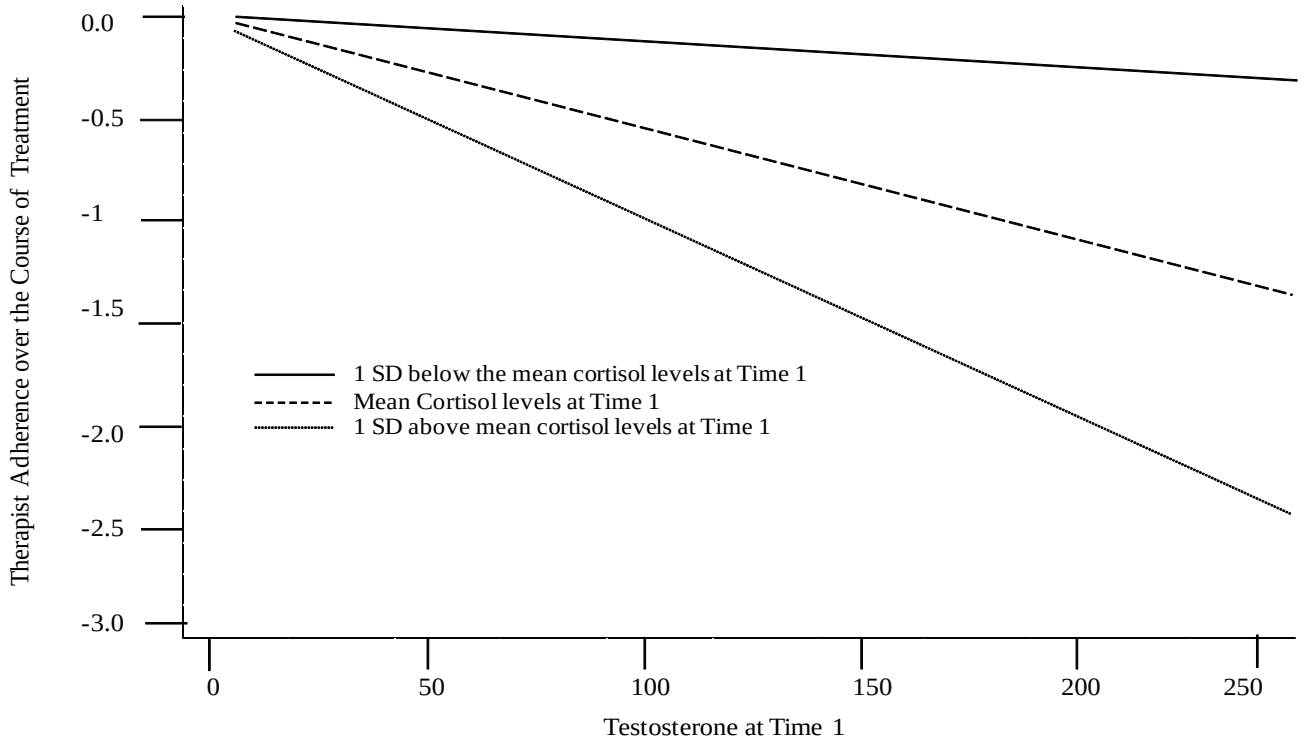

Figure 2

Simple slope plot illustrating the interaction of Time 1 testosterone and Time 1 deviant peer affiliation on changes in CBCL aggression over the course of treatment.

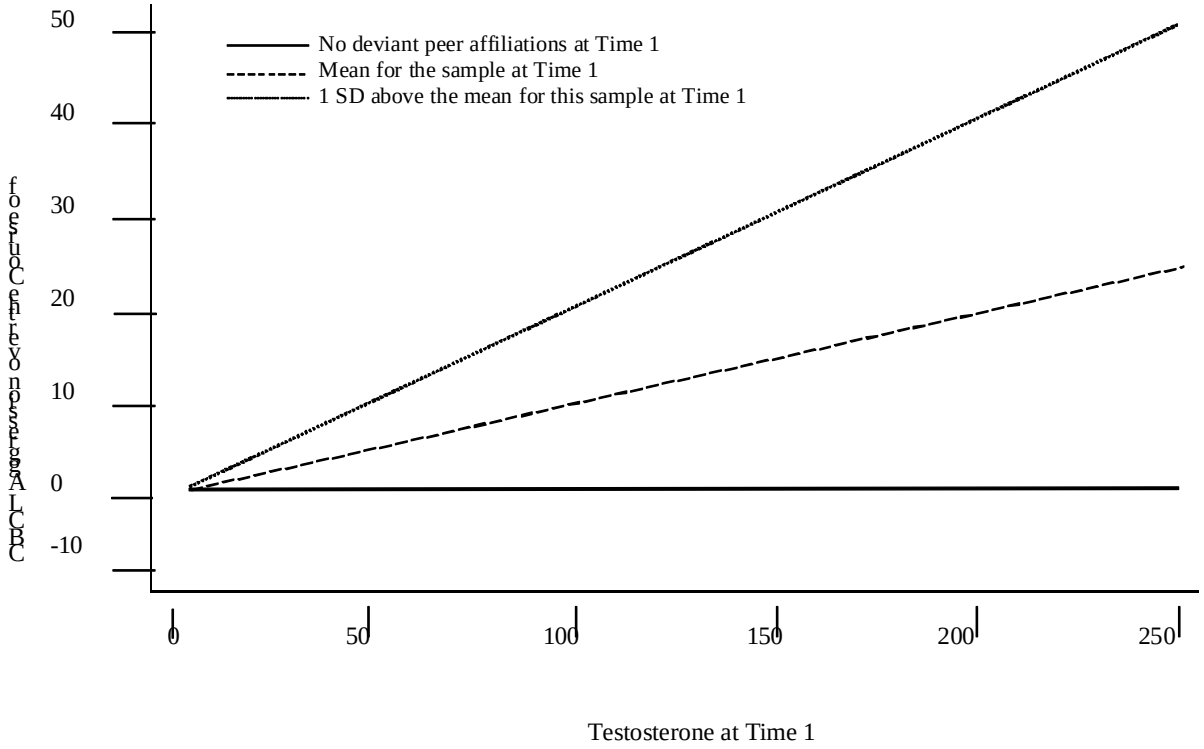

Figure 3

Simple slope plot illustrating the interaction of Time 1 testosterone and Time 1 deviant peer affiliation on change in CBCL delinquency over the course of treatment.

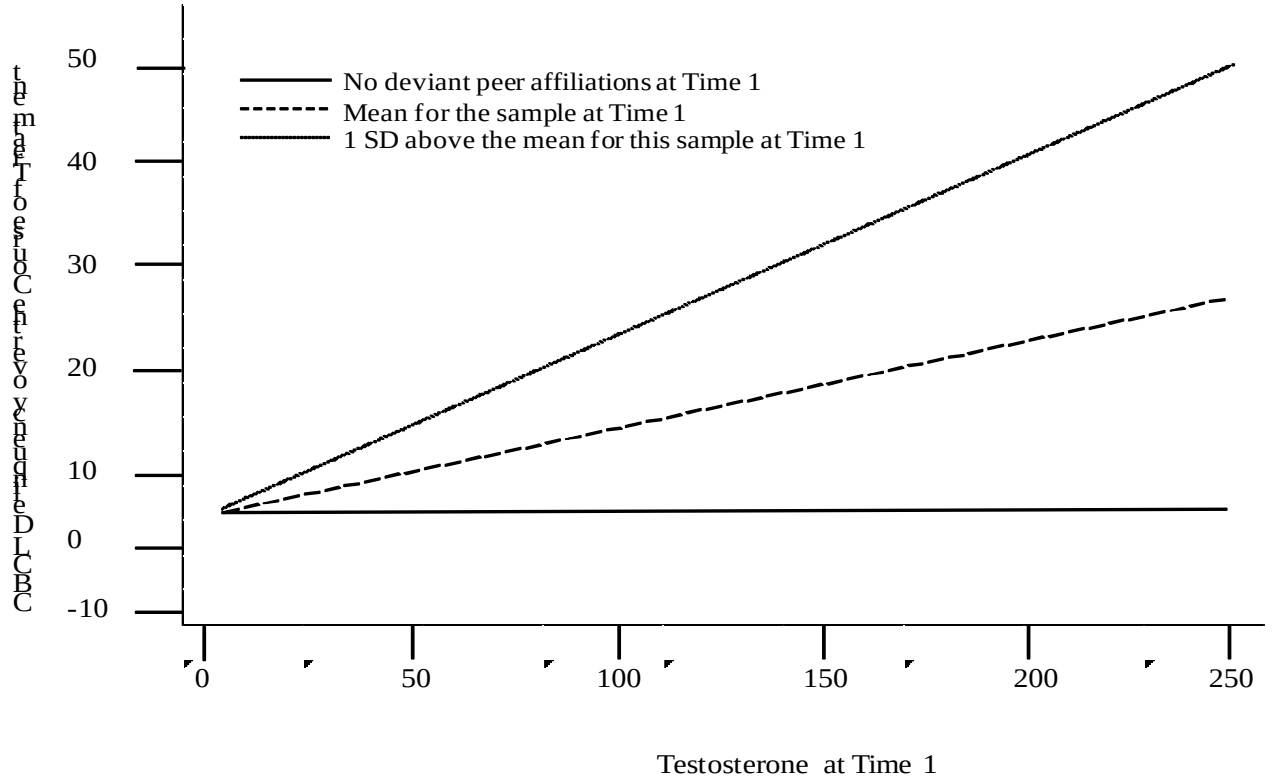

Figure 4

Simple slope plot illustrating the interaction of Time 1 testosterone and Time 1 caregiver report of parent-child relationship quality on changes in CBCL delinquency over the course of treatment.