

Testosterone, Antisocial Behavior, and Social Dominance in Boys: Pubertal Development and Biosocial Interaction

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Background: Studies linking testosterone and antisocial behavior in humans have produced mixed results. Adolescence offers a promising period to study this relationship; circulating testosterone increases dramatically in boys during puberty, and antisocial behavior increases during the same period.

Methods: Our analyses were based on boys aged 9–15 years who were interviewed during the first three waves of the Great Smoky Mountains Study. Measures included interview assessment of DSM-IV conduct disorder (CD) symptoms and diagnosis, blood spot measurement of testosterone, Tanner staging of pubertal development, and assessment of leadership behaviors and peer deviance.

Results: The adolescent rise in CD was primarily attributable to an increase in nonphysically aggressive behaviors. Increasing levels of circulating testosterone and association with deviant peers contributed to these age trends. There was no evidence that physical aggression was related to high testosterone. Evidence of biosocial interactions was identified; testosterone was related to nonaggressive CD symptoms in boys with deviant peers and to leadership in boys with nondeviant peers.

Conclusions: The results are consistent with the hypothesis that testosterone relates to social dominance, with the assumption that behaviors associated with dominance differ according to social context.

Key Words: Testosterone, dominance, puberty, conduct disorder, biosocial, antisocial

Gender differences in forms and rates of physical aggression, risk taking, and sexual violence long have stimulated scrutiny of the role of testosterone in social behaviors. Sustained investigation has gradually revealed a more complex picture than was initially anticipated, with expectations that testosterone would relate directly to gender-differentiated behavior (e.g., aggression) receiving only partial empiric support. Early correlational studies of nonhuman primates identified direct associations of testosterone with aggression (Rose et al 1971) but could not distinguish the extent to which the association was a consequence or a cause of the behavior (Rose et al 1972; Sapolsky and Ray 1989). Experimental studies have indicated that increased testosterone leads to increased aggression in a range of species (Monaghan and Glickman 1992) including humans (Book et al 2001). But more fine-grained naturalistic studies in nonhuman primates have shown that the relationships of testosterone to social dominance and aggressive behavior depend on social context. Correlation of testosterone with dominance ranking among savanna baboons, for example, differs by degree of social stability and ecologic stress; relationships of testosterone to aggression and dominance emerge only under conditions of social disruption, marginalization, and resource uncertainty (Sapolsky 1993). Relationships between hormones and behavior in humans are therefore expected to be reciprocal and informed by social as well as developmental factors (Mazur

and Booth 1998). For example, it has been found that testosterone levels fall during the years surrounding marriage and increase around the time of divorce (Mazur and Michalek 1998). It is to be expected, therefore, that failure to consider potential moderating effects of the perceived social situation will result in confused findings.

A recent meta-analysis of correlational studies from community and selected samples reported a statistically significant mean weighted correlation of .14 between testosterone and aggression in males (Book et al 2001). The authors noted, however, that exclusion of published reports with inadequate statistical details about nonsignificant findings, together with a bias toward publishing significant results, might have inflated the apparent strength of the relationship. Mazur and Booth (1998) have also emphasized the possibility that testosterone is not related to aggression per se, but to the achievement or maintenance of high social status. For example, they note that competitive success, such as winning at tennis (Booth et al 1989) or chess (Mazur et al 1992), has been associated with transient increases in circulating testosterone.

Antecedent individual differences also contribute to distinctive responses to experimental manipulation of testosterone. A recent study, for example, found that increasing testosterone under prolonged pharmacologic treatment was associated with increasing aggression scores during a competitive computer-based game (Pope et al 2000). Yet the average effect was driven by a small group of “responders,” a 16% minority who manifested notable effects of testosterone treatment, whereas the rest showed minimal effects. All of these findings suggest that hormone–behavior linkages are likely to be complex.

In adolescence, testosterone has generally been used as an index of pubertal maturation, but any direct effect of testosterone on behavior is difficult to disentangle from the cognitive, social, other neuroendocrine, and morphologic changes of puberty. A link is certainly plausible at this stage, because there is a dramatic rise in circulating testosterone during puberty coincident with a well-documented increase in antisocial behavior (Maughan et al, in press; Rutter et al 1998). Once again, however, it is unclear whether physical aggression will be the key correlate. According to the influential dual-pathway model of Moffitt (1993), adoles-

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Received April 15, 2003; revised September 16, 2003; accepted October 15, 2003.

cent onset of antisocial behavior is characterized by nonphysically aggressive behaviors, such as vandalism and status violations, whereas childhood-onset antisocial behavior will feature more physical aggression during the teens. Because physical aggression tends to become *less* common during the adolescent period (Tremblay 2000), it might be that testosterone is associated with nonphysically aggressive antisocial behaviors rather than with physical aggression per se.

Studies of the relationship between testosterone and antisocial behavior in adolescence have produced mixed results that are at least partly attributable to study design and technical issues (Halpern et al 1998). An early study of 15–17-year-old boys reported patchy cross-sectional relationships between testosterone and certain types of aggression (Olweus et al 1988). Udry (1990) found a relationship between testosterone and antisocial behavior in boys aged 12 to 13 years. A further longitudinal study found that high testosterone at 12 years predicted later antisocial behavior (Drigotas and Udry 1993), but the absence of cross-sectional relationships between testosterone and antisocial behavior at older ages (15 to 16 years) contradicted direct effects of testosterone on problem behavior. Tremblay et al (1998) found that high testosterone at age 12 did not predict aggression at age 13 but did predict peer-rated social dominance. Although all these studies were conducted in the community, sample sizes were generally low (57–126 boys), and this might have contributed to the inconsistency of the findings.

A further issue concerns the moderating role of social context in adolescence. Biosocial interactions have attracted increasing attention in developing models of risk for antisocial behavior (Dodge and Pettit 2003; Raine 2002), but few studies have examined hormonal influences from this perspective. One recent report has, however, established the utility of this approach. In a study of established two-parent families including boys aged 8–18 years, the quality of parent–child relationships moderated the association of the son's testosterone with risk behavior. A high-quality parent–son relationship diminished the impact of testosterone on risk behavior, whereas poor parent–son relationships strengthened this link (Booth et al 2003). In a similar way, studies of men suggest the potential for feed-forward effects of early-established emotional–behavioral regulation and later capacity for social integration via moderation of endocrine–behavior interaction. For instance, in the Vietnam Veterans' Experience Study, the relationship between adult deviance and testosterone was moderated by social integration as indexed by educational achievement, participation in organized groups, job stability, and marital status (Booth and Osgood 1993). This study found that testosterone was an important risk factor for adult deviance for men who were not well integrated into society but that the relationship between testosterone and deviance was much weaker for well-integrated men. In a similar vein, Susman (1997) highlights the possibility that the peer group context might play an important role in the complex relationships between hormones and behavior during adolescence. A large body of literature indicates that association with deviant peers is a strong correlate of, and influence on, the development of antisocial behaviors in the teens (Moffitt 1993; Thornberry and Krohn 1997). If testosterone is involved in dominance, we might thus expect the characteristics of the peer group to have an important effect on the mechanisms through which dominance is achieved.

Against this background of emerging biosocial models, the study reported here aimed to evaluate the relationship of testosterone with antisocial behavior and social dominance in a large study of male adolescents (the Great Smoky Mountains Study

[GSMS]). First, the development of both conduct disorder (CD) and constituent symptom subtypes during puberty was examined. Second, we tested the extent to which developmental changes in CD behaviors could be attributed to social and biological correlates of puberty. Third, the hypothesis that testosterone is related to social dominance was directly examined. Fourth, we tested whether the correlates of testosterone differed as a function of peer-group deviance.

Methods and Materials

Sample

Based in a predominantly rural area of the southern United States, the GSMS is a longitudinal study of psychiatric disorder in children and adolescents. The accelerated cohort (Schaie 1965), two-phase sampling design, and measures are described in detail elsewhere (Costello et al 1996). Briefly, a representative sample of 4500 9-, 11-, and 13-year-old boys and girls resident in western North Carolina were selected with a household equal probability design. In the screening phase, a parent (usually the mother) completed a questionnaire containing items regarding behavioral disorders from the Child Behavior Checklist (Achenbach and Edelbrock 1983). The interview phase included all children scoring above a predefined cut-off on this screen (designed to identify the most pathologic 25% of the population), along with a 10% random sample of the remainder. All age-eligible American Indian children from the area were also recruited. Between 80% and 94% of those selected took part at each of the three annual interviews. The current analyses include all the boys from the first three waves of the study. This provides a data set that includes 2125 observations of 789 individuals.

The study received ethical approval from the Duke University Health System institutional review board. Separate informed consents for the interview, Tanner stage rating, and finger-stick portions of the study protocol were obtained from a parent or guardian, and complementary assents to each of these components were obtained from the children/adolescents.

Measures

Conduct Disorder Symptoms and Diagnosis. At each wave, the child and the primary caretaker (usually the mother) were separately interviewed with use of the Child and Adolescent Psychiatric Assessment (CAPA) (Angold and Costello 2000). The CAPA assesses the child's psychiatric status over the preceding 3 months according to DSM-IV criteria (American Psychiatric Association 1994). The CD diagnosis had a κ reliability of .55, and the CD symptom scale had an intraclass correlation of .62 in a sample of 77 clinically referred children interviewed on two occasions with the child version of the CAPA (Angold and Costello 1995). The standard "or" rule (Costello et al 1996; Simonoff et al 1997), whereby a symptom is endorsed if either the child or primary caretaker report meets the symptom threshold, was used to combine reports from the two informants.

As well as measuring DSM-IV CD diagnosis, we also analyzed symptom counts of behaviors involving physical aggression, referred to as aggressive CD symptoms, and behaviors that did not involve physical aggression, referred to as nonaggressive CD symptoms. The aggressive symptoms were bullying, fighting, weapon use, cruelty to people, cruelty to animals, stealing with confrontation, and forced sexual advances. The nonaggressive symptoms were lying, stealing without confrontation, breaking in, property damage, fire setting, running away, and truancy. The staying out late CD symptom was omitted from the nonaggressive

sive symptom count because it was not assessed at wave 1, which was conducted before DSM-IV was finalized.

Peer Deviance And Leadership. A self-completion questionnaire assessing the child's social functioning was presented at interview to both child and parent. The respondent was asked to indicate the extent to which statements about children in general were applicable to the target child by using a line bisection format. The line was 100 mm long; the leftmost end was labeled "Not at all" and the rightmost end was labeled "Exactly." Each response was measured in terms of its distance in millimeters away from the "Not at all" end of the line. We focused on items that measured peer deviance (child hangs out with a bad crowd) and leadership (often chosen by other children to be the leader). The exact wording of the questions is provided in Appendix 1. In the current sample, the correlations across study waves for parent and child ratings ranged from .34 to .53 for peer deviance, and from .39 to .70 for leadership. Parent and child ratings were averaged to produce a single score. The ratings were used as continuous scores and were also collapsed to form groups of boys who definitely had deviant peers, possibly had deviant peers, and definitely did not have deviant peers. The ratings were heavily skewed, with many boys rated as "not at all" the kind of child that has deviant peers. Therefore, splitting the continuum to identify boys who definitely did not have deviant peers inevitably produced a large group. We formed groups such that 33.1% of the sample definitely did not have deviant peers and 13.5% definitely had deviant peers. The remaining 53.4% made up a group with possibly deviant peers.

Pubertal Physical Development. Self-ratings of pubertal morphologic status, based on the standard Tanner staging system (Tanner 1962), were made with schematic drawings of pubertal development. This technique has been shown to correlate well with Tanner stages assessed from direct observation (e.g., Morris and Udry 1980). Ratings are made on a five-point scale ranging from I (prepubertal) to V (adult) developmental level, for both different amounts of male pubic hair and stage of growth of testes, scrotum, and penis. The two ratings (hair and genitals) were averaged to produce a single score.

Testosterone Measurement. Two finger-prick blood spot samples, taken 20 min apart, were collected on standardized filter papers at the beginning of the interviews. Samples were immediately refrigerated upon drying, express-shipped to the laboratory (without refrigeration) within 2 weeks of collection, and then stored at -23°C until analysis. The blood samples were assayed for a number of hormones, including testosterone, by immunometric methods described elsewhere (Worthman and Stallings 1997). Assessments of gonadal steroids in blood spots have been found to correlate better with psychobehavioral outcomes than do measures taken from saliva (Shirtcliff et al 2002). The testosterone value for each observation was taken as the average of the two samples to minimize the effects of pulsatility. Assay sensitivity was 6.3 ng/dL; coefficient of variation for low, medium, and high pools (mean value 52.0 ng/dL, 294.2 ng/dL, and 548.2 ng/dL, respectively) was 7.6%, 8.3%, and 7.0% within assays, and 13.9%, 12.3%, and 11.8% between assays, respectively.

Timing of blood sample collection was constrained by family convenience for scheduling an interview in their homes. Interview times ranged from 8:00 AM to 9:00 PM, with the majority (70%) conducted between 11:00 AM and 6:00 PM. As expected, overall testosterone levels showed diurnal variation, with higher levels in the morning and lower values in the evening; however, diurnal variation was dependent on developmental stage. Time

of measurement was related to testosterone level in those aged 13 and older ($r = -.17$, $p = .02$), whereas there was no relationship in children aged 12 and younger ($r = -.02$, $p = .6$). In addition, there was no significant variation in testosterone levels for older children in samples collected between 11:00 AM and 6:00 PM ($r = .01$, $p = .9$). We checked that diurnal variation in testosterone did not influence the results by rerunning key analyses in the subsample of boys whose data were collected between 11:00 AM and 6:00 PM. No substantive differences were found in these analyses. In addition, time of interview was unrelated to our measures of aggressive CD symptoms (proportional odds ratio [POR] = 1.0, $p = .7$), nonaggressive CD symptoms (POR = 1.0, $p = .9$) and leadership ($r = -.06$, $p = .2$).

Missing Data. As noted above, separate permissions were required from the parent and child for the Tanner stage ratings and finger-prick blood sampling. Tanner stage ratings were unavailable for between 8.2% and 11.3% of the observations at each wave. Missing Tanner data showed some relationship to lower rates of CD diagnosis (5.3% vs. 2.7%; $p = .053$), nonaggressive CD symptoms (means .36 symptoms vs. .21 symptoms; $p = .048$) and leadership (standardized means .03 vs. $-.30$; $p = .035$) but was unrelated to age (11.9 vs. 11.8 years; $p = .9$), aggressive CD symptoms (mean .19 symptoms vs. .18 symptoms; $p = .9$) or peer deviance (standardized means .01 vs. $-.12$; $p = .3$). Testosterone measurement was missing for between 19.3% and 26.8% of observations at each wave. Once again, missing data were negatively associated with CD diagnosis (5.6% vs. 2.9%; $p = .02$) and nonaggressive CD symptoms (mean .37 symptoms vs. .25 symptoms; $p = .04$) and positively associated with Tanner stage (mean Tanner stages 2.7 vs. 3.0; $p = .03$). There was no relationship with aggressive CD symptoms (.19 symptoms vs. .17 symptoms; $p = .6$), peer deviance (standardized means $-.01$ vs. .04; $p = .6$), leadership (standardized means .02 vs. $-.06$; $p = .4$), or age (11.8 vs. 12.0 years; $p = .3$).

Observations with missing testosterone data were excluded from all analyses, resulting in a data set of 1669 observations of 713 boys with complete data on all variables except Tanner stage. Analyses including Tanner stage were based on a further reduced data set of 1596 observations.

Statistical Analysis

The GSMS was approached as a cross-sectional time series data set and analyzed with the Survey models of Stata (StataCorp 2001). These models allow observations to be weighted to the inverse of their sampling probability (as required by the two-phase sampling design) and account for the correlations among sets of responses from the same individual to provide unbiased parameter estimates and standard errors. Diagnostic outcome was analyzed by logistic regression, and symptom count outcomes were analyzed by ordinal logistic regression. Ordinal logistic regression models calculate POR, which can be interpreted as the increase in odds of crossing any particular threshold on the dependent variable given a single unit rise in the independent variable. Testosterone levels and the peer deviance scale, both heavily skewed measures, were analyzed by ordinary linear regression with robust test statistics (Huber 1967), as was the normally distributed measure of leadership.

When included as predictors in regression models, testosterone, Tanner stage, and deviant peer relationships were standardized to facilitate comparison of effects. Leadership was also standardized when included as an outcome measure, because the original scale was not naturally meaningful. Unless otherwise stated, age was measured in rounded years.

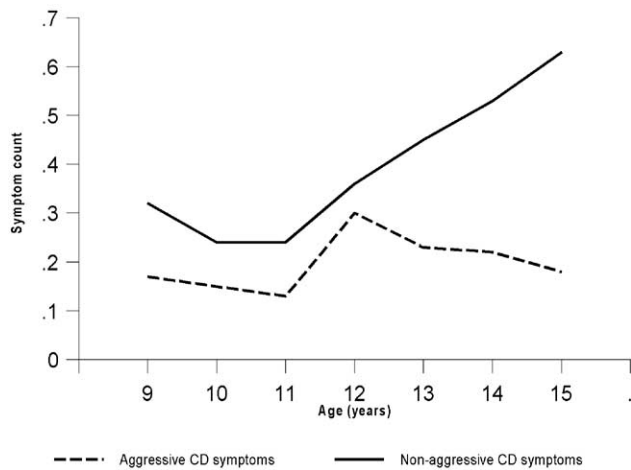


Figure 1. Mean aggressive and nonaggressive conduct disorder symptom counts across age.

Results

Developmental Trends in Antisocial Behavior

The sample comprised boys aged 9–15 years with percent-ages at Tanner stages I–V of 14.2, 33.2, 24.6, 22.0, and 6.0 respectively. Conduct disorder diagnosis showed a significant linear age trend (odds ratio [OR] = 1.3, 95% confidence interval [CI] 1.1, 1.5; $p = .006$), rising from 4% at age 9 to 10% at age 15. Figure 1 shows mean aggressive and nonaggressive CD symptom counts by age. These two subtypes of antisocial behavior showed different age trends: aggressive CD symptoms showed no evidence of linear change with age (POR = 1.0, 95% CI .9, 1.2; $p = .5$), but nonaggressive CD symptoms increased linearly with age (POR = 1.2, 95% CI 1.0, 1.3; $p = .005$).

We examined whether Tanner stage provided a better marker of development for nonaggressive CD symptoms than chronologic age. Tanner stage was a significant univariate predictor (POR = 1.3, 95% CI 1.0, 1.6; $p = .02$). When both age and Tanner stage were modeled jointly, however, the prediction from age (POR = 1.3, 95% CI 1.0, 1.7; $p = .07$) was better than that from Tanner stage (POR = 1.1, 95% CI .8, 1.4; $p = .7$). We therefore focused on chronologic age as our index of development in subsequent analyses.

Developmental Trends in Testosterone and Peer Deviance

As expected, the mean level of circulating testosterone increased dramatically across pubertal development, rising from a mean of 6 ng/dL at age 9 to 115 ng/dL and 364 ng/dL at ages 13 and 15, respectively. Association with deviant peers also increased across puberty; at age 9, 8.0% of boys definitely had deviant peers, and the proportion increased to 14.9% and 25.4% at ages 13 and 15, respectively. Both of these factors showed significant strong linear relationships with age; being 1 year older was associated with a 54-ng/dL (95% CI 46.8, 61.3; $p < .001$) increase in circulating testosterone and an OR of 1.3 (95% CI 1.1, 1.5; $p = .003$) of definitely having deviant peers.

Developmental Predictors of Nonaggressive and Aggressive CD Symptoms

We went on to examine whether these factors might contribute to the developmental trends in nonaggressive CD symptoms in early adolescence, using additive models. Testosterone was significantly related to nonaggressive CD symptoms in a univar-

Table 1. Models of Nonaggressive Conduct Disorder Symptoms

Standardized Predictor	Univariate Relationship with Nonaggressive Behaviors	Multiple Predictor Model
Age ^a	1.3 ^b (1.1, 1.6)	1.0 (.8, 1.4)
Testosterone	1.4 ^b (1.1, 1.6)	1.2 (.9, 1.5)
Peer Deviance	1.9 ^c (1.5, 2.2)	1.8 ^c (1.5, 2.2)

Results are presented as proportional odds ratios (95% confidence interval).

^aAge was standardized in these analyses.

^b $p < .01$

^c $p < .001$

iate model, as shown in Table 1. We also checked for a nonlinear relationship by adding a quadratic term. This effect was nonsignificant (POR = .9, 95% CI .8, 1.1; $p = .3$). Peer deviance was strongly related to nonaggressive behaviors ($p < .001$). In a multiple predictor model including testosterone and peer deviance, the effect of age was substantially reduced to a POR of 1.0 ($p = .8$), whereas the effect of peer deviance remained strong ($p < .001$), and testosterone fell short of significance ($p = .15$). After age was removed from the model, however, testosterone did have a significant effect (POR = 1.2, 95% CI 1.0, 1.4; $p = .02$).

Although aggressive CD symptoms did not vary with age, we examined whether testosterone predicted aggressive CD given the previous theoretic interest in the relationship. In a simple model there was no evidence to support such a relationship (POR = .9, 95% CI .7, 1.1; $p = .4$).

Social Dominance

Next, we explored predictors of social dominance as indexed by our measure of peer leadership. As shown in Table 2 neither chronologic age nor Tanner stage were significantly related to leadership. Leadership was positively related to testosterone ($p = .03$) in a univariate model. An additional quadratic term did not add significantly to the prediction ($p = .4$), which indicates that there was no significant nonlinearity in the relationship. Leadership was negatively related to peer deviance ($p = .02$) and nonaggressive CD symptoms ($p = .01$) in single predictor models. All these effects were significant when considered jointly.

Biosocial Interaction

To explore the possibility of biosocial interaction, we began by testing whether the relationship between testosterone and the antisocial behavior scales differed according to peer deviance. The relationship between testosterone and aggressive symptoms

Table 2. Models of Leadership

Standardized Predictor	Univariate Relationship with Leadership	Multiple Predictor Model ¹
Tanner Stage	.08 (-.01, .18)	
Age ^a	.05 (-.05, .15)	
Testosterone	.09 ^b (.01, .16)	.13 ^c (.05, .20)
Peer Deviance	-.09 ^b (-.18, -.01)	-.09 ^b (-.18, -.01)
Aggressive Conduct Disorder Symptoms	-.06 (-.12, .00)	
Nonaggressive Conduct Disorder Symptoms	-.10 ^b (-.18, -.02)	-.09 ^b (-.17, -.02)

Results are presented as β coefficients (95% confidence interval).

^aAge was standardized in this analysis.

^b $p < .05$

^c $p < .01$

Table 3. Prediction from Testosterone to Nonaggressive Conduct Disorder Symptoms and Leadership Disaggregated by Peer Deviance

Prediction from Testosterone in Peer Deviance Groups	Nonaggressive CD Symptoms ^a	Leadership ^b
Definitely Do Not Have Deviant Peers	1.0 (.6, 1.7)	.23 ^c (.08, .37)
Possibly Have Deviant Peers	1.2 (.9, 1.4)	.10 (.00, .20)
Definitely Have Deviant Peers	1.8 ^d (1.3, 2.5)	.02 (–.12, .16)

^aProportional odds ratios and 95% confidence intervals from ordinal logistic regression models.

^bBeta coefficients and 95% confidence intervals from ordinary regression models.

^c $p < .01$

^d $p < .001$

showed no variation by peer deviance. There was, however, an interaction with regard to nonaggressive symptoms. As shown in Table 3, testosterone was more strongly related to nonaggressive symptoms in boys who definitely had deviant peers compared with boys who definitely did not (interaction POR = 1.7, 95% CI 1.0, 3.1; $p = .062$) and those who possibly had deviant peers (interaction POR = 1.6, 95% CI 1.1, 2.3; $p = .016$). We examined a number of potential confounds for this interaction; there was no evidence of similar interactions between either age or Tanner stage and peer deviance in predicting nonaggressive CD symptoms.

Examining the relationships between testosterone and leadership in differing peer contexts, we found evidence of biosocial interaction in this domain also. Table 3 shows that testosterone was strongly associated with leadership in boys who definitely did not have deviant peers, less strongly associated in boys who possibly had deviant peers, and unassociated with leadership in boys who definitely had deviant peers. The interaction tests indicated that the relationship between testosterone and leadership was significantly stronger in boys who definitely did not have deviant peers ($\beta = .21$, 95% CI .004, .41; $p = .046$) but not significantly different from the relationship in boys who possibly had deviant peers ($\beta = .13$, 95% CI –.03, .29; $p = .111$). Once again there was no evidence that interactions between peer deviance and either age or Tanner stage were involved in the pattern of results reported.

Discussion

The GSMS offered a strong basis for studying the relationships among testosterone, aggressive and nonaggressive CD symptoms, and social dominance during puberty. The study provided a relatively large number of observations across an appropriate age range, with blood sample measurement of circulating testosterone, and clinical interview measures of CD symptoms and diagnosis; however, a number of limitations should be considered when evaluating the results. It is not inconceivable that the patterns of missing data for the Tanner stage and testosterone measures might have biased the findings. Missing data are inevitable in studies of this type, in which proper consent for sensitive or intrusive measures is sought. Given that the missing data were apparently most prominent in boys with less CD symptomatology, however, there seems to be no reason to suspect any very substantial bias. It should also be noted that the peer deviance and leadership measures are based on single questionnaire items. Replication of these findings with more extensive assessment of the peer context would be a useful goal for future research.

We set out to examine the extent to which the dramatic increase in circulating testosterone during male puberty was associated with the parallel increase in antisocial behavior over the same period, and also whether it was associated with social dominance. These questions were posed against the background of physical maturation and social development that characterize the pubertal period. The expected developmental rise in testosterone levels was indeed observed, as was an increase in nonaggressive (but not physically aggressive) antisocial behaviors. Association with deviant peers was a strong correlate of pubertal increases in antisocial behavior. Testosterone showed a relationship with nonaggressive CD symptoms and also with our measure of social dominance. Consistent with the existing literature (Book et al 2001), the strength of the bivariate relationship between testosterone and antisocial behavior was relatively modest. Because most adult males reach and surpass the levels of circulating testosterone present in the boys studied here without being extremely antisocial or socially dominant, we were particularly interested in examining whether the effects of testosterone interacted with other factors.

Existing evidence indicated that the correlates of testosterone might be influenced by social context (Booth and Osgood 1993; Booth et al 2003). Given the importance of the peer group for antisocial behaviors in adolescence (Thornberry and Krohn 1997), we focused on peer deviance as our measure of social context. As hypothesized, the relationship between testosterone and antisocial behavior did vary by peer context: testosterone was only related to nonaggressive CD symptoms in boys with deviant peers. By contrast, we found that testosterone was related to leadership rather than to antisocial behavior in boys who definitely did not have deviant peers, which suggests that high testosterone might be associated with socially valued characteristics in prosocial environments. This finding provides further support for the hypothesis that testosterone is associated with social dominance during adolescence (Schaal et al 1996; Tremblay et al 1998), at least among boys without deviant peers. It would be over-interpreting the data, however, to suggest that testosterone is unrelated to social dominance in boys with deviant peers. The phrasing of the leadership question (see Appendix 1) was designed to index a prosocial type of leadership rather than characteristics that might be relevant to dominance in a deviant peer group. This is illustrated by the negative relationship found between leadership and nonaggressive CD symptomatology. Indeed, we might speculate that a dominance-related model could also underlie the relationship between antisocial behaviors and testosterone that we identified in boys with deviant peers. Nonaggressive CD behaviors, such as vandalism, fire setting, and stealing might constitute social achievements in deviant peer contexts, and so contribute to dominance in those settings. If these speculations are correct, they imply that a similar testosterone-dominance relationship underlies the correlates of testosterone in both deviant and nondeviant peer groups but that the behaviors that index dominance differ according to social context.

In contrast to the results for nonaggressive antisocial behaviors and leadership, we found no support for the hypothesis that testosterone is related to physical aggression during adolescence. As noted in the introduction, previous studies have produced rather mixed results on this question and have faced problems of low sample size. In our relatively large sample, we found that rates of physically aggressive behaviors were fairly stable across age in later childhood and the early teens. In other studies, physical aggression has been argued to be most prevalent during

early childhood and to decline during middle childhood and adolescence (Tremblay 2000). Given that circulating testosterone levels are very low during childhood and increase dramatically during adolescence, it would be difficult to support the thesis that high levels of testosterone cause increased physical aggression during puberty. The possibility remains, however, that physical aggression might index dominance in some specific contexts, and a relationship between testosterone and physical aggression would be predicted in those situations (Tremblay et al 1998). Our data did not offer the opportunity to study such contexts, but it seems that testing for this type of biosocial interaction might be an important step in understanding the etiology of antisocial behavior.

This work was supported in part by Medical Research Council program Grant G9901475 (RR, BM). Participant interviewing was supported by National Institute of Mental Health Grant MH-48085 (EJC). Hormone analyses were funded by the W.T. Grant Foundation (94–1489–98; CMW).

We thank Louise Arseneault and Andrew Pickles for helpful discussions during the preparation of the manuscript.

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Appendix 1. Wording of Peer Group Questions

The wording from the parent Child and Adolescent Psychiatric Assessment (CAPA) is shown below. The wording in the child CAPA was altered to make the question refer to the respondent, and “kids” was used instead of “children.”

Peer Deviance

Some children play with or hang out with a “bad crowd.” In other words, their friends get into trouble a lot, are disruptive to others a lot, are disrespectful to adults a lot, or do other things,

like drink or steal. How much is your child the kind of child who hangs out with a bad crowd?

Leadership

Some children are more often chosen by other children to be the leader. They are good at organizing and running the group or team. Other children like to have these children in charge. How much is your child the kind of child that is chosen to be the leader a lot?