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Testosterone, Physical Aggression, Dominance, and Physical Development in Early Adolescence

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The associations among testosterone, physical development, social dominance, and antisocial behaviour during early adolescence were assessed in a sample of boys followed from 6 to 13 years. Saliva testosterone level was positively correlated with height, and uncorrelated with measures of fatness, including the body mass index. Physical aggression and social dominance were not significantly correlated. Regression analyses revealed that testosterone level and body mass additively predicted social dominance, whereas only body mass predicted physical aggression. Thus, early adolescents with high levels of testosterone were more likely to be socially dominant, especially if they had a large body mass. Those who had a large body mass were more likely to be physically aggressive, independently of their testosterone level. The observed pattern of correlations between testosterone, body mass, dominance, and physical aggression offers an interesting example of the complex hormone-physique-behaviour relations at puberty. They support the hypothesis that testosterone level and social dominance are related, and that the association between testosterone level and physical aggression is probably observed in contexts where physical aggression leads to social dominance.

Experimental manipulations on a wide range of nonhuman mammals provided an initial theoretical framework to investigate the influence of gonadal androgens in the peri-natal organisation and later activation of aggressive behaviour in humans (Archer, 1988; Bouissou, 1983; Rose, Bernstein, Gordon, & Catlin, 1974). The evidence available so far remains limited (Albert, Walsh, & Jonik, 1993; Archer, 1991; Archer, 1994; Eichelman, 1992; Rubinow & Schmidt, 1996), yet a growing literature suggests a reciprocal association between systemic androgens and aggressive dispositions assessed from a variety of criteria. In adult humans, for example, comparisons between groups of individuals selected for their high or low aggressiveness indicated higher levels of testosterone in the more aggressive groups (Dabbs, Frady, Carr, & Besch, 1987; Dabbs & Morris, 1990; Ehrenkrantz, Bliss, & Sheard, 1974; Kreuz & Rose, 1972; Mattsson, Schalling, Olweus, Low, & Svenson, 1980; Rada, Laws, Kellner, Stivastava, & Peake, 1983; Virkkunen et al., 1994; Windle & Windle, 1995). In nonselected cohorts, correlational relationships between testosterone and dimensions of aggressiveness are low but generally positive (Christiansen & Knussmann, 1987; Dabbs & Ruback, 1988; Gladue, 1991; Gray, Jackson, & McKinlay, 1991; Lindman, Jarvinen, & Vidjeskog, 1987; Scaramella & Brown, 1978). However, these concurrent correlational data do not provide much information on the direction of causal effects in androgen-behaviour interactions.

Some investigations have shown that testosterone level is affected by social experience which occurred shortly prior to the hormone sampling (e.g. Booth, Shelley, Mazur, Tharp, & Kittok, 1989; Elias, 1981; Gladue,

Boechler, & McCaul, 1989; Mazur, Booth, & Dabbs, 1992; McCaul, Gladue, & Joppa, 1992). To date we dispose indeed of much more demonstrations of hormone responses to behaviour than of the reverse causality. Because a direct manipulation of the hormone levels in healthy subjects presents ethical problems, nearly all researchers involved in the field of behavioural endocrinology have made a plea for a systematic attention to the correlates of natural variations of androgen levels around puberty (Archer, 1991; Brown, 1981; Buchanan, Eccles, & Becker, 1992; Hays, 1981; Mazur, 1983; Nottelman, Inoff-Germain, Susman, & Chrousos, 1990; Reiss & Roth, 1993; Susman, Nottelmann, Dorn, Gold, & Chrousos, 1989). A close scrutiny of that period of life with a longitudinal research design was called for to provide a better insight into the behavioural and attitudinal consequences of increased androgen release.

Cross-sectional studies have been oriented in that direction. Two studies of unselected pubertal males revealed a strong testosterone dependence of self-reported norm violation behaviour (Udry & Talbert, 1988) and of parent-child conflict at home (Steinberg, 1987). One study of 37 boys and three girls (30 African-American and 10 Caucasian) in a summer treatment programme for children with disruptive disorders reported a significant positive correlation between morning saliva testosterone in the first week of treatment, and staff ratings of verbal and physical ratings over the seven-week programme (Scerbo & Kolko, 1994). Mattsson et al. (1980) found no significant differences in testosterone levels when they compared a group of 40 institutionalised serious youth offender males (14 to 19 years old) to 58 high school males (15 to 17 years old). Looking at the testosterone-aggression link within the delinquent group Mattsson et al. (1980) reported no significant relationship with staff ratings and behaviour, and one out of four significant correlations with self-reports of aggressive propensity. They also reported significant positive correlations between testosterone and self-reports on items involving response to provocation in the high school sample (Olweus, Mattsson, Schalling, & Low, 1988). In a cross-sectional study of a sample of 56 boys aged 9–14 years, the NIMH-NICHD Collaborative Programme found no relationships between testosterone and: (i) self-rated dispositions related to negative affect (Susman et al., 1987); (ii) maternal ratings of delinquency and rebelliousness (Susman et al., 1987); (iii) direct observations of anger expression and attempts to dominate either parent in a four-hour laboratory session (Inoff-Germain et al., 1988). Constantino et al. (1993) compared levels of serum testosterone in 18 4- to 10-year-old boys hospitalised for aggression problems with levels of serum testosterone in a group of age and race matched controls. No significant differences were found.

Two studies have provided longitudinal data on the relationship of testosterone level and deviant behaviour during early adolescence.

Drigotas and Udry (1993) provided data from a three-year follow-up of a random sample of 12- to 13-year-old boys ($N = 126$) targeting the influence of testosterone on "problem behaviour". They observed that plasma testosterone level at age 12–13 was positively correlated with concurrent and subsequent self-reports of problem behaviours. However, subsequent assessment of plasma testosterone level was never correlated with concurrent or subsequent self-reported problem behaviours. They concluded that testosterone did not show an effect on "problem behaviour", and that age 12–13 testosterone level appeared to be a marker for a general trajectory of early development. In the course of a longitudinal study of low socioeconomic status Caucasian boys, Tremblay et al. (1997) obtained four-yearly assessments of saliva testosterone level between 13 and 16 years of age. A comparison of 64 boys rated physically aggressive by elementary school teachers to 65 boys rated not frequently aggressive showed that the differences in testosterone level between the groups changed over the years. At age 13 the physically aggressive boys had significantly lower levels of testosterone; however, that difference was reduced at age 14, had disappeared at age 15, and by age 16 the physically aggressive boys had substantially higher levels of saliva testosterone compared to the boys who had never been rated physically aggressive by their elementary school teachers. Thus, the evidence for a direct activational association between the pubertal surge in gonadal androgens and qualitative or quantitative modifications of aggressive responsiveness for pre- and early adolescents remains controversial.

A major problem in the study of the testosterone-aggression linkage has been the operational definition of "aggression". Boxers, tennis players, and chess players can all be ranked on an "aggressivity" continuum. In this context, the term "aggressive" usually means assertive, energetic, and vigorous; a behavioural style characterised by attacking the opponent rather than taking a defensive position. This assertive style is often considered to be a key to success (i.e. leading to social dominance). The testosterone-aggressive behaviour style link must be differentiated from the testosterone-physically violent behaviour link. Unfortunately, most aggression scales used to assess children's and adolescents' aggression are a mix of items referring to disruptive, assertive, and physically aggressive behaviours (Tremblay, 1991; Tremblay et al., 1991). They cannot clearly differentiate aggressive individuals who favour physical aggression as a means of solving problems, from aggressive individuals who use other means for achieving their goals. An aggressive-assertive behavioural style is generally considered an asset in modern societies. However, a physically aggressive style is generally condemned. An important difference between human and nonhuman species may be that physical aggression is less often used to achieve dominance. Even among kindergarten children, the

physically aggressive are generally rejected (Vitaro, Tremblay, & Gagnon, 1992).

Mazur and Booth (in press) argue that adults with high levels of testosterone have a propensity to achieve a dominant status. The positive association between testosterone and physical aggression would thus be observed only in groups where physical aggression is an effective means of achieving dominance. This could explain why studies of prisoners (e.g. Dabbs et al., 1987) and disruptive children in treatment (e.g. Scerbo & Kolko, 1994) have found significant associations between physical aggression and testosterone. In population samples, testosterone should be linked to assertive, but not physically aggressive behaviour.

The first aim of this study was to examine the association between testosterone level, physical development, and concurrent assessments of antisocial behaviour within a sample of early adolescents. As mentioned earlier, the strength of the testosterone-aggression link at adolescence remains controversial. The possibility of an indirect influence of testosterone, mediated through general physical growth, was approached by examining the association between physical development measures and synchronous ratings of aggressiveness. The relationship between these dimensions has not been well investigated in early pubertal boys (Paikoff & Brooks-Gunn, 1990), and available findings are contradictory. Although two studies reported an absence of linkage between pubertal status and aggression (Olweus et al., 1980; Simmons & Blyth, 1987), numerous other sources (e.g. Buchanan et al., 1992; Flannery, Rowe, & Gulley, 1993) indicated positive links between pubertal maturation and aggressivity-related traits (delinquency, behaviour problems, family conflict). Thus, the current state of the data would rather suggest a positive association between pubertal development and aggression measures.

The second aim was to assess the association of antecedent childhood antisocial behaviour with pubertal salivary testosterone, and physical growth at early adolescence. Although puberty has been shown to be a period of intensification of unconventionality, opposition, and delinquency (e.g. Elliott, 1994), these patterns of response do not emerge abruptly. Indeed, children described as aggressive at adolescence were often aggressive throughout childhood (Farrington, 1994; Haapasalo & Tremblay, 1994; Huesmann, Eron, Lefkowitz, & Walder, 1984; Moffitt, 1990; Pulkkinen, 1987; Tremblay et al., 1992), when the androgenic priming was very low. After having found that high testosterone at age 12–13 predicted problem behaviours 12 months later, Drigotas and Udry (1993) suggested that this link should be explained by earlier development. Belsky, Steinberg, & Draper (1991) have hypothesised that behaviour disorders during childhood would lead to early onset of puberty, whereas Susman et al. (Susman, Dorn, & Chrousos, 1991; Susman et al., 1987) hypothesised

that they would lead to late onset of puberty. Our aim was to test these alternative hypotheses with repeated assessments of physical aggression, opposition, and antisocial behaviour during childhood, and assessments of somatic maturation and androgen levels in early adolescence.

The final aim of this paper was to test the hypothesis that the testosterone-aggression link is in fact a testosterone-dominance link. Mazur and Booth (in press) conclude that this would be true in late adolescence and adulthood when testosterone in the bloodstream directly activates target receptors in the brain. They suggest that testosterone during adolescence affects physical maturation (increased size, muscle mass, and secondary sexual characteristics), rather than behaviour. However, dominance behaviours, and dominance hierarchies are present from early childhood, and throughout adolescence (Savin-Williams, 1979; Strayer & Trudel, 1984). If testosterone plays an important role in establishing dominance relationships, it would be surprising that this suddenly occurs in late adolescence. From an evolutionary and developmental perspective, if testosterone is a key to dominance behaviours it should play a role at least as early as the start of adolescence, when mating behaviours are already present (Flannery et al., 1993; Malo & Tremblay, 1997).

METHOD

Subjects

The subjects included in the study were part of a wider ongoing longitudinal programme on the development of male antisocial behaviour (Tremblay, Pihl, Vitaro, & Dobkin, 1994; Tremblay, Mâsse, Pagani, & Vitaro, 1996b). Caucasian boys (N = 1161; mean age: 6.12 years; SD: 0.33 years) from low socioeconomic areas of Montreal were rated by their teachers at the end of their kindergarten year. To control for cultural and socioeconomic background, only boys from French-speaking parents born in Canada, and with low educational status, were enrolled (N = 1037). A small random sample was selected for laboratory assessments, including testosterone levels at the start of puberty. Data on testosterone, physical aggression, physical growth, and social dominance at ages 12 and 13 were available for 57 boys.

Parents' age (at the birth of the target child), educational and occupational status, and family composition (at age 6) were obtained through questions to the mothers. A familial adversity score integrated all of these variables and characterised the psychosocial and economic disadvantage met by each subject in its family environment (Tremblay et al., 1991). Mothers' and fathers' age at birth of their son was respectively 24.5 (SD = 4.5) and 28.7 (SD = 7.2). Mothers had completed a mean of

10.5 years in school (SD = 2.1), whereas fathers had completed a mean of 9.7 years in school (SD = 2.3). Using the Blishen and McRobert (Blishen & McRoberts, 1976) Socioeconomic Index for Canadians, mothers' job SES had a mean of 37.2 (SD = 10.02), and fathers' a mean of 38.1 (SD = 11.4). The majority of these families had two children (52.6%), with 19.3% having one child, and 28.1% having 3 to 5 children. Most of the boys were living with both their biological parents when the study started in kindergarten (70.2%); 21.1% were living only with their mothers, and 8.7% were living in other types of family arrangements.

Behavioural Measures

Behaviour assessments were obtained from teachers (at ages 6, 10, 11, 12), peers (at ages 10, 11, 12), and the boys themselves (at ages 10, 11, 12, 13). Direct physical examinations were made at the lab, concomitantly with hormonal measures made via saliva samples (ages 11, 12, and 13). All measures of a given years' data collection wave were realised in a time window as narrow as possible (6 months, from March through August).

1. *Teachers' Ratings.* They were obtained using the Social Behaviour Questionnaire (SBQ; Tremblay et al., 1991). Items were scored on a three-level scale composed of "does not apply", "applies sometimes", and "frequently applies". Previous investigations using this scale have reported high internal consistency, and high test-retest reliability with both preschool and primary schoolchildren (Tremblay, Desmarais-Gervais, Gagnon, & Charlebois, 1987; Tremblay et al., 1991). For the present study, two scores representing two types of aggressive expression were derived by using items from the disruptive behaviour subscale: (i) the *opposition score* included the items "does not share materials", "irritable", "disobedient", "blames others", and "inconsiderate"; (ii) the *physical aggression score* included the items "fights", "kicks, bites, hits", and "bullies or intimidates other children". The range of possible values was 0–6 for the fighting scores, and 0–10 for the opposition score. These measures were repeated five times at the end of the school year by different school teachers when the boys were 6, 10, 11, and 12 years old. Inter-item reliabilities (alpha) for these scores were between .78 and .87 at the four measurement times (mean = .82). Means and standard deviations for each measured variable are presented in Table 1.

2. *Peer Ratings.* The Pupil Evaluation Inventory (Pekarik, Prinz, Liebert, Weintraub, & Neale, 1976) was used to obtain peer ratings at ages 10, 11, and 12. Each child in the class was asked to nominate four peers who best fit the description of each item of the questionnaire. We

TABLE 1
Means and Standard Deviations for Each of the Measured Variables ($n = 57$)

Age	*6*	*10*	*11*	*12*	*13*
Teacher					
Physical aggression	1.86 (1.77)	1.54 (1.88)[a]	1.30 (1.55)	0.75 (1.31)[b]	–
Opposition	2.82 (2.30)	3.02 (2.74)[a]	2.89 (2.45)	2.11 (2.38)[b]	–
Peers					
Physical aggression	–	0.18 (0.89)[c]	0.24 (1.04)[d]	0.10 (0.95)[a]	–
Social dominance	–	–	–	–	0.46 (0.45)
Self-reported delinquency					
Physical aggression	–	10.05 (2.62)	8.98 (2.49)	9.09 (2.89)	9.05 (2.57)
Total delinquency	–	34.72 (5.86)	31.72 (6.90)	31.88 (6.77)	32.35 (6.71)
Physical characteristics					
Testosterone	–	–	3.19 (2.07)[e]	12.76 (6.79)	42.04 (22.45)
Height	–	–	–	147.56 (7.30)	156.35 (8.65)
Weight	–	–	–	43.00 (8.33)	49.21 (9.73)
Wrist circumference	–	–	–	146.72 (10.32)	159.74 (10.20)
Head circumference	–	–	–	544.77 (17.76)	554.04 (20.10)
Tricep skinfold	–	–	–	116.11 (54.06)	129.06 (68.60)
Shoulder skinfold	–	–	–	85.88 (52.44)	94.91 (57.94)
Abdominal skinfold	–	–	–	121.11 (85.42)	146.08 (111.45)
Weight/height	–	–	–	119.68 (3.20)	220.05 (3.17)

Note: Dashes indicate data were not obtained.
[a] $n = 54$; [b] $n = 55$; [c] $n = 44$; [d] $n = 52$; [e] $n = 51$.

generated a physical aggression score from the PEI by using two items from the original 35 items (“those who start a fight over nothing”, “those who say they can beat everybody”). The Cronbach’s alpha for this scale was .87, .91, .90, at age 10, 11, and 12 respectively.

At age 13 the boys came to the laboratory by groups of five unfamiliar peers. Ratings of toughness and leadership were obtained from individual interviews during which every subject from a peer group was asked to nominate the leader (“who would you choose as leader?”) and identify the toughest boy (“who was the toughest?”). The interviews were made at approximately 10.30am after a 3-hour period during which they were picked up at home and driven together to the lab in a van, they were individually seen to assess personality and cognitive functioning, and finally took part in a competitive group task to provide an opportunity to measure social interactions. This task consisted of a 15-minute competitive game during which they threw sand bags in holes to win points and exchange them for money. No rules were set except that bags had to be thrown from a fixed distance. When the task was completed, the winner received two dollars, but all the other boys were told they had done well and were given a dollar each. Each subject received a toughness and a leadership score based on the ratio of the number of nominations he received (including self-nominations). A social dominance score was obtained by summing the leadership and toughness scores. The scores could vary from 0 to 2.

3. *Self-reported Delinquency.* A 27-item delinquency questionnaire was answered by the boys at 10, 11, 12, and 13 years of age (Tremblay et al., 1994). The items rated on a 4-point scale (never, once or twice, often, very often) included: “steal objects worth more than $10 in school”, “steal from store”, “steal objects worth more than $100”, “take money from home”, “keep object worth less than $10”, “keep objects worth between $10 and $100”, “steal a bicycle”, “sell stolen goods”, “enter without paying”, “breaking and entering”, “trespassing”, “take drugs”, “take alcohol”, “get drunk”, “destroy school material”, “destroy other material”, “vandalism at school”, “destroy objects at home”, “vandalism of cars”, “set a fire”, “strong-arm”, “gang-fights”, “use weapon in a fight”, “fist fight”, “beat up someone”, “carry a weapon”, “throw objects at persons”. The 27 items generate a total delinquency score (mean Cronbach’s alpha = .89). Seven items (last seven of the list) were used to obtain a self-reported physical aggression score (mean Cronbach’s alpha = .79). At age 10, the boys were asked to report lifetime frequency for each of the delinquent behaviours. At age 11, 12, and 13 they were asked to report the frequency over the past 12 months.

Hormonal Measures

At ages 11, 12, and 13, testosterone was titrated from three saliva samples taken at 8.30, 10.00, and 13.00, the day when the boys were brought to the laboratory for multiple psychobiological assessment. Saliva was spit into sterile tubes and immediately frozen at – 20°C until radioimmunoassay. When salivation was difficult, it was stimulated by giving to the subject a small piece of unsweetened chewing gum. Soft chewing movements were recommended in order not to contaminate the saliva sample because of gingival bleeding.

The assays were performed blindly at the Laboratoire d'Endocrinologie, INRS-Santé. The procedure was a variant from that established by Vittek, L'Hommedieu, Gordon, Rappaport, and Southern (1984), with testosterone-assay kits purchased from ICN Biomedicals Inc., Montreal. Once centrifugated, 500µl of saliva were pipetted, and extracted with 2ml of ether. One ml of the organic phase was taken and evaporated to dryness. The residue was incubated at 37°C for 120 minutes with 50µl of steroid diluent. After incubation, 100µl of sex-hormone-binding-globulin inhibitor, 400µl of 125 iodine-testosterone, and 400µl of antitestosterone were added and incubated overnight. A separation antibody was then added and allowed to incubate for 90min at 37°C. After 15min of centrifugation, the supernatant was discarded and the tube was counted in a gamma counter. Precision of the analytical procedure was improved by extraction of the standard curve. Intra-assay and inter-assay coefficients of variation were 6.3% and 12.3%, respectively. Regarding the specificity of the assay, no significant cross-reactions of the antibody were measured, except for 5-alpha-dihydrotestosterone (3.4%).

At 11 years of age the testosterone levels of most boys (88.2%) were under the detection limit of the assay method (5pg/ml). Thus, age 11 testosterone data were not used in the later statistical analyses. At age 12 and 13 the testosterone levels of almost all of the boys (96.5% and 98.2%, respectively) were above the detection limit. As expected, the testosterone levels tended to decrease from the first sample taken at 8.30am to the third taken at 13.00pm. The 8.30am value was used for the statistical analyses.

Physical Measures

At ages 12 and 13, height, weight, wrist, and head circumferences, as well as tricep, shoulder, and abdominal skinfold thickness were measured during the subjects' visit to the laboratory. Quetelet's body mass index (BMI) (Garrow & Webster, 1985) was calculated. This index (weight/height^2) has been shown to be a good estimate of body fat. Garrow and Webster (1985) found that the correlation between Quetelet's BMI and

the average of body density, body water, and body potassium was .96 for women and .94 for men.

RESULTS

1. *Association between Indices of Pubertal Development at Age 12.* Pearson correlations between the indices of pubertal development at age 12 are presented in Table 2. The boys' testosterone level was not significantly correlated with any of the morphological variables. However, there were clear trends for height, weight, and wrist circumference. Height was highly correlated with weight ($r = .57, P < .001$), wrist circumference ($r = .52, P < .001$), and head circumference ($r = .45, P < .001$). Weight was highly correlated with all the morphological measures. The BMI was also highly correlated with all the morphological measures, except height. The skinfold measures were highly correlated among each other, and, as expected, were most highly correlated with the BMI. Like the BMI, they were not correlated to height. Thus height and the BMI appear to capture two different components of the morphological structure. Both have been used as an index of pubertal development (Brooks-Gunn & Reiter, 1990; Tanner, 1982). They were used with testosterone levels for the following analyses.

2. *Associations between Indices of Pubertal Development and Concurrent Antisocial Behaviour at Age 12.* The aim of the following analyses was to examine the association between indices of hormonal and physical development, and concurrent assessments of antisocial behaviour. It can be seen from Table 3 that height and testosterone levels at 12 years of age were not significantly correlated to physical aggression or more generally defined antisocial behaviours assessed by teachers or by self-reports. However, the BMI was significantly correlated with self-reported physical aggression ($r = .52, P < .001$) and with self-reported total delinquency ($r = .54, P < .001$). Boys with a larger body mass at 12 years of age reported themselves to be more physically aggressive and to be more delinquent than boys with smaller body mass.

3. *Association between Indices of Pubertal Development at Age 12 and Prior History of Antisocial Behaviour.* The aim of these analyses was to verify alternative hypotheses concerning the effects of a history of deviant behaviour on the timing of puberty (Belsky et al., 1991; Susman et al., 1987, 1991). Table 4 reports the correlations of age 12 testosterone and physical growth with teacher, peer, and self-ratings of physical aggression, opposition and delinquency at ages 6, 10, and 11. The results replicate those from the previous analyses. Height and testosterone level at 12 years

TABLE 2
Correlations between Physical Characteristics at Age 12 (n = 57)

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*
1. Height								
2. Weight	.57**							
3. Wrist circumference	.52**	.87**						
4. Head circumference	.45**	.68**	.58**					
5. Shoulder skinfold	.02	.73**	.58**	.39*				
6. Tricep skinfold	– .06	.66**	.53**	.35*	.85**			
7. Abdominal skinfold	.04	.73**	.57**	.39*	.88**	.90**		
8. Body mass	.07	.86**	.73**	.56**	.87**	.85**	.87**	
9. Testosterone	.22	.22	.24	.12	– .03	– .03	.00	.11

**P < .001; *P < .01.

TABLE 3
Correlations between Physical Characteristics and Aggressive Behaviour at Age 12

	Height	*Body Mass*	*Testosterone*
Teacher ratings			
Physical aggression[a]	.08	.09	.21
Opposition[a]	.06	.01	.06
Peer ratings			
Physical aggression[b]	.07	.20	.15
Self-reports			
Physical aggression[c]	– .04	.52*	– .06
Total delinquency[c]	– .11	.54*	– .05

[a] $n = 55$; [b] $n = 54$; [c] $n = 57$.
*$P < .05$.

TABLE 4
Correlations between Physical Characteristics at Age 12 and Antecedent Aggressive Behaviour from Age 6 to 11 Years

	Height	*Body Mass*	*Testosterone*
Teacher-rated			
Physical aggression			
Age 6[a]	– .12	.12	.19
Age 10[b]	.01	.12	.23
Age 11[a]	.19	.09	.21
Opposition			
Age 6[a]	– .02	– .02	.06
Age 10[b]	.00	.18	.16
Age 11[a]	.21	.10	.11
Peer-rated			
Physical aggression			
Age 10[c]	.08	.13	.06
Age 11[d]	.18	.13	.04
Self-reported			
Physical aggression			
Age 10[a]	– .16	.30*	.02
Age 11[a]	.13	.43**	.18
Total delinquency			
Age 10[a]	– .15	.33	– .03
Age 11[a]	.01	.36	.15

[a] $n = 57$; [b] $n = 54$; [c] $n = 44$; [d] $n = 52$.
**$P < .01$; *$P < .05$.

were not correlated to any of the antisocial behaviour assessments between 6 and 11 years of age. The only significant correlations were found between the age 12 BMI and the age 10 and 11 self-reported aggression and self-reported delinquency. Boys who reported more physically aggressive and delinquent behaviours at ages 10 and 11 were more likely to have a large body mass at 12 years of age. The correlations are lower but in the same direction as those observed in concurrent data reported earlier (Table 3).

4. *Antecedents of Physical Aggression and Dominance at 13 Years of Age.* The aim of the following analyses was to test at early adolescence the testosterone-dominance hypothesis that Mazur and Booth (in press) proposed for adults. From this hypothesis, we expected that testosterone level would be a better predictor of social dominance than of physical aggression.

Table 5 describes the correlations between each of the variables. First, it can be observed that there was no significant correlation between the two dependent variables at 13 years of age: Social dominance with unfamiliar peers in a laboratory situation and self-reported physical aggression. Second, social dominance at age 13 was correlated with testosterone level ($r = .35, P < .01$) and body mass ($r = .32, P < .05$) assessed at age 12. Third, self-reported physical aggression at age 13 was correlated with age 12 and 13 body mass ($r = .50, P < .001$; $r = .47, P < .001$), and with a mean score of self-reported physical aggression between age 10 and 12 ($r = .83, P < .001$). Height at age 12 and 13 was not correlated with social dominance, nor with physical aggression, but was highly correlated with testosterone level at age 13 ($r = .48, P < .001$; $r = .62, P < .001$). Testosterone levels at age 12 and 13 also showed a substantial correlation ($r = .41, P < .01$).

Hierarchical multiple regressions were used to test the predictive value of the independent variables for both dependent variables. The mean physical aggression score between age 10 and 12 was not included in this prediction of physical aggression at age 13 because it would have prevented any other variable from making a significant contribution.

Results for physical aggression are presented in Table 6. It can be seen that once the age 12 BMI is included in the regression, no other variable measured at age 12 or 13 makes a significant contribution to the prediction. The BMI at age 12 explains 24% of the variance in self-reported physical aggression at age 13.

Results of the hierarchical multiple regression for social dominance presented in Table 7 show that age 12 testosterone and body mass both make independent contributions to explain social dominance at age 13. They explain 18% of the variance in social dominance. Once these two

TABLE 5
Correlations between Social Dominance, Physical Aggression, and Physical Characteristics at 12 and 13 Years of Age ($n = 57$)

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*
Outcomes at age 13									
1. Social dominance									
2. Physical aggression	.18								
Predictors at age 12									
3. Testosterone	.35**	.09							
4. Height	.18	.05	.22						
5. Body mass	.32*	.50***	.11	.07					
6. Phys. agg. (10–12 yrs)	.21	.83***	.05	– .03	.50***				
Correlates at age 13									
7. Testosterone	.23	.00	.41**	.48*	.01	– .07			
8. Height	.24	.04	.30*	.94*	.07	.00	.62***		
9. Body mass	.24	.47***	.13	.10	.94***	.44**	.03	.07	–

*$P < .05$; **$P < .01$; ***$P < .001$.

TABLE 6
Results of Hierarchical Multiple Regressions for the Prediction of Physical Aggression at 13 Years of Age

	Beta[a]	*F*(*df*)	*P-value*	*Adjusted* R^2
Body mass (Age 12)	.502	18.515 (1,55)	< .001	.238
		0.048 (2,53)	.953	.211
Testosterone (Age 12)		0.012 (1,53)	.915	
Height (Age 12)		0.068 (1,53)	.796	
Age 13 predictors		0.010 (4,51)	> .999	.179
Body mass		0.001 (1,51)	.977	
Testosterone		0.015 (1,51)	.902	
Height		0.006 (1,51)	.938	
Social dominance		0.024 (1,51)	.876	

[a] Betas are reported only for significant variables (at 5% level) and are estimated in the absence of the nonsignificant variables.

TABLE 7
Results of Hierarchical Multiple Regressions for the Prediction of Social Dominance at 13 Years of Age

	Beta[a]	*F*(*df*)	*P-value*	*Adjusted* R^2
		7.080 (2,54)	.002	.178
Testosterone (Age 12)	.322	6.966 (1,54)	.011	
Body mass (Age 12)	.291	5.688 (1,54)	.021	
		0.384 (2,52)	.683	.159
Height (Age 12)		0.548 (1,52)	.462	
Physical aggression (Age 10–12)		0.273 (1,52)	.603	
Age 13 predictors		1.059 (4,50)	.387	.182
Body mass		2.974 (1,50)	.091	
Testosterone		0.094 (1,50)	.760	
Height		0.520 (1,50)	.474	
Physical aggression		0.004 (1,50)	.951	

[a] Betas are reported only for significant variables (at 5% level) and are estimated in the absence of the nonsignificant variables.

variables are included in the equation no other variable at age 12 or 13 adds to the prediction.

DISCUSSION

The first aim of this paper was to examine the association between testosterone level, physical development, and concurrent assessments of antisocial behaviour within a sample of 12-year-old males from low socioeconomic status families. Correlations between testosterone and physical development showed that saliva testosterone level tended to be positively associated with height, weight, and wrist circumference, but not with head circumference, three measures of skinfold, and BMI. At age 13 testosterone level and height were highly correlated. The correlation trends between saliva testosterone levels, height, weight, and body mass replicate those obtained with a cross-sectional sample of 7- to 14-year-old disruptive children (Scerbo & Kolko, 1994). Similar patterns of interrelations between plasmatic testosterone level and physical features at adolescence were obtained by Nottelman et al. (1987) in a cross-sectional sample of boys aged 10 to 14 years (for height and weight), and by Olweus et al. (1980) in a cross-sectional sample of adolescents aged 15 to 17 years (but only for height, not for weight or wrist circumference). Dabbs, Jurkovic, and Frady (1991) also noted a significant positive correlation between salivary testosterone level and weight in 17- to 18-year-old male offenders, although one study failed to find any significant relationship between testosterone and physical variables (Bishop et al., 1988).

Concerning the concurrent association of antisocial behaviour with physical and hormonal development, significant correlations were found with the BMI. Boys with the largest body mass tended to report the highest number of physical aggressions, and delinquent behaviours. This result could be taken as evidence that physical maturation increases the likelihood of antisocial behaviour. However, if this was the case, significant correlations should also have been observed for height, and testosterone level. There was no evidence that any of the five types of antisocial behaviour assessments were associated with testosterone level or height.

The results concerning the association between testosterone level and antisocial behaviour are in line with those from two studies with pre- and early adolescents (Constantino et al., 1993; Susman et al., 1987), and at odds with two others (Drigotas & Udry, 1993; Scerbo & Kolko, 1994). These five studies are not easy to compare. The Constantino et al (1993) and Scerbo and Kolko (1994) studies were cross-sectional and used small clinical samples (one of 18 boys, one of 37 boys and 3 girls), from different

ethnic backgrounds (one unspecified, one African-American, and Caucasian), that varied in age (one ages 4–10, one 7–14); Susman et al.'s (1987) was a cross-sectional sample of 56 healthy 9- to 14-year-old boys (mostly Caucasian); Drigotas and Udry's (1993) was a longitudinal study of 126 boys (race unspecified) from a school population (ages 12–16); and the present study is a longitudinal one with a sample of 57 Caucasian boys, from a 12- to 13-year-old school population. Some of the studies measured testosterone in the plasma, others in saliva, and each relied on different measures of deviant behaviour. A positive testosterone-aggression link may have been observed in the Scerbo and Kolko (1994) study because it was the only study which used adult ratings of overt verbal and physical aggression in the context where the saliva testosterone was collected. However, in this latter study the positive association may also be due to other factors. A likely factor is social dominance. Because the disruptive children were interacting with each other in a summer programme, there is a strong possibility that the most aggressive were the most dominant. If this was the case, then social dominance and aggression would be confounded. Mazur and Booth (in press) argued that individuals with higher levels of testosterone have a greater tendency to dominate. If this rule is applied to a group of children with disruptive disorders who are forced to interact daily, or for that matter to a group of adult criminals who live together in a prison (Dabbs, Carr, Frady & Riad, 1995), one would expect that those with the greatest need to dominate would use physical aggression more frequently and with more intensity; they would also be those who have a history of being highly physically aggressive. If the rule is applied to a group of prosocial children forced to live together, then verbal assertiveness might correlate more strongly with testosterone than physical aggression. In the only other study where testosterone was correlated with "problem behaviour", Drigotas and Udry (1993) argued that it probably did not indicate a hormone effect. They suggested that testosterone level at the start of puberty was rather an index of the earlier developmental trajectory. Schaal, Tremblay, Soussignan, and Susman (1996) offered a similar argument to explain why 13-year-old boys with a history of physical aggression during elementary school had a lower level of testosterone compared to boys with no history of physical aggression in elementary school. From this perspective, testosterone levels are outcomes of social adjustment rather than causes of social adjustment.

The second aim of this study followed this logic by assessing the association of antecedent childhood antisocial behaviour with pubertal salivary testosterone, and physical growth at 12 years of age. Balsky et al. (1991) had suggested that antecedent social adjustment could accelerate the timing of puberty, and thus have an impact on the patent (somatic development) and latent (testosterone level) measures of maturation,

whereas Susman et al. (1989, 1991) suggested the reverse effect. Our results indicated that testosterone level and height at age 12 were not significantly related to any of our five assessments of antisocial behaviour by teachers, peers, or self, from age 6 to 11 years of age. However, as observed with the concurrent data, body mass at age 12 was significantly correlated with age 10 and 11 self-reported physical aggression and delinquency. Thus, there was some evidence linking externalising behaviour problems during preadolescence to early somatic maturation at age 12. However, that evidence was not strong. If the correlation between externalising behaviour problems and body mass was a reflection of the impact of externalising behaviour problems on puberty onset, a similar correlation should have been observed between externalising behaviour problems, testosterone levels, and height.

One explanation for the association between antisocial behaviour and body mass is that boys with a larger body mass can more easily impose themselves on others (i.e. dominate others). If this was the case, and if Mazur and Booth's (in press) testosterone-dominance link hypothesis holds for early adolescents, we should have observed a correlation between body mass and testosterone. We did not.

The third aim of the paper was to investigate specifically the links between dominance, testosterone, physical aggression, and physical development. We observed that social dominance, defined by peers' and self-ratings' of leadership and toughness after a brief period of interaction with unfamiliar peers at 13 years of age, was significantly correlated to the previous years' testosterone level and body mass assessed in a similar context. However, social dominance at age 13 was not significantly correlated to concurrent physical aggression nor to physical aggression over the previous three years. On the other hand, body mass was the best predictor of physical aggression after antecedent physical aggression.

To our knowledge this is the first demonstration that male testosterone level at the start of puberty predicts social dominance a year later. This result tends to confirm Mazur and Booth's (in press) hypothesis of the testosterone-dominance link. However, they had hypothesised that this link would not exist before late adolescence, and that it would explain the testosterone-aggression link. It is difficult to understand why the testosterone-dominance link would not be present in early adolescence. Mazur and Booth (in press) appear to have reached that conclusion because they did not find any clear evidence, as they did for adults, that testosterone in early adolescence is linked to problem behaviour. But this logic contradicts their argument that the testosterone-aggression link in fact derives from a testosterone-dominance link. They appear to equate dominance with problem behaviour during early adolescence; possibly

because the testosterone-behaviour studies during early adolescence measured problem behaviours rather than social dominance.

We suggest that the testosterone-dominance link, if it exists, should be present from infancy onwards. Competition for objects and physical aggression appear to be more frequent during the first 36 months after birth than at any other time in life (Hay & Ross, 1982; Restoin et al., 1985; Tremblay et al., 1996a). Dominance hierarchies are clearly present during the preschool years (Strayer & Trudel, 1984). If peri-natal testosterone exposure has an impact on sexual and social behaviour through its impact on the organisation of the brain (Breedlove, 1992; Collaer & Hines, 1995), why would it not have an impact on early expression of dominant behaviour? And why would early dominance experience not have an impact on dominance during the early adolescent years and adulthood? Jacklin, Maccoby, and Doering (1983) did show that cord blood testosterone level at birth was inversely correlated with timidity in boys during the first two years after birth. Children probably learn early on from their environment who and how to successfully dominate. The better socialised will learn to use language and prosocial behaviour instead of physical aggression, the less well socialised will continue to use physical aggression. Depending on the social context they encounter, their behavioural style will lead to successful or unsuccessful dominant status. Physical aggression can lead to dominance in a camp for disruptive children, in a delinquent gang, or in a prison. In these samples testosterone, aggression, and dominance should be correlated. However, in the average classroom, or the average workplace, physical aggression does not lead to social dominance. In these samples we should observe a correlation between testosterone level and social dominance, but not with physical aggression.

The pattern of correlations we observed between body mass, testosterone, dominance, and physical aggression offer an interesting example of the complex dominance-hormone-physique-behaviour relations at puberty. Physical aggression and social dominance were not correlated, indicating that the boys were living in a social context that did not reward physical aggression. Body mass and testosterone level were not correlated, confirming that the body mass index is not an index of pubertal maturation in early adolescent males (see Scerbo & Kolko, 1994). However, body mass and testosterone additively predicted social dominance, whereas only body mass predicted physical aggression. Thus, early adolescents with a large body mass were more likely to be physically aggressive or socially dominant; those who were socially dominant tended not to be physically aggressive and tended to have higher testosterone levels than the physically aggressives.

It is important to consider that the high levels of testosterone at age 12, which anticipate social dominance at age 13, could be the product of social

dominance as much as the cause of social dominance. Unfortunately, we did not have a measure of social dominance before age 13 that could have been used to test the hypothesis of reciprocal causation. Large-scale longitudinal studies from pregnancy to adulthood are clearly needed to clarify the multiple reciprocal causation loops between hormones, physical development, and social development. The availability of easy to use assessments of hormones like testosterone should convince developmentalists to include these measures in large longitudinal cohorts.

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