Raging Hormones in Puberty: Do They Influence Adolescent Risky Behavior?

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Abstract

The rapid and dramatic changes in hormone levels during adolescence have been linked to the onset of a number of problematic behaviors. Using a unique data set that contains information on testosterone and cortisol levels, we examine whether these variables directly affect a variety of outcomes and indirectly affect the magnitude and interpretation of several key family background variables. Further, using information on access to two randomly assigned interventions designed to promote child development we identify the causal effect of alternative child rearing practices.

We find strong evidence that child rearing practices are endogenous and that active supervision is substantially more effective than simply imposing rules. Testosterone levels and their growth rates are significantly associated to a variety of risky and criminal activities. Cortisol levels are related to gang activity, property crime and illicit drug use. While the inclusion of hormones is found to have minor impacts on parental education and income, they substantially affect the magnitude and significance of adolescent height. Finally, although we find adolescent hormone levels are not correlated with birth outcomes or early behavior, we suggest they may proxy for the dynamic relationship between genes and an individual's environment.

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1 Introduction

The origins of violence and aggression can be found in nature. Certain areas of the brain are well known to be associated with aggressive behavior among other outcomes in humans and other animals. In response to signals from the brain, the endocrine system releases hormones. Hormones are organic chemical messengers that coordinate the physiology and behavior of an animal by regulating, integrating and controlling its bodily function. Recent advances in behavioral endocrinology have made possible the noninvasive assessment of several key hormones. In this paper we discuss the associations of two hormones, cortisol and testosterone, with a variety of adolescent problematic behaviors. While to the best of our knowledge studies in economics have yet to use such information, a consensus among researchers in sociology and endocrinology exists that hormones such as testosterone affords a unique view into the interacting effects of biological, contextual and behavioral forces of development.

Understanding the causal mechanisms through which family background affects child development and subsequent labor market outcomes has long been of interest to social scientists.[1] Recently, increased attention has been paid to the active role of child rearing practices and their relationship to family background and juvenile behavior.[2] While the role of parenting style as a determinant of adolescent outcomes has a long history in developmental psychology and sociology, determining causality remains a challenge. To deal with the potential endogeneity of child rearing practices, we

[1] Economists tend to view the relationship between parental characterisctics and child development through the Becker and Tomes (1986) model of family production. The challenge facing researchers is to ensure that variation in parental characteristics is credibly exogenous.

[2] Weinberg (2001) finds that child rearing practices vary positively with family income. Aizer (2003) finds that adult supervision reduces the probability that an adolescent skips school, uses alcohol or marijuana, steals or gets involved in fights.

exploit exogenous variation in programs that provided guidance as well as instruction to parents, and were assigned randomly across schools. We also investigate whether the inclusion of hormone measures affects the magnitude and interpretation of several key family background variables.[3] This is of importance since understanding the root causes of many social problems typically involves disentangling the effects of family background variables from the effects of genetic endowments. Further, the success of many public policies require that the environment and not biology plays a key role in determining socioeconomic outcomes.

We focus on the role of hormones in adolescence since the second stage of puberty (gonadarche) is a period of physical development accompanied by dramatic increases in the circulating levels of many hormones. Testosterone levels in males on average experience an 18 fold increase during puberty (Nottelman et al. (1987)). This natural rise in hormone levels provides for an experiment in nature. Our outcome measures include a variety of risky adolescent behaviors including property and violent crime activity.[4]

This paper has a natural relationship to the contentious decades–old "nature versus nurture" debate. Studies in economics typically remove genetic effects using a differencing strategy between siblings or twins to focus exclusively on time varying family environment effects. While twin studies have inherent scientific value at removing time invariant inheritable traits, studies report that the correlations in hormone levels between monozygotic twins are less than 60%.[5] Further, by

[3]Note the available scientific evidence suggests that hormone levels are not correlated between parents and their sons. Further, hormone levels should be uncorrelated with omitted factors such as parental preferences and tastes that are thought to bias estimates of the causal effect of parental characteristics.

[4]Testosterone patterns in males follow an identical pattern to the crime-age profile, increasingly rapidly during adolescence and peaking in the late teens and then a steady decline throughout adult life in men. Numerous studies have postulated that biological factors such as testosterone are a significant determinant of juvenile crime. For example, Orlebeke (2001) hypothesizes that the recent decreasing trend in U.S. juvenile delinquency is partially attributable to shifts in perinatal biological circumstances as children in utero having been exhibited to lower levels of testosterone.

[5]While it is plausible that experiential effects are responsible for individual differences in hormone levels between

differencing, any reciprocal causality between biology and social behavior is assumed away. Yet scientific evidence indicates that interactions among hormones, brains, and behaviors are incredibly complex.

This paper is organized as follows. In section 2, we provide a brief survey of the interdisciplinary research on the effects of testosterone and cortisol on adolescent outcomes. The data set used in our analysis is described in section 3. The economic model that underlies our empirical strategy and econometric methodology are described in section 4. In contrast to empirical work using information on hormones in other disciplines we are careful to include predetermined hormone readings (i.e. that occur sufficiently prior to the outcome under consideration) rather than treating the hormone measures as being strictly exogenous. Our results are presented in section 5. We find that testosterone levels are strongly related to a variety of adolescent risky behavior and delinquency outcomes. Higher cortisol levels are associated only with gang activity, illicit drug use and criminal activities. We find strong evidence that child rearing practices are endogenous to adolescent risky behavior and that active supervision is substantially more effective than imposing rules at reducing the likelihood a child engages in riskier activities. While hormone levels have weak relationships with parental education and income measures, there are strong positive associations between adolescent height and testosterone. Including testosterone in the estimation equation renders the coefficients

identical twins, testosterone is thought to pass between twins in utero leading to differences across twins with hormone measures taken immediately after birth. Harris et al., (1998) report correlations of approximately 60% in testosterone levels between adolscent male monozygotic twins. Further, they find no resemblance between testosterone value of either parent and their sons. Thus, child testosterone is not proxying for omitted parental hormone levels. Similarly, Young et al. (2000) find that around 40-45% of the total variance in salivary cortisol is shared by monozygotic twins. Finally, in other species, testosterone secretion in utero has been shown to influence the development of male primary sexual characteristics and has organizational effects on the brain and behavior (See Goy et al., 1988 or Wilson et al., 1981). Using magnetic resonance microscopy technology, Koshibo et al. (2004) demonstrated with rodents that sex hormones alter the development of certain brain structures (hippocampus, amygdala and ventricles) during puberty and that these effects persist into adulthood.

on adolescent height to become statistically insignificant. This finding is of interest since height is widely believed to be an important ingredient of professional and personal success and academics have investigated and found evidence that labor markets reward height separate from other factors. Most recently, Persico, Postlewaite and Silverman (2004) find that the height premium in wages can be ascribed to adolescent (and not adult) height. Our results suggest that while adolescent height may proxy for hormone when omitted, these hormone levels (in contrast to adolescent height) are unrelated to both early health endowments and early behaviors. We interpret these results as suggesting that hormones may proxy for the dynamic relationship between genes and an individual's environment. Our findings are summarized and directions for future research are discussed in a concluding section.

2 Primer on Biological Connections

Despite media sensationalism, genes are not specifically coded for sexuality or criminal behavior.[6] Genes are known to directly affect memory, impulse control and sensation-seeking in the case of antisocial behavior. Genetic influences operate in part through hormones which are chemical messengers in the body. Hormones translate differential experiences into variability in gene expression, which in turn affects protein synthesis and changes in physiology and behavior.[7] In this section, we provide a brief overview of the interdisciplinary research on the relationship of two hormones with adolescent behavior.

[6] Much attention has recently been paid to the role of hormones in professional sports. For example, baseball star Ken Caminiti told Sports Illustrated that not only are at least half of major league ball players using hormones, but he also was using them when he won the National League's Most Valuable Player Award in 1996. Mark McGwire broke the single-season home run record while taking the steroid androstenedione. Finally, at the Seoul Olympics in 1988, Ben Johnson was stripped of his gold medal and world record for running the 100 metres in 9.79 seconds after testing positive for steroid use, which boosted his testosterone level.

[7] See Granger et al. (1999) for a discussion.

2.1 Testosterone

Testosterone is the most potent hormonal determinant of physical and behavioral masculinization. Produced by the testes in regulation of the hypothalamic pituitary gonad axis, the hormone propels prepubescent boys toward deeper voices, hairy chests and other primary and secondary sex characteristics. The most compelling evidence of the effect of hormones on behavior is provided by controlled laboratory studies in which hormone levels are altered by the experimenter. Using this methodology, researchers in the health sciences have established a causal relationship between testosterone and aggressive behavior in rodents.[8] In response, Mazur and Booth (1998) argue that higher testosterone levels cause dominance and studies with rodents are unable to distinguish between aggression and dominance since rodents dominate aggressively. Supporting this hypothesis, Tremblay et al., (1998) found that increasing body mass coupled with testosterone boosts dominance but only body mass predicts physical aggression in adolescent males.

Evidence on a causal relationship between testosterone and behavior within humans is not as clear.[9] In one of the few clinical studies, Finkelstein et al. (1997) reports that aggressive behavior increases for hypogonadal (low testosterone) adolescent males administered with doses of testosterone versus placebo. Hypogonodal boys administered with testosterone also resulted in significant increases in self reports of nocturnal emissions, touching girls and being touched by girls. Similarly, Van Goozen et al. (1994) manipulated gonadal hormones in transsexuals and found that

[8] See Beatty (1992) for a review of this literature. The exact channel through which testosterone influences aggressive behavior remains an active area of research in genetics. The results strongly suggest that testosterone affects aggressive behavior in male mice either through direct action on androgen receptors in the brain or through the conversion of testosterone to estradiol. These studies find that aggressive behavior in rodents increases in puberty.

[9] Direct manipulation of hormone levels in healthy human subjects presents ethical concerns. An additional difficulty in interpreting and comparing results from non-experimental data is that researchers implicitly treat hormone readings as strictly exogenous and do not account for the time of the reading. We use predetermined measures and account for time of reading as discussed in section 4.

increases in androgen levels increased aggressive behavior. Finally, with small samples O'Connor et al. (2002) using limited exogenous increments in testosterone found no association with increased aggression or mood, whereas larger exogenous increases in testosterone used in O'Connor et al. (2004) resulted in significant increases in anger-hostility and mood changes.

The majority of evidence on the effects of testosterone in human subjects is non-experimental and employs observational data. Testosterone is known to work differently perinatally, at puberty and in adulthood. Studies that use data on adolescents have generally found positive correlations between testosterone levels and aggressive or anti social behavior.[10] Research on adolescents has also found that higher testosterone levels in adolescents is associated with increased sexual activity (Halpern, et al., 1998), age at first sexual intercourse (Dunne et al, 1997), increased criminal activity (Booth and Osgood, 1993), increased tobacco and alcohol use (Zitzmann and Nieschlag, 2001). In terms of criminal behavior Dabbs et al. (1995) conducted a fascinating study of 692 male prison inmates, finding testosterone related to type of crime and to behavior in prison. Testosterone was highest among inmates convicted of child molestation, rape, homicide, and assault, and it was lowest among inmates convicted of burglary, theft, and drug offenses. Finally, high testosterone levels have been related to low occupational status and periods of unemployment for young adults (Dabbs, 1992). Psychological studies have found that both socioeconomic status and the quality of the parent child relationship can moderate the effects of testosterone on behavior.[11] Yet, in the absence of controlled experiments many of these findings remain an active subject of debate.

There is a great deal of evidence on humans that suggests that there is reciprocal causation (i.e. testosterone levels respond in the short run to behavior). For example, testosterone rises

[10] See Mazur and Booth (1998) for a critical survey of this literature.

[11] Weinberg (2001) finds that child rearing practices vary positively with family income. Evidence form the psychological lierature indiates that parents with poorer quality relationships with their children are associated with higher testosterone levels. Since hormone levels are not correlated across generations it is doubtful that child testosterone is strongly correlated with parent child relationships.

prior to matches for athletes and immediately following the match testosterone levels of winners are high relative to those of losers. The function of these short run fluctuations in testosterone is not known but it has been hypothesized that higher testosterone levels exist as winners prepare to take on more challengers while losers withdraw from competition.[12] Dabbs (1998) notes that the key to understanding the link between behavior and testosterone is to use measures of *baseline testosterone* since individual differences in levels are large and stable over time; and not short run changes in testosterone. Mazur and Booth (1998) conclude that the effects of testosterone on behavior in adolescence work primarily through the long term reorganization of the body and neurohormonal system.

2.2 Cortisol

Cortisol is a steroid hormone that is released from the zona fasciculata of the adrenal cortex in response to stress. The stressors that stimulate the release of this glucocorticoid hormone are many in number and include drastic changes in temperature, surgery, heavy exercising or even falling in love. Cortisol is considered to be such a reliable indicator of stress upon a system that many physiologists define stress as an event that elicits increased levels of cortisol.

It is well known that stress affects a wide range of bodily systems and behaviors.[13] Yet, research on the relationships between cortisol and behavior in adolescence reaches conflicting and contradictory evidence. The available evidence suggests that higher cortisol levels are associated with smoking (Canals et al., 1997) and marijuana use (Moss et al., 1999) but not with drinking

[12] Dabbs (1992) concludes that testosterone should be considered as a trait and a state. Testosterone can be considered a trait variable since individual differences in levels are large and stable over time. The rapid short run changes in hormone levels in response to environmental factors in testosterone allow testosterone to also be considered a state..

[13] In animals it has been shown to affect brain development, immune compentence and behavior. See Gunner et al. (2001) for a recent survey.

in adolescent males (Canals et al., 1997). In contrast, research with laboratory animals (Le et al., 1998) finds that rodents respond to stressful situations by seeking alcohol. Animals with higher blood cortisol levels consume greater amounts of alcohol.[14]

Antisocial behavior in boys has been associated with low resting cortisol levels (McBurnett et al., 2000), especially in boys exhibiting physical aggression. Finally, studies have found significant interactions between salivary cortisol and testosterone. Dabbs et al. (1991) examined 113 teen-age male prison inmates and found that cortisol moderates the correlation between testosterone and violence of crime.

A difficulty with much of this work is that it employs small clinical samples, is cross sectional in nature and compares current readings with current behavior. This is problematic since levels of cortisol are feted by a variety of environmental processes (Stansbury and Gunnar, 1994). Yet, the available evidence suggests that stress and alcohol or tobacco seeking behavior initiate similar hormonal responses. Second, most studies do not adjust for time of cortisol reading relative to normal wake up time. Chronobiologists find that hormone levels fluctuate during the day and the normal cortisol and testosterone concentration in saliva of humans peak at normal wake-up and fall throughout the day (see Dabbs (1990) or Wust et al., 2000).

3 Data

The subjects in this study were part of a longitudinal study that started in the spring of 1984. Kindergarten teachers in the 53 schools of the lowest socioeconomic areas in Montreal were asked to rate the behavior of each boy in their classroom. Eighty-seven percent of the kindergarten teachers agreed to participate, and 1161 boys were rated. The sample was reduced to 1037 boys by including only those boys born from Caucasian, French-speaking parents themselves born in

[14] Gianoulakis (1998) conjectures that studies finding no correlations between cortisol and alcohol are due in part to the small alcohol concentrations administered.

Canada to preclude cultural and socioeconomic biases.[15] Informed consent was regularly obtained from mothers and the youth throughout the study.

Following the initial teacher assessments in kindergarten, the mothers provided demographic information through a telephone survey. The mother provided information on family structure, years of education, date of birth, employment status including occupation for the most recent job for each parent. Parents' mean age at the birth of their son was 25.4 (SD = 4.8) for mothers, and 28.4 (SD = 5.6) for fathers. The mean number of school years completed by the mothers was 10.5 (SD = 2.8), and 10.7 (SD = 3.2) for fathers. The majority of the parents were unskilled workers. The mean and median family income when the boys were age 10 years (1988) was between $25,000 and $30,000 (Canadian dollars) which is substantially lower than the 1987 median Canadian income of $44,000 for couples with children. Approximately 67% of the boys lived with both biological parents, 24% lived with the mother alone and the remaining 9% lived in other family arrangements.

Parents, continually interviewed on approximately a biannual basis until the subject was 16 years old, providing information on changes in family structure and the family environment. Participation rates in follow-up interviews were high ranging between 70 - 85%.

Sub samples of the subjects were randomly brought to the University of Montreal laboratory during the summer holidays to obtain a variety of cognitive, behavioral, neurophysical and psychological measures. During the laboratory visit, testosterone, cortisol and physical development (height, weight, wrist robustness, and skin-fold measurements at triceps, shoulder, and abdomen) data were collected. Pubertal status was established using the Petersen et al. (1988) pubertal development scale. The status is based on self-reports of growth spurt, skin changes, voice changes, and body and facial hair development. The responses were coded and combined to classify the boys into specific stages: (1) pre-puberty, (2) beginning of puberty, (3) mid-puberty, (4) end of puberty and (5) post-puberty.[16]

[15] In addition, this elimination includes a handful of families that refused to participate or could not be located.

[16] We control for stage of pubertal development in our empirical analyses since it is a major source of variation in

Testosterone data were first collected when the subjects were 11 years of age and continued to the time they were 16 years of age. Testosterone levels were assayed from multiple saliva samples collected during a 1-day laboratory visit (at approximately 8:30 A.M., 10:00 A.M., 11:30 A.M., and 3:30 P.M.) . The first two saliva samples were collected before any competitive interactions, which could cause short run fluctuations in testosterone levels. Subjects were requested to donate saliva into sterile vials which were immediately frozen (-20 degrees Celsius) until radioimmunoassay.[17] The titration of testosterone from saliva was preferred to any other way of obtaining similar data for practical and theoretical reasons. Theoretically, salivary testosterone levels being highly correlated with the unbound fraction of circulating testosterone, is assumed to be a precise indicant of the behaviorally active fraction of testosterone (Riad-Fahmy et al., 1982; Wang et al., 1981). Practically, the handling of saliva is uncomplicated in comparison with the handling of blood or urine. Further, since the assessment is unobtrusive, it does not interfere with stress-elicited alterations of testosterone.[18]

hormone levels across adolescent individuals (Susman et al., 1987).

[17]The assays were performed blindly. The procedure was a variant of that established by Vittek et al. (1984) with testosterone-assay kits purchased from ICN Biomedicals Inc. Once centrifuged, 500 micro Liter of saliva was pipetted and extracted with 2 mL of ether. One milliliter of the organic phase was taken and evaporated to dryness. The residue was incubated at 37 degrees Celsius for 120 minutes with 50 micro Liter of steroid diluent. After incubation, 100 micro Liter of sex-hormone-binding-globulin inhibitor, 400 micro Liter of125 Iodine-testosterone, and 400 micro Liter of anti-testosterone were added and incubated overnight. A separation antibody was then added and allowed to incubate for 90 minutes at 37 degrees Celsius. After 15 minutes 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. Intraassay and interassay 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%).

[18]Salivary testosterone level, being highly correlated with the unbound fraction of circulating testosterone, is assumed to be a precise indicant of the behaviorally active fraction of testosterone (Riad-Fahmy et al., 1982; Wang et al., 1981).

Cortisol data were first collected when the subjects were 13 years of age and also continued to the time they were 16 years of age. During the laboratory visit, the subjects were asked to provide additional salivary samples at six points during the day. The first reading occurred upon arrival at the laboratory and the subjects were asked to place a piece of filtered paper in their mouths for 15 to 20 seconds. This collection was then repeated at five additional points in the afternoon. The filtered papers were analyzed to provide readings on cortisol levels. Since not all of the participants had accurate readings and these cortisol readings were not taken during each laboratory visit we have cortisol information for only 58.4% of the subjects with testosterone data.

In our study, we consider academic and non academic outcomes such as substance abuse, criminal activity and gang activity. Information pertaining to the boys' level of substance use, sexual behavior, delinquency and parent's child rearing practices were assessed with a self-report questionnaire. This information was gathered annually each spring from ages 10 to 17 during visits to the schools the boys attended. Questions pertaining to alcohol, drug, and cigarette use were assessed using a 7 point scale. In our analysis we employ indicator variables for whether in the last year the subject has i) got drunk from alcohol ii) smoked cigarettes iii) used marijuana and iv) used harder drugs such as cocaine, heroin, amphetamines, etc. Responses to delinquency and rearing practice items were ordinal on a 4 point scale that corresponds to never, once or twice, often and very often.[19] We consider total fighting and criminal activity rather than creating indicator variables as in Levitt and Lochner (2001). Individuals were also asked to report whether they were a member of a juvenile gang as well as the type of activities the gang engaged in. Finally, the panel structure of the data also served to cross-validate the information provided by the boys and to identify inconsistencies in

[19] The items were: steal from school; steal from store; steal from home, keep object worth less than $10; steal bicycle, sell stolen goods, keep object worth between $10 and $100, steal objects worth more than $100, breaking and entering; enter without paying; trespassing; take drugs; take alcohol, get drunk, destroy school material, destroy other material, vandalism at school, destroy objects at home, vandalize car, 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 data.

We also consider dropping out of school where being a dropout is defined as an individual who stopped attending school at a point in time whether or not he reentered school at a later time. Dropout status was determined in two methods. First, the subjects completed a questionnaire and provided a self-report. Second this information was verified using the computerized lists of the Montreal school board and the Ministry of Education. If a participant was not on the annual School board list, the Ministry of Education was asked to verify whether he was enrolled in another school board within the province.[20]

Several parental characteristics measured in the kindergarten interview are included in the analysis. These measures include years of schooling for each parent and family structure. Following Gather and Pollack (2003), we define family structure as an indicator of whether in kindergarten the child was being reared in a nuclear household.[21] We use post kindergarten parent interviews to construct long run family income, which we define as the average of parent's income across earlier years as in Solon (1992) and Blau (1999). The children reported parental child reporting practices. Measures of supervision are based on three items, determining i) the presence of a parent / guardian at home after school, ii) parental knowledge of where children are when they go out and iii) parental knowledge of the child's peer group. Parental rules were assessed based on the child's report of their existence as well as whether they were punished if the rules were violated and the severity of the punishment.

[20] For all but one participant did the information match. This participant reported himself as dropped out although he was registered with the School board and it is likely that he dropped out after the official lists were compiled.

[21] Our results are not sensitive to contemporaneous family structure, but we employ the earlier measure in our analysis since it is more prevalent in the data.

4 Empirical Framework

4.1 Economic Model

In this section, we provide a simple two stage model that guides our estimation strategy and describe how we handle concerns regarding endogeneity. In the first stage, altruistic parents select the optimal child rearing practices j^* for child i in period T, which provides the highest indirect utility for their household V_{ij}^*,

$$V_{ij} \equiv V_{ij}(X_i, C_j | I_{iT-1}), \text{ for each } j \text{ available to child } i \tag{1}$$

where X_i are observable family characteristics of the child i; C_j is the time and monetary cost of providing strategy j,and I_{iT-1} is all the information parents have on the full history of the child's behavior and human capital achievement.

Family characteristics of the child may have causal effects if better educated parents are more adept at stimulating their child's interest, identifying developmental problems, structuring educational activities, helping with school work and monitoring as well as influencing the child's peer group. Similarly, family's socioeconomic status may influences the amount of human capital developed by the child since the additional income allows parents to meet their child's health or nutritional needs, live in better neighborhoods, afford tutoring and after school activities among other channels.

In the second stage, the child decides whether to engage in risky activity, R, or to engage in non-risky activity, N. Mathematically, define a child i's instantaneous utility at time t, u_{it} as

$$u_{it} = u(c_{it}, l_{it}; p_{ij^*t}, h_{it}, X_i, \varepsilon_{it}) \tag{2}$$

where u is any twice differentiable function, c_{it} is a child's current consumption vector, l_{it} is the current amount of leisure, p_{ij^*t} are parental practices, ε_{it} are utility shocks and h_{it} reflects the internal hormonal factors that define an individual's exogenous biological state at time t. We assume

these variables are measured prior to making the decision to engage in risky behavior. Hormonal factors directly affect the probability of engaging in risky activity. The selection of risky behavior such as sexual activity or drug use directly affects the child's consumption vector.

We define U_{it}^N to be a fully rational person's expected intertemporal utility at period t for engaging in behavior N,

$$U_{it}^N = u_{it}^N + \sum_{s=t+1}^{\infty} \delta_N^{s-t} u_{is} \tag{3}$$

where δ_N is a discount factor. Similarly define U_{it}^R to be a fully rational person's expected intertemporal utility at period t for engaging in behavior R,

$$U_{it}^R = u_{it}^R + \sum_{s=t+1}^{\infty} \delta_R^{s-t} u_{is} \tag{4}$$

where δ_R is a discount factor such that $0 \preceq \delta_R < \delta_N \preceq 1$. An individual engages in risky activity if $U_{it}^R > U_{it}^N$ since they attach too little weight to their well being later in life. Note, certain risky behaviors such as sexual activity may yield higher instantaneous utility. Higher hormone levels lead people to be excessively myopic relative to what would maximize their true welfare.[22]

4.2 Empirical Model

We do not have data rich enough to directly estimate the structural model described above. Thus, we empirically model risky behavior by taking a linear approximation to the intertemporal utility functions in equations 3 and 4 yielding

$$\alpha_p^N p_{it} + \alpha_h^N h_{it} + \alpha_x^N X_{it} + \varepsilon_{it}^N \tag{5}$$

$$\text{and } \alpha_p^R p_{it} + \alpha_h^R h_{it} + \alpha_x^R X_{it} + \varepsilon_{it}^R \tag{6}$$

respectively where $\alpha' s$ are loading factors and the X matrix contains family characteristics.

[22] The notion that changes in exogenous states can affect the well being of an individual and the effectiveness of public policy has been formally modeled in Laibson (2001) and Bernheim and Rangel (2002).

An individual engages in risky behavior if $U_{it}^R > U_{it}^N$ which implies that

$$\alpha_h^R h_{it} + \alpha_{hc}^R hc_{it} + \varepsilon_{it}^R - (\alpha_h^N h_{it} + \alpha_{hc}^N hc_{it} + \varepsilon_{it}^N) \geq 0 \tag{7}$$

We define Y_{it}^* as the propensity to engage in risky behavior and Y_{it} as dichotomous indicators for whether adolescent (i) in year (t) has been engaged in a particular delinquent, criminal or antisocial behavior. Rearranging terms yields

$$\begin{aligned} Y_{it}^* &= \theta_0 + \theta_1 X_{it} + \theta_2 h_{it} + \theta_3 p_{it} + \mu_{it} \\ Y_{it} &= 1 \; if \; Y_{it}^* \geq 0 \end{aligned} \tag{8}$$

where $\mu_{it} = \varepsilon_{it}^R - \varepsilon_{it}^N$.[23]

4.3 Estimation and Omitted Variable Bias

Our first primary aim is to determine if omitting data on time varying biological data leads to bias on nurture variables. We consider estimation of the following two equations for each risky behavior. First, we estimate a model which includes hormonal variables

$$Y_{it} = 1\{\gamma_0 + \gamma_1 X_{it} + \gamma_2 h_{it-1} + v_t + \varepsilon_{it}^\gamma\} \tag{9}$$

as well as a model that excludes these variables

$$Y_{it} = 1\{\delta_0 + \delta_1 X_{it} + v_t + \varepsilon_{it}^\delta\} \tag{10}$$

[23] For the model to be consistent with a large body of evidence from behavioral genetics and psychology one could allow future hormone levels to be influenced by immediate past choices. This property can be used to explain why several of these risky behaviors become addictive. However, Dabbs (1992) finds that in humans individual differences in hormone levels are large and stable over time since.(and as discussed in the earlier section) success or failure in risky behavior may lead to a large and immediate change in hormone levels but very small changes over longer periods of time. Continued success or failure in risky behavior is necessary for these changes to permanently alter hormone levels.

from the analysis. In each equation, we control for year unobserved heterogeneity v_t. We compare the coefficient estimates between equations 9 and 10. A linear probability estimator is used since alternative binary response models estimate coefficients up to scale. For policy purposes, $\delta_1 = \gamma_1$ if either $\gamma_2 = 0$ or the hormone variables (h_{it-1}) are uncorrelated with the parental characteristics (X_{it}).[24]

Intuitively there is a direct and indirect effect from leaving hormonal levels out of a regression. The direct effect operates through the relationship between hormone levels and the outcome variable (γ_2) and the indirect effect operates through the relationship between hormone levels and the other regressors. Deriving the sign of omitted variable bias when there are multiple regressors in a model is difficult and cannot be done with sample covariances. Further, reducing omitted variable bias, the estimated variance of the coefficient estimates decreases leading to more precise estimates.

4.3.1 Endogeneity

Two empirical concerns relate to the endogeneity of h_{it} and p_{it}. In our analysis we use hormone readings taken approximately a year prior to the individual subject surveys. Our hormone measures can be treated as a predetermined and exogenous regressors.[25] We assume that the measures reflect baseline readings (i.e. trait and not state). Each of these hormonal readings were taken at a laboratory at the University of Montreal and we account for differences in time of the reading relative to normal wake-up to a quadratic. A final concern may exist that testosterone levels are correlated with the genotype that affects the development of human capital and is implicitly

[24] Note for several outcomes such as fighting and criminal activity the dependent variable is not a discrete dummy. We also OLS estimators and these equations can be thought of as the direct latent variable equation in equation 8. Also we include time of reading and pubertal status in the hormone vector.

[25] This distinction is of great importance as studies in other disciplines generally regress past outcomes on current testosterone readings which leads to results that are difficult to interpret. The exogeneity of hormone readings is not solved in longitudinal studies as hormone levels are implicitly required to be strictly exogenous when within-individual models are estimated.

included in μ_{it}. Since the concordance rate in testosterone and cortisol between parents and their sons is extremely low, we suggest any potential correlations are likely to be small.

Concerns regarding the endogeneity of p_{it} are addressed using an instrumental variables procedure.[26] We make use of two randomized interventions that occurred at an early age for the parents of these children. The first intervention was designed to promote academic success and occurred in 28 of the 53 schools from which the sampling occurred. The program was offered by the Montreal Catholic School Commission in randomly chosen low income neighborhoods. The program provided the opportunity for parents of the boys to attend their child's class one afternoon a week. The main focus of these meetings was to inform parents about the philosophy of the program, advise them on personal problems and teach positive child rearing practices vis a vis: supervision of homework, consistency in discipline and stimulating themselves and their children. Since attendance for the program was not mandatory the resulting estimate should be interpreted as a compiler average causal effect.

The second intervention occurred three years later when the boys were 7 years of age and normally starting second grade. Data from the kindergarten interviews of teachers as well as their parents were used to compute delinquency risk using the disruptiveness scale of the Social Behavior Questionnaire. In total, 259 boys were considered to be disruptive and were randomly assigned to one of the following groups i) treatment prevention group (n=75), ii) sensitization-contact group (n=124) iii) control group (n=60). The treatment prevention group included two components

[26] We argue that an instrumental variables approach is preferred to using a fixed effect strategy to estimate the causal effect of child rearing practices. While the fixed effects approach controls for family unobserved heterogeneity it implicitly assumes that child rearing practices do not respond to either current or past random shocks to children's behavior. Intuitively, it is highly unlikely that unobserved to the econometrician factors that are related to both children's behavior and their parents' child rearing practices are fixed over time. Rather it is reasonable to expect that parent's would respond to transitory shocks such as changes in the composition of a child's peer group, changes in after school activities as well as transitory socioeconomic circumstances.

targeting both the boys themselves as well as their families when the subjects were between 7 and 9 years old. The family component, adapted from the Oregon Social Learning Center, intended to improve parents' disciplinary practices and supervision deficits. The sensitization-contact group was designed to assess the presence of Hawthorne effects. Each family was assigned a contact person who would offer advice in the event of a crisis. In our analysis, we include scores from the delinquency scale in the first stage regressions since assignment to the intervention is based on observables. Our instruments are a series of program indicator variables equal to one if assigned to that program.

5 Empirical Results

Ordinary least squares regressions of our baseline equations (9 and 10) that treat child rearing practices as exogenous are presented in Tables 1 and 2. In each table, the top panel presents estimates where hormones are included (equation (9)) and the bottom panel excludes this information (equation (10)).

Table 1 presents information for the testosterone sample. Subjects with higher testosterone levels are associated with being members of a juvenile gang, selling drugs, using drug excluding marijuana, smoking cigarettes and dropping out of school. Testosterone levels are not associated with either alcohol or marijuana use. The marginal effects of testosterone are large relative to paternal education and small relative to family income. Evaluated at mean levels a 1% decrease in father's education is equivalent leads to a 9.68% and 14.86% increase in testosterone levels for illicit drug use and the selling of narcotics respectively.

Tests of the joint significance of the hormone variables are accepted for each outcome at the 5% level. The omission of testosterone from the estimating equation in general does not significantly affect the magnitude of any of the nurture variable coefficients with the exception of father's education. Excluding testosterone results in the linear effect of father's education becoming statistically

significant for juvenile gang membership, and both the linear and quadratic term becoming significant for the amount of property crime and delinquent activities. For the remaining outcomes, the concave effects of father's schooling generally dampens in magnitude with the inclusion of testosterone. Similarly, the effect of family permanent income is generally smaller when testosterone is included in the specification. Finally, parental supervision is negatively and significantly related to each outcome irrespective of the inclusion of the testosterone readings.

Table 2 presents results for the cortisol sample. Cortisol levels are only significantly associated with a few of the more problematic adolescent behaviors. Lower cortisol levels are associated with both juvenile gang membership, amount of property crime activities and the use of illicit drugs such as the use of cocaine and amphetamines. Further, a concave relationship exists between cortisol levels and gang activity.

Tests of the joint significance of the cortisol variables are accepted for each outcome at the 5% level, supporting their inclusion. The inclusion of cortisol in the specification has substantial impacts on the coefficients and fit of the regression explaining school drop out. The inclusion of cortisol results in the negative effect of family income and parental rules on drop out status becoming insignificant, while parental supervision gains statistical significance. The greater fit is explained by the inclusion of the difference between wake-up and time of the hormonal reading in the regression equation. Specifically, there is a strong association in the data between wake-up time in year $t-1$ and being a high school drop out in year t.[27] Similarly, permanent income becomes smaller in magnitude when cortisol is included in the specifications explaining the last four outcomes in Table 2. For the amount of criminal activity, the effect of a 1% decrease in family income is equivalent to a 2.61% decrease in cortisol levels evaluated at mean values. In contrast to testosterone, the marginal effects of cortisol are generally the same size or larger than those of permanent income.

Instrumental variable estimates of equations 9 and 10 that account for endogenous child rearing

[27] Note this pattern and substantial improvement in the R-squared criterion also holds for the testosterone sample in Table 1.

practices are presented in Table 3. Since there are few differences in sign and magnitude for both the nurture and hormone variables, we present estimates of the child rearing practices only. These results are striking and substantially larger in magnitude relative to Tables 1 and 2. For the testosterone sample, parental supervision becomes insignificant only for two outcomes (selling drugs and alcohol use) while that of parental rules becomes insignificant for three outcomes (cigarette smoking, amount of stealing and destructive behavior). Once endogeneity is corrected parental rules develop a significant negative effect relative to the OLS estimate for five outcomes (selling drugs, illicit drug use, marijuana use and alcohol use). The results suggest that parental rules influence common adolescent risky behaviors. Further, active parental supervision has a large causal impact on reducing the likelihood that an individual engages in each risky behaviors; with the exception of selling narcotics.

Similarly, for the cortisol sample parental supervision plays a strong role. Relative to the estimates in Table 2, parental supervision is negatively and significantly related to all of the behaviors with the exceptions of selling narcotics and marijuana use only if cortisol is included in the regression. The IV estimates report a statistically significant relationship with four additional outcomes relative to the OLS estimates. The IV estimates of parental rules are statistically insignificant for all outcomes. This differs from the OLS results that reported a role for parental rules. Hausman tests reject the assumption of exogeneity of child rearing practice for each outcome with both hormone samples.

From a causal perspective, the results are easiest to interpret if the effects of parental practices are the same across the children in the sample. If the effects are heterogeneous, the estimates using information on randomly assigned programs that were designed to promote parent child relationships early in childhood can be interpreted as a compiler average causal effect (as opposed to an intent to treat). That is, the measured effect captures the average effect only for those parents who only received the intervention that improved parental practices since they were assigned to the program.

Estimates from the first stage regression for both parental supervision and parental rules are

provided in Appendix Table 1. Coefficients on the instruments and exogenous regressors in both columns are reasonable in sign and magnitude. Family income and parental education are positively related to child rearing practices. Children living in a nuclear family were less likely to be supervised or be faced with rules. The instruments from the kindergarten level interventions are statistically significant predictors of both parental behaviors and the F-statistics on their joint significance are respectively above current cutoffs (i.e. Staiger and Stock (1997)) for weak instruments.

5.1 Robustness Checks

5.1.1 Growth Rates versus Levels

To examine the robustness of our results we make use of the experiment in nature that occurs during puberty when testosterone levels increase rapidly.[28] We modify equation 9 to include hormone growth rates Δh_{ig} (as opposed to levels)

$$Y_{itg} = 1\{\gamma_0 + \gamma_1 X_{it} + \gamma_2 \Delta h_{ig_t} + v_g + \varepsilon_{itg}\} \tag{11}$$

where v_g accounts for permanent time gap heterogeneity.

OLS estimates of equations 10 and 11 are presented in Table 4. Growth rates in testosterone are significantly associated with each outcome with the exceptions of dropping out of school and fighting. In general, including testosterone growth rates reduces the magnitude of the coefficients associated with father's schooling and permanent family income. Interestingly, for school drop out behavior, illicit drug use and marijuana use, the permanent income coefficient becomes positive and statistically significant only when testosterone growth rates are included. In contrast, the linear effect of father's education becomes insignificant for alcohol use, whereas the linear effect of father's education becomes significant for fighting, if testosterone growth rates are included. Finally, as in Table 1, parental supervision is significantly and negatively associated with each of

[28] Very few subjects had multiple cortisol readings so we do not consider changes in cortisol.

the behaviors with the exception of gang membership. Parental rules are only significantly related to three outcomes but have a surprising positive relationship with each.

The magnitude of the effects of testosterone growth rates are similar to those of testosterone levels relative to the other regressors when evaluated at mean values. For example, a 1% increase in father's education is equivalent to a 3.78% and 5.59% decrease in testosterone growth rates in illicit drug use and selling narcotics respectively. Similarly a 1% increase in family income is equivalent to a 0.15% and 0.065% increase and 0.28% decrease in testosterone growth for illicit drug use, property crime and alcohol use respectively.

Table 5 presents instrumental variables results of equation 11. As with the testosterone levels sample there are large differences relative to the OLS estimates. However, the effects of parental supervision while large in magnitude, are statistically insignificant for five outcomes including cigarette smoking, marijuana use, gang membership, stealing and delinquency.[29] Parental supervision seems to be an effective deterrent for infrequent behaviors. Parental rules are negatively and significantly related to the selling of narcotics and gang membership. Permanent income is significantly positively related to five outcomes and the magnitude of its effect increases when testosterone growth rates are included. As before, the effect of father's education reduces in magnitude when testosterone growth rates are included. Hausman tests reject the assumption of exogeneity of child rearing practices for each outcome with this sample.

5.1.2 Alternative Biological Measures

While the majority of data sets used to conduct economic analysis do not contain accurate information on hormones, they do provide information on a variety of anthropometric measures of health status. Since numerous biological changes occur during puberty outside of changes in hormone levels, we first consider including height to check the robustness of our results. The role of

[29] Note the standard errors are corrected for correlation within individuals only, and do not account for repeated units for those individuals who had testosterone collected in three or more years.

height has been linked to a variety of socioeconomic outcomes and Behrman and Rosenzweig (2004) demonstrated that the relationship between height and wages cannot be ascribed to different family circumstances or differences in time invariant genetic endowments. However, one could argue that either hormones are a proxy for height or that height serves as a proxy variable for hormone levels since height itself is a result of not just early nutrition and health inputs but is formed by genetic, hormonal and biochemical factors.

We reestimate equations 9, 10 and 11 including direct (not self-reported) measures of height. OLS estimates of the height and hormone variables are presented in Table 6. The first panel presents height and testosterone levels. Notice that height is positively and significantly related to the first five outcomes as well as property crime and delinquency if testosterone levels are omitted. This result is only robust for cigarette smoking and alcohol use if testosterone is included. Interestingly, the significant and negative effect of height on fighting nearly doubles in magnitude with testosterone levels. Relative to Table 1, a significant relationship between testosterone levels and fighting as well as violent crime appears, while that between dropping out of school and cigarette smoking disappears.[30]

The third panel contains estimates with testosterone growth rates. Notice that if growth rates are omitted from the specification, height is associated with a variety of risky behaviors. However, including testosterone growth rates renders the effect of height to be statistically insignificant for 4 of the 8 risky behaviors. In contrast to testosterone levels, the inclusion of testosterone growth rates does not affect the significance but rather generally dampens the magnitude of the height variable. The results for the height coefficients for cigarette smoking and alcohol use were robust to the inclusion of testosterone growth rates. Evaluated at mean values, the effect of a 1% increase in height is equivalent to a 0.18% increase in 0.84% decrease in testosterone growth rates for illicit drug use alcohol use respectively. The marginal effect of testosterone (in levels and growth rates)

[30]In general, these differences are due to the omission of individuals with testosterone but not height measures.

is smaller in magnitude than that of height for each discrete outcome

We also examined whether these results were robust to accounting for weight and changes in anthropometric measures (i. e. growth spurts) in the remaining panels.[31] The results are robust to the inclusion of weight. Interestingly, weight is only associated with fighting and gang member; with the latter finding being robust to the inclusion of testosterone. Finally, using changes in height and weight (as opposed to levels), the significant associations with gang membership, fighting and delinquency outcomes disappears with the inclusion of testosterone growth rates. Overall, we interpret these results as suggesting that for several outcomes, levels of adolescent height proxy for testosterone levels, but changes in testosterone have a weaker association with both levels and changes in height. Since measures of height and hormones in adolescence and hormones are potentially products of early experiences as well as genetic, hormonal and biochemical factors we attempt to understand their relationships with earlier biological and behavioral measures.

5.2 Sources of Heterogeneity in Biological Measures

5.2.1 Initial Health Conditions

Since differences in hormone levels across individuals are fairly stable over time, one may postulate that the hormone levels are driven by initial health endowment. We collected information on the birth delivery from hospital records for 831 of the 1034 subjects who participated in the original study. These records provide detailed information on anthropometric measures of health at birth (including weight, height, circumference of the head), initial health status as measured by both the 1 minute and 5 minute Apgar scores, as well as detailed information on any sort of complication or medical intervention. Our interest is to first determine whether there is an association between any of these variables from the birth delivery with adolescent hormone and anthropometric measures.

Table 7 presents OLS regression results relating testosterone, cortisol and adolescent height

[31] We also considered nonlinearities in height and body mass index.

with birth delivery information. Each column is associated with a different specification where we progressively add information from the delivery. In addition, we control for mother's age at birth in several of the regressions since hormones are known to transfer between parent and child in utero.

In column 1, we note that there is positive relationship between birth weight and testosterone levels. As controls are added to the regression the statistical significance of birth weight disappears. Interestingly, only early infant sickness and factors related to the mother's pre-natal care are related to adolescent testosterone levels. In terms of adolescent cortisol levels, birth weight included as the sole input is negatively and significantly related to cortisol only in column 1. As controls are added to the specification only the negative and significant relationship between birth height and cortisol levels remains.

In contrast to hormone levels, adolescent height is highly associated with a number of initial health variables. Taller adolescents are associated with greater birth weight irrespective of the inclusion of controls. There is no additional reduction in height for child born under 2500 grams. Initial health as measured by the 5 minute Apgar score is positively and significantly related to height. Surprisingly, children who are born in sickness are taller in adolescence. Finally, children whose mothers are older at birth tend to be taller in adolescence. As a whole, adolescent height has strong associations with early health measures, whereas there are few if any significant associations between these measures and hormone levels suggesting that the hormone levels may be related to early experiences.

5.2.2 Early Behavior

We next examine the potential association between hormone levels and early behavior rated by kindergarten teachers using the Social Behavior Questionnaire. Teachers indicated whether items regarding the subject: did not apply (0), applied sometimes (1) or applied frequently (2). The results of the regressions of hormone levels on initial behavior are shown in Table 8. With the sole

exception between inattentive behavior in kindergarten and adolescent testosterone levels, none of the kindergarten behaviors are related individually to later hormone or height measures. Surprisingly, inattention in kindergarten is significantly negative related to testosterone and unrelated to cortisol which is counter-intuitive. While inattention has been shown to be a good predictor of school failure in the developmental psychology literature the general channel postulate has students suffering adjustment problems due to school failure leading to increased cortisol and reduced testosterone, yet the increased stress does not materialize as increase cortisol. Further, tests of joint significance of the early behaviors reject a significant association between kindergarten behavior and adolescent testosterone levels, cortisol levels and height.

While hormone levels are unrelated to early experiences it remains possible that they may capture information on recent experiences. Not surprisingly, a regression of hormone levels on recent teacher readings yield significant different results.[32] We find there are strong relationships between contemporaneous teacher rated behavior and both current and one period lagged hormone levels. Specifically, subjects with higher testosterone levels are more likely to have low scores on questions related to pro-social behavior. Subjects with higher cortisol levels receive higher teacher ratings of inaptitude. In contrast there are no significant relationship between current or lagged height with either early or contemporaneous teacher ratings of behavior. Taken with the results from the birth records, we suggest these results indicate that hormone levels proxy for the dynamic relationship between genes and an individual's environment.

6 Conclusions

This paper considers the possible actions that parents and society can take to reduce the likelihood that teenagers engage in risky behavior. While most of the attention in the social sciences has linked teenage risky behavior with either parental controls or external factors, they implicitly

[32] These results are available from the authors by request.

assumed away any role that time-varying biological forces may play. Biological changes at puberty are triggered by events in an adolescent's brain, which instruct the pituitary gland to produce hormones that stimulate the secretion of sex hormones. These hormones, in interaction with external influences, have powerful effects on many tissues of the body, including the brain. We find strong evidence of significant associations between hormone levels and risk-taking behavior during puberty. Testosterone levels and their growth rates contribute to risky and criminal activity including the selling of narcotics, gang activity, smoking, marijuana, alcohol and other drug use. Cortisol levels are related to gang activity, property crime and illicit drug use. Finally, we find strong evidence that child rearing behaviors are endogenous and the importance of active roles of supervision are generally more successful than inactive roles (such as establishing guidelines and rules) at reducing addictive risky behavior and gang activity while the converse holds for non-addictive behaviors such as dropping out of school and criminal activity.

From a policy perspective, reducing risky behavior in adolescence is of substantial importance since there is a strong relationship between engaging in risky behaviors as an adolescent and negative consequences later in life. Similar to past research, we find that family socioeconomic status is important for reducing the likelihood that an individual engages in risky activity and these results are robust to the inclusion of biological variables. However, the inclusion of hormone readings substantially affects the importance of other biological measures such as adolescent height, potentially reducing their importance as a form of human capital. Hormone levels are not correlated with initial health information from birth or early kindergarten behavior, suggesting they may proxy for genetic responses to earlier experiences.

While this study has found a strong active role for certain hormones in adolescent risky behavior, several concerns remain. First, recent research in neurobiology has detected that the brain is still developing in adolescence, undergoing significant neuronal transformation, affecting such functions as self-control, emotional regulation, organization and planning. Not only did we not incorporate data on the development state of the frontal lobe which could act as a brake for the subjects in

our study other genetic factors such as the neurotransmitter dopamine could affect risk seeking behavior.[33] Second, we proxy the quality of the environment in which the children were raised with both a family structure indicator and measure of long run family income.

We feel that future theoretical and empirical work on child development in economics should move beyond simply considering the implications of individual and parental actions in determining adolescent outcomes (including the development of cognitive abilities) and consider the dynamic relationship between genes and an individual's environment.[34] Future data collection events should exploit recent advances to incorporate information on genetic, hormonal and data on brain development through affordable MRI imaging that could provide additional insight to the relative role of neurobiological, family and societal influences on juvenile risky behavior. In conclusion, recent years have witnessed an explosion of findings on the causes and correlates of adolescent outcomes in the behavioral and social sciences as well as in neurobiology, but a wide gap remains between research in these areas that if bridged may hold clues as to the direction of future policies to promote child development.

[33] Data on the executive function for a subset of these subjects has been collected and is analyzed in Seguin et al. (2004) and Seguin et al. (1999). Data on genetic materials could be obtained in subsequent waves of collection. See Lehrer et al. (2004) for an example using data collected on genetic markers within a population study.

[34] Carneiro and Heckman (2003) reach a similar conclusion following a survey of recent findings in the biological literature and the economics literature.

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Table 1: Ordinary Least Squares Estimates of Equation Including and Omitting Testosterone Levels

Outcome	Drug Selling	Illicit Drug Use	Marijuana Use	Cigarette Smoker	Alcohol Use	Drop Out of School	Gang Member	Property Crime	Delinquency Index	Fighting Index	Violent Crime
Regression Including Hormones											
Parental Rules	0.026 (0.102)	-0.019 (0.105)	-0.113 (0.145)	0.263 (0.174) ♫	-0.119 (0.159)	-0.082 (0.074)	0.050 (0.125)	1.821 (1.058) €	1.523 (2.307)	-0.327 (0.723)	0.893 (0.485) €
Parental Supervision	-0.139 (0.090) ♫	-0.218 (0.094) ♣	-0.380 (0.129) ♣	-0.737 (0.147) ♣	-0.594 (0.143) ♣	-0.152 (0.074) ♣	-0.196 (0.108) €	-6.650 (0.975) ♣	-17.611 (2.193) ♣	-4.596 (0.656) ♣	-2.448 (0.511) ♣
Years of Mother Educ	-0.164 (0.250)	-0.905 (0.310) ♣	-0.508 (0.354) ♫	-0.933 (0.470) ♣	-0.032 (0.378)	-0.528 (0.237) ♣	-0.047 (0.355)	-0.834 (2.789)	-1.092 (6.044)	0.165 (2.183)	0.715 (1.040)
Years of Mother Edu ^{2}	0.153 (0.145)	0.550 (0.175) ♣	0.331 (0.194) €	0.548 (0.247) ♣	0.066 (0.204)	0.195 (0.109) €	0.065 (0.184)	0.771 (1.424)	1.715 (3.052)	0.069 (1.073)	-0.118 (0.522)
Years of Father Educ	0.199 (0.125) ♫	0.334 (0.152) ♣	-0.080 (0.267)	0.547 (0.360) ♫	0.106 (0.319)	-0.144 (0.155)	0.218 (0.191)	1.852 (1.852)	4.296 (4.042)	1.049 (1.395)	1.681 (0.783) ♣
Years of Father Edu ^{2}	-0.106 (0.056) €	-0.186 (0.066) ♣	0.031 (0.122)	-0.236 (0.162) ♫	-0.044 (0.140)	0.042 (0.059)	-0.123 (0.076) ♫	-0.819 (0.764)	-2.398 (1.716) ♫	-0.872 (0.587) ♫	-0.812 (0.328)♣
Family Income	-0.521 (0.401) ♫	0.250 (0.506)	0.561 (0.656)	0.005 (0.791)	2.381 (0.777) ♣	0.486 (0.395)	-0.291 (0.519)	-8.935 (4.511) ♣	-16.952 (9.935) €	-6.873 (3.394) ♣	-5.056 (1.872) ♣
Testosterone	0.245 (0.354)	0.634 (0.369) €	0.290 (0.496)	0.884 (0.565) ♫	0.308 (0.559)	0.252 (0.190) ♫	0.060 (0.397)	0.827 (2.990)	-1.767 (7.312)	-1.770 (2.550)	-0.338 (1.489)
Testosterone Squared	0.588 (0.393) ♫	-0.682 (0.373) €	0.251 (0.577)	-0.739 (0.712)	-0.158 (0.692)	-0.349 (0.210) €	0.864 (0.458) €	-0.528 (3.416)	7.108 (8.077)	1.932 (2.552)	1.883 (1.688)
R Squared	0.11	0.16	0.15	0.14	0.21	0.42	0.12	0.17	0.22	0.21	0.12
Regression Omitting Hormones											
Parental Rules	0.003 (0.098)	-0.056 (0.101)	-0.125 (0.138)	0.188 (0.170)	-0.120 (0.158)	-0.174 (0.095) €	0.054 (0.122)	1.576 (1.013) ♫	0.779 (2.228)	-0.523 (0.765)	0.778 (0.465) €
Parental Supervision	-0.165 (0.090) €	-0.222 (0.091) ♣	-0.400 (0.129) ♣	-0.728 (0.145) ♣	-0.628 (0.142) ♣	-0.137 (0.083) €	-0.240 (0.110) ♣	-6.796 (0.952) ♣	-18.135 (2.188) ♣	-4.776 (0.671) ♣	-2.481 (0.505) ♣
Years of Mother Educ	-0.194 (0.267)	-0.926 (0.332) ♣	-0.542 (0.361) ♫	-0.931 (0.473) ♣	-0.089 (0.367)	-0.535 (0.265) ♣	-0.100 (0.359)	-1.229 (2.756)	-2.364 (6.043)	-0.314 (2.392)	0.685 (1.018)
Years of Mother Edu ^{2}	0.169 (0.154)	0.569 (0.188) ♣	0.350 (0.200) €	0.555 (0.250) ♣	0.104 (0.201)	0.220 (0.126) €	0.085 (0.187)	1.024 (1.417)	2.523 (3.087)	0.370 (1.178)	-0.084 (0.511)
Years of Father Educ	0.263 (0.132) ♣	0.359 (0.157) ♣	-0.039 (0.265)	0.545 (0.343) ♫	0.189 (0.321)	-0.073 (0.178)	0.292 (0.186) ♫	2.553 (1.807) ♫	6.519 (4.071) ♫	1.841 (1.451)	1.814 (0.757) ♣
Years of Father Edu ^{2}	-0.130 (0.058) ♣	-0.203 (0.070) ♣	0.015 (0.121)	-0.242 (0.154) ♫	-0.083 (0.142)	-0.003 (0.070)	-0.146 (0.074) ♣	-1.140 (0.749) ♫	-3.420 (1.742) ♣	-1.246 (0.622) ♣	-0.877 (0.318) ♣
Family Income	-0.492 (0.388)	0.173 (0.487)	0.525 (0.641)	-0.087 (0.791)	2.370 (0.752) ♣	-0.027 (0.424)	-0.259 (0.494)	-9.334 (4.466) ♣	-18.241 (9.910) €	-7.486 (3.340) ♣	-5.171 (1.820) ♣
R Squared	0.07	0.12	0.13	0.11	0.19	0.12	0.09	0.15	0.17	0.15	0.10
Sample Size	501	500	499	499	498	487	619	672	672	672	672

Note: Robust standard errors clustered at the individual level in parentheses. Regressions include indicators for year of hormone readings as well as family structure. Hormone regressions also account for time of reading and pubertal status. ♣, €, ♫ denote significance at 5%, 10%, 20% level respectively.

Table 2: Ordinary Least Squares Estimates of Equation Including and Omitting Cortisol Levels

Outcome	Drug Selling	Illicit Drug Use	Marijuana Use	Cigarette Smoker	Alcohol Use	Drop Out of School	Gang Member	Property Crime	Delinquency Index	Fighting Index	Violent Crime
Regression Including Hormones											
Parental Rules	-0.083 (0.101)	-0.163 (0.127) ♫	-0.235 (0.199)	-0.026 (0.248)	-0.175 (0.240)	-0.140 (0.162)	0.183 (0.147)	2.196 (1.461) ♫	1.851 (3.016)	-0.348 (0.846)	1.005 (0.726) ♫
Parental Supervision	0.006 (0.104)	-0.184 (0.118) ♫	-0.232 (0.159) ♫	-0.927 (0.220) ♣	-0.617 (0.233) ♣	-0.202 (0.149) ♫	-0.145 (0.127)	-7.215 (1.428) ♣	-18.444 (3.251) ♣	-4.588 (0.840) ♣	-2.671 (0.778) ♣
Years of Mother Educ	-0.077 (0.268)	-0.507 (0.553)	-0.419 (0.606)	-0.723 (0.758)	-0.205 (0.600)	-1.036 (0.488) ♣	-0.157 (0.440)	-1.048 (3.657)	1.325 (8.616)	1.604 (2.481)	0.729 (1.932)
Years of Mother Edu ^{2}	0.104 (0.160)	0.296 (0.304)	0.260 (0.338)	0.439 (0.410)	0.191 (0.331)	0.401 (0.236) €	0.122 (0.233)	0.596 (1.897)	-0.344 (4.419)	-0.946 (1.238)	-0.279 (0.957)
Years of Father Educ	0.095 (0.150)	0.113 (0.199)	0.107 (0.288)	0.319 (0.479)	0.372 (0.438)	-0.058 (0.316)	0.202 (0.248)	0.664 (2.437)	2.683 (5.522)	0.076 (1.868)	1.489 (1.148) ♫
Years of Father Edu ^{2}	-0.043 (0.070)	-0.067 (0.086)	-0.073 (0.124)	-0.155 (0.210)	-0.116 (0.202)	-0.023 (0.130)	-0.108 (0.099)	-0.245 (1.027)	-1.496 (2.361)	-0.372 (0.781)	-0.649 (0.499) ♫
Family Income	-0.377 (0.469)	0.027 (0.643)	-0.758 (0.850)	-0.794 (1.148)	1.751 (1.148) ♫	0.252 (0.760)	-0.408 (0.676)	-12.624 (6.204) ♣	-27.035 (13.23) ♣	-8.461 (4.197) ♣	-7.635 (2.788) ♣
Cortisol	0.482 (1.386)	-1.546 (2.107)	-2.217 (2.291)	1.023 (4.423)	-2.728 (4.332)	-0.254 (14.85)	8.139 (3.693) ♣	-32.861 (18.11) €	-19.486 (50.31)	6.516 (18.31)	11.300 (13.06)
Cortisol Squared	-0.345 (0.326)	-0.821 (0.371) ♣	0.456 (1.784)	-0.584 (1.913)	1.272 (2.298)	-2.855 (47.85)	-0.698 (0.480) ♫	-7.604 (7.35)	-12.157 (20.30)	-0.662 (5.065)	0.652 (5.506)
R Squared	0.09	0.17	0.14	0.22	0.24	0.46	0.13	0.17	0.20	0.20	0.12
Regression Omitting Hormones											
Parental Rules	-0.080 (0.094)	-0.123 (0.113)	-0.185 (0.182)	-0.006 (0.246)	-0.130 (0.227)	-0.298 (0.208) ♫	0.221 (0.154) ♫	2.030 (1.387) ♫	1.696 (2.911)	-0.192 (0.938)	0.978 (0.692) ♫
Parental Supervision	0.007 (0.100)	-0.178 (0.118) ♫	-0.219 (0.161) ♫	-0.902 (0.219) ♣	-0.638 (0.230) ♣	-0.154 (0.166)	-0.149 (0.127)	-7.278 (1.410) ♣	-18.552 (3.222) ♣	-4.644 (0.835) ♣	-2.688 (0.771) ♣
Years of Mother Educ	-0.103 (0.248)	-0.617 (0.543)	-0.498 (0.588)	-0.864 (0.716)	-0.128 (0.551)	-1.018 (0.616) €	-0.142 (0.433)	-1.261 (3.480)	0.180 (8.289)	0.765 (3.107)	1.048 (1.820)
Years of Mother Edu ^{2}	0.114 (0.151)	0.348 (0.300)	0.297 (0.329)	0.498 (0.390)	0.159 (0.310)	0.413 (0.296) ♫	0.116 (0.231)	0.788 (1.822)	0.461 (4.280)	-0.481 (1.526)	-0.404 (0.902)
Years of Father Educ	0.097 (0.155)	0.139 (0.208)	0.085 (0.277)	0.279 (0.457)	0.383 (0.426)	0.046 (0.389)	0.229 (0.248)	1.175 (2.381)	4.273 (5.446)	0.706 (1.995)	1.591 (1.113) ♫
Years of Father Edu ^{2}	-0.047 (0.074)	-0.081 (0.090)	-0.061 (0.117)	-0.145 (0.200)	-0.115 (0.197)	-0.075 (0.160)	-0.121 (0.098)	-0.459 (0.998)	-2.176 (2.309)	-0.645 (0.845)	-0.686 (0.482) ♫
Family Income	-0.422 (0.461)	-0.195 (0.631)	-0.904 (0.813)	-0.994 (1.158)	1.755 (1.125) ♫	-1.240 (0.781) ♫	-0.464 (0.631)	-13.864 (6.063) ♣	-33.062 (13.16) ♣	-10.920 (4.171) ♣	-8.207 (2.694) ♣
R Squared	0.06	0.08	0.10	0.16	0.22	0.17	0.10	0.15	0.17	0.16	0.10
Sample Size	221	221	221	221	220	208	346	387	387	387	387

Note: Robust standard errors clustered at the individual level in parentheses. Regressions include indicators for year of hormone readings as well as family structure. Hormone regressions also account for time of reading and pubertal status. ♣, €, ♫ denote significance at 5%, 10%, 20% level respectively.

Table 3: Instrumental Variables Estimates of Parental Child Rearing Practices Including and Omitting Hormone Levels

Outcome	Drug Selling	Illicit Drug Use	Marijuana Use	Cigarette Smoker	Alcohol Use	Drop Out of School	Gang Member	Property Crime	Delinquency Index	Fighting Index	Violent Crime
Regression Including Testosterone											
Parental Rules	-1.409 (0.684) ♣	-0.938 (0.684) ♫	-0.246 (0.809)	-0.156 (0.987)	-3.349 (1.320) ♣	-0.525 (0.781)	-0.671 (1.059)	-3.827 (9.537)	-13.981 (22.95)	-2.158 (7.203)	1.329 (4.462)
Parental Supervision	-0.454 (0.415)	-1.023 (0.487) ♣	-1.102 (0.612) €	-1.459 (0.795) €	-0.840 (0.991)	-1.093 (0.492) ♣	-1.632 (0.733) ♣	-13.156 (5.199) ♣	-44.305 (12.76) ♣	-12.840 (3.938) ♣	-7.968 (2.428) ♣
Regression Omitting Hormones Testosterone Sample											
Parental Rules	-1.428 (0.682) ♣	-0.815 (0.654)	-0.136 (0.766)	-0.222 (0.966)	-3.192 (1.280) ♣	-0.802 (0.868)	-0.555 (1.051)	-1.279 (9.108)	-7.431 (22.46)	-0.257 (7.212)	2.181 (4.213)
Parental Supervision	-0.509 (0.430)	-0.971 (0.455) ♣	-1.084 (0.584) €	-1.377 (0.776)	-1.217 (0.981)	-1.278 (0.542) ♣	-1.533 (0.671) ♣	-12.818 (5.102) ♣	-44.332 (12.45) ♣	-12.884 (3.840) ♣	-7.359 (2.344) ♣
Sample Size	501	500	499	499	498	487	619	672	672	672	672
Regression Including Cortisol											
Parental Rules	-0.535 (0.767)	-0.090 (1.023)	0.213 (1.117)	0.283 (1.946)	-1.248 (1.585)	0.004 (0.974)	-1.258 (1.650)	-16.479 (21.96)	-11.768 (50.34)	12.662 (14.92)	-3.148 (12.10)
Parental Supervision	-0.392 (0.489)	-1.180 (0.751) ♫	-1.456 (0.906) ♫	-3.622 (1.415) ♣	-1.184 (1.145)	-1.345 (0.643) ♣	-1.589 (1.025) ♫	-14.154 (8.534) €	-45.648 (17.49) ♣	-10.450 (4.836) ♣	-8.087 (3.982) ♣
Regression Omitting Hormones Cortisol Sample											
Parental Rules	-0.491 (0.641)	0.087 (0.875)	0.471 (0.955)	1.044 (1.703)	-1.621 (1.483)	0.114 (1.375)	-1.196 (1.593)	-15.061 (19.22)	-11.583 (45.18)	10.291 (13.10)	-4.832 (10.79)
Parental Supervision	-0.481 (0.463)	-1.128 (0.715) ♫	-1.394 (0.960) ♫	-3.710 (1.508) ♣	-1.639 (1.224) ♫	-1.193 (0.679) €	-1.583 (1.008) ♫	-15.537 (8.163) ♣	-48.148 (17.13) ♣	-10.580 (4.506) ♣	-8.383 (3.884) ♣
Sample Size	221	221	221	221	220	208	346	387	387	387	387

Note: Robust standard errors clustered at the individual level in parentheses. Regressions include controls as in Table 1 and 2 for the respective samples. ♣, €, ♫ denote significance at 5%, 10%, 20% level respectively.

Table 4: Ordinary Least Squares Estimates of Equation Including and Omitting Testosterone Growth Rates

Outcome	Drug Selling	Illicit Drug Use	Marijuana Use	Cigarette Smoker	Alcohol Use	Drop Out of School	Gang Member	Property Crime	Delinquency Index	Fighting Index	Violent Crime
Regression Including Hormones											
Parental Rules	-0.017 (0.127)	-0.065 (0.136)	-0.125 (0.199)	0.370 (0.205) €	-0.025 (0.181)	-0.006 (0.063)	-0.057 (0.107)	1.355 (0.914) ♫	0.395 (2.100)	-0.769 (0.702)	0.825 (0.448) €
Parental Supervision	-0.171 (0.109) ♫	-0.451 (0.115) ♣	-0.477 (0.163) ♣	-0.600 (0.170) ♣	-0.603 (0.152) ♣	-0.112 (0.045) ♣	0.038 (0.093)	-5.630 (0.802) ♣	-14.913 (1.984) ♣	-3.508 (0.576) ♣	-1.698 (0.461) ♣
Years of Mother Educ	-0.204 (0.298)	-1.346 (0.350) ♣	-0.754 (0.384) ♣	-1.049 (0.510) ♣	-0.447 (0.430)	-0.834 (0.197) ♣	0.112 (0.301)	2.397 (2.479)	2.299 (5.626)	1.831 (1.886)	0.404 (1.208)
Years of Mother Edu ^{2}	0.147 (0.170)	0.752 (0.193) ♣	0.450 (0.205) ♣	0.607 (0.263) ♣	0.167 (0.227)	0.333 (0.090) ♣	-0.070 (0.152)	-1.092 (1.249)	-0.532 (2.825)	-0.867 (0.957)	-0.135 (0.586)
Years of Father Educ	0.216 (0.149) ♫	0.388 (0.197) ♣	-0.147 (0.310)	0.653 (0.420) ♫	0.421 (0.400)	-0.076 (0.119)	0.128 (0.190)	-0.570 (1.933)	-2.621 (4.250)	-1.991 (1.390) ♫	1.614 (0.896) €
Years of Father Edu ^{2}	-0.126 (0.065) €	-0.211 (0.087) ♣	0.082 (0.142)	-0.250 (0.196)	-0.163 (0.177)	0.014 (0.047)	-0.075 (0.076)	0.241 (0.777)	0.678 (1.772)	0.388 (0.588)	-0.699 (0.374) €
Family Income	-0.431 (0.506)	1.345 (0.645) ♣	1.671 (0.851) ♣	0.094 (0.912)	2.848 (0.821) ♣	0.841 (0.272) ♣	-0.376 (0.432)	-8.106 (3.783) ♣	-12.028 (8.923) ♫	-7.149 (2.705) ♣	-5.669 (1.804) ♣
Testosterone Growth	0.259 (0.163) ♫	0.205 (0.159) ♫	0.338 (0.257) ♫	0.468 (0.276) €	-0.804 (0.231) ♣	-0.052 (0.058)	0.198 (0.155)	-0.547 (0.879)	-0.508 (2.207)	-0.291 (0.673)	0.243 (0.448)
Testosterone Gr. Squared	0.671 (0.239) ♣	-0.569 (0.218) ♣	0.094 (0.425)	-0.555 (0.463)	1.162 (0.318) ♣	0.077 (0.090)	0.822 (0.260) ♣	2.335 (1.537) ♫	7.232 (3.228) ♣	0.757 (0.792)	1.310 (0.619) ♣
R Squared	0.16	0.25	0.17	0.14	0.20	0.48	0.18	0.21	0.23	0.26	0.09
Regression Omitting Hormones											
Parental Rules	-0.028 (0.128)	-0.086 (0.134)	0.000 (0.193)	0.360 (0.206) €	-0.026 (0.182)	-0.059 (0.086)	0.000 (0.106)	0.000 (0.000)	-0.509 (0.000) ♣	-1.071 (0.000) ♣	000 (0.000)
Parental Supervision	-0.269 (0.108) ♣	0.000 (0.114)	-0.534 (0.162) ♣	0.000 (0.169)	0.000 (0.152)	-0.090 (0.059) ♫	-0.086 (0.095)	-5.800 (0.763) ♣	000 (1.969)	-3.847 (0.608) ♣	-1.774 (0.440) ♣
Years of Mother Educ	-0.215 (0.324)	-1.328 (0.379) ♣	-0.832 (0.389) ♣	-1.046 (0.525) ♣	-0.384 (0.420)	-0.847 (0.226) ♣	0.155 (0.315)	2.255 (2.416)	2.174 (5.592)	2.120 (1.932)	0.291 (1.150)
Years of Mother Edu ^{2}	0.156 (0.184)	0.766 (0.210) ♣	0.478 (0.209) ♣	0.624 (0.272) ♣	0.158 (0.224)	0.368 (0.107) ♣	-0.084 (0.160)	-0.938 (1.237)	-0.056 (2.866)	-0.803 (0.990)	-0.060 (0.563)
Years of Father Educ	0.360 (0.163) ♣	0.425 (0.200) ♣	-0.107 (0.310)	0.703 (0.414) €	0.527 (0.401) ♫	0.077 (0.137)	0.218 (0.189)	0.424 (1.895)	0. 808 (4.260)	-0.806 (1.403)	1.927 (0.861) ♣
Years of Father Edu ^{2}	-0.187 (0.069) ♣	-0.250 (0.088) ♣	0.062 (0.142)	-0.292 (0.192) ♫	-0.215 (0.179)	-0.061 (0.055)	-0.113 (0.076) ♫	-0.148 (0.770) €	-0.823 (1.789)	-0.173 (0.591)	-0.817 (0.361) ♣
Family Income	-0.390 (0.490)	0.003 (0.641)	0.792 (0.832)	-0.152 (0.897)	2.565 (0.812) ♣	0.244 (0.313)	-0.368 (0.430)	-9.420 (3.820) ♣	-17.251 (9.024) €	-9.691 (2.805) ♣	-6.077 (1.731) ♣
R Squared	0.09	0.18	0.14	0.10	0.16	0.15	0.12	0.17	0.15	0.14	0.07
Sample Size	438	436	435	435	435	823	751	821	821	821	821

Note: Robust standard errors clustered at the individual level in parentheses. Regressions include indicators for years between hormone readings as well as family structure. Hormone regressions also account for time of reading and pubertal status. ♣, €, ♫ denote significance at 5%, 10%, 20% level respectively.

Table 5: Instrumental Variables Estimates of Equation Including and Omitting Testosterone Growth Rates

Outcome	Drug Selling	Illicit Drug Use	Marijuana Use	Cigarette Smoker	Alcohol Use	Drop Out of School	Gang Member	Property Crime	Delinquency Index	Fighting Index	Violent Crime
Regression Including Hormones											
Parental Rules	-1.247 (0.595) ♣	-1.276 (0.648) ♣	-0.228 (0.799)	0.729 (0.905)	-2.590 (1.063) ♣	0.262 (0.585)	-1.365 (0.889) ♫	0.798 (8.967)	-14.611 (19.09)	-14.547 (7.404) ♣	6.197 (4.303) ♫
Parental Supervision	0.352 (0.519)	-1.081 (0.621) €	0.288 (0.728)	-0.873 (0.879)	-1.628 (1.140) ♫	-0.696 (0.265) ♣	-0.037 (0.608)	1.319 (5.771)	-15.535 (12.27)	-8.836 (4.424) ♣	0.569 (3.006)
Years of Mother Educ	-0.248 (0.314)	-1.283 (0.416) ♣	-0.798 (0.398) ♣	-1.038 (0.502) ♣	-0.353 (0.532)	-0.726 (0.232) ♣	0.210 (0.335)	0.769 (3.183)	4.438 (8.142)	4.960 (2.937) €	-0.862 (2.149)
Years of Mother Edu ^{2}	0.179 (0.177)	0.668 (0.234) ♣	0.513 (0.214) ♣	0.591 (0.268) ♣	0.025 (0.300)	0.267 (0.111) ♣	-0.126 (0.180)	-0.133 (1.657)	-1.630 (4.217)	-2.557 (1.538) €	0.551 (1.091)
Years of Father Educ	0.043 (0.235)	0.104 (0.311)	-0.103 (0.368)	0.698 (0.481) ♫	-0.146 (0.515)	-0.167 (0.175)	-0.006 (0.272)	0.775 (2.338)	-4.212 (5.981)	-4.410 (2.403) €	2.596 (1.653) ♫
Years of Father Edu ^{2}	-0.077 (0.099)	-0.096 (0.134)	0.051 (0.162)	-0.261 (0.219)	0.061 (0.222)	0.058 (0.066)	-0.036 (0.106)	-0.359 (0.963)	1.195 (2.445)	1.285 (0.982) ♫	-1.066 (0.657) ♫
Family Income	-1.032 (0.804) ♫	2.153 (0.976) ♣	0.767 (1.248)	0.403 (1.333)	4.177 (1.669) ♣	1.279 (0.376) ♣	-0.051 (0.877)	-13.687 (6.282) ♣	-9.379 (15.34)	-0.841 (5.445)	-8.283 (3.974) ♣
Testosterone Growth	0.314 (0.179) €	0.146 (0.216)	0.414 (0.261) ♫	0.440 (0.299) ♫	-0.897 (0.337) ♣	-0.120 (0.070) €	0.197 (0.188)	0.201 (1.017)	-0.262 (2.550)	-0.569 (0.959)	0.372 (0.503)
Testosterone Growth^2	0.805 (0.286) ♣	-0.651 (0.326) ♣	0.240 (0.438)	-0.616 (0.506)	1.042 (0.542) €	-0.013 (0.100)	0.785 (0.272) ♣	3.447 (2.101) ♫	6.870 (4.246) ♫	-0.343 (1.352)	1.770 (0.641) ♣
Regression Omitting Hormones											
Parental Rules	-1.282 (0.562) ♣	-1.148 (0.668) €	-0.065 (0.804)	0.941 (0.928)	-2.744 (1.145) ♣	0.396 (0.602)	-1.336 (0.841) ♫	2.753 (7.868)	-7.997 (16.91)	-10.368 (5.905) €	7.978 (4.442) €
Parental Supervision	0.212 (0.479)	-1.290 (0.620) ♣	0.191 (0.707)	-0.811 (0.824)	-1.944 (1.148) €	-0.696 (0.280) ♣	-0.089 (0.532)	2.988 (5.122)	-11.395 (10.73)	-6.290 (3.389) €	1.988 (2.976)
Years of Mother Educ	-0.288 (0.353)	-1.266 (0.442) ♣	-0.882 (0.403) ♣	-1.031 (0.509) ♣	-0.313 (0.540)	-0.767 (0.264) ♣	0.247 (0.346)	-0.017 (2.998)	2.187 (7.567)	3.978 (2.541) ♫	-1.588 (2.134)
Years of Mother Edu ^{2}	0.201 (0.197)	0.666 (0.250) ♣	0.550 (0.221) ♣	0.616 (0.274) ♣	-0.006 (0.310)	0.313 (0.130) ♣	-0.136 (0.184)	0.432 (1.588)	0.035 (3.999)	-1.819 (1.350) ♫	0.997 (1.107)
Years of Father Educ	0.190 (0.266)	0.062 (0.332)	0.031 (0.393)	0.797 (0.512) ♫	-0.258 (0.572)	-0.008 (0.202)	0.077 (0.271)	2.617 (2.363)	0.966 (5.894)	-2.419 (2.084)	3.607 (1.802) ♣
Years of Father Edu ^{2}	-0.138 (0.113)	-0.095 (0.144)	-0.007 (0.175)	-0.322 (0.231) ♫	0.106 (0.249)	-0.017 (0.079)	-0.070 (0.107)	-1.112 (0.980)	-0.996 (2.441)	0.427 (0.864)	-1.475 (0.726) ♣
Family Income	-0.987 (0.765) ♫	1.994 (0.989) ♣	0.913 (1.220)	0.042 (1.304)	4.136 (1.756) ♣	0.676 (0.377) €	-0.141 (0.777)	-16.771 (5.747) €	-19.835 (13.61) ♫	-6.456 (4.293) ♫	-10.105 (4.018) ♣
Sample Size	438	436	435	435	435	823	751	821	821	821	821

Note: Robust standard errors clustered at the individual level in parentheses. Regressions include indicators for years between hormone readings as well as family structure. Hormone regressions also account for time of reading and pubertal status. ♣, €, ♫ denote significance at 5%, 10%, 20% level respectively.

Table 6: Ordinary Least Squares Estimates of Equation with Anthropometric Measures Including and Omitting Testosterone

Outcome	Drug Selling	Illicit Drug Use	Marijuana Use	Cigarette Smoker	Alcohol Use	Drop Out of School	Gang Member	Property Crime	Delinquency Index	Fighting Index	Violent Crime
Regression Including Hormones											
Height	-0.739 (1.292)	-0.330 (1.779)	2.702 (2.546)	6.186 (2.886) ♣	11.874 (2.859) ♣	-0.004 (1.003)	-0.245 (2.460)	16.071 (17.233)	29.583 (40.933)	-37.748 (15.421) ♣	9.883 (7.474) ♫
Testosterone	0.485 (0.344) ♫	0.455 (0.317) ♫	0.004 (0.440)	0.356 (0.493)	-0.075 (0.483)	-0.109 (0.150)	-0.236 (0.381)	2.618 (2.953)	5.552 (7.083)	3.283 (2.532) ♫	-0.220 (1.379)
Testosterone Squared	0.377 (0.384)	-0.490 (0.329) ♫	0.570 (0.516)	-0.248 (0.594)	0.135 (0.593)	0.073 (0.157)	1.168 (0.438) ♣	-1.902 (3.409)	-0.299 (8.219)	-3.364 (2.483) ♫	1.842 (1.567)
R Squared	0.13	0.14	0.13	0.15	0.20	0.38	0.11	0.19	0.24	0.22	0.16
Regression Omitting Hormones											
Height	1.860 (1.466)	1.310 (1.523)	4.989 (2.253) ♣	5.371 (2.569) ♣	13.042 (2.478) ♣	0.394 (1.116)	2.080 (2.223)	26.123 (14.812) €	77.651 (36.038) ♣	-17.875 (13.802) ♫	11.311 (6.697) €
R Squared	0.06	0.11	0.11	0.12	0.19	0.10	0.08	0.16	0.19	0.16	0.13
Sample Size	444	443	442	442	441	281	423	443	443	443	443
Adding Weight Regression Including Hormones											
Height	-0.004 (1.627)	-0.509 (1.975)	2.709 (2.888)	6.771 (3.232) ♣	11.275 (3.079) ♣	-0.596 (1.257)	-3.311 (2.740)	22.608 (19.965)	15.443 (48.543)	-55.365 (18.845) ♣	8.442 (8.808)
Weight	-0.001 (0.001)	0.000 (0.001)	-0.000 (0.002)	-0.001 (0.002)	0.001 (0.002)	0.001 (0.001)	0.004 (0.002) ♣	-0.008 (0.010)	0.018 (0.025)	0.023 (0.010) ♣	0.002 (0.005)
Testosterone	0.504 (0.345) ♫	0.450 (0.317) ♫	0.004 (0.444)	0.370 (0.492)	-0.089 (0.491)	-0.119 (0.154)	-0.319 (0.381)	2.774 (2.955)	5.214 (7.129)	2.862 (2.501)	-0.255 (1.383)
Testosterone Squared	0.364 (0.378)	-0.487 (0.329) ♫	0.570 (0.518)	-0.258 (0.587)	0.144 (0.604)	0.076 (0.156)	1.230 (0.412) ♣	-2.015 (3.353)	-0.055 (8.408)	-3.059 (2.577)	1.867 (1.585)
R Squared	0.13	0.14	0.13	0.15	0.20	0.39	0.13	0.19	0.25	0.23	0.16
Regression Omitting Hormones											
Height	2.321 (1.787) ♫	0.856 (1.801)	4.700 (2.704) €	5.837 (3.018) €	12.320 (2.837) ♣	-0.462 (1.485)	-1.566 (2.686)	30.117 (18.128) €	51.080 (45.735)	-42.594 (18.274) ♣	10.097 (8.110)
Weight	-0.001 (0.001)	0.001 (0.001)	0.000 (0.002)	-0.001 (0.002)	0.001 (0.002)	0.001 (0.001)	0.004 (0.002) ♣	-0.005 (0.010)	0.032 (0.027)	0.029 (0.011) ♣	0.001 (0.005)
R Squared	0.06	0.11	0.11	0.12	0.19	0.11	0.10	0.16	0.19	0.17	0.13
Sample Size	444	443	442	442	441	281	423	443	443	443	443
Using Growth Rates Regression Including Hormones											
Height	-1.612 (2.065)	-4.671 (2.659) €	4.005 (3.281)	5.694 (3.449) €	8.819 (3.327) ♣	-0.273 (0.782)	-3.923 (3.240)	18.678 (22.353)	44.640 (52.141)	-28.920 (19.260) ♫	17.464 (7.650) ♣
Testosterone Growth	0.290 (0.170) €	0.181 (0.166)	0.184 (0.271)	0.321 (0.277)	-0.850 (0.234) ♣	-0.052 (0.061)	0.466 (0.254) €	0.488 (1.330)	1.109 (4.046)	0.812 (1.314)	-0.074 (0.795)
Testosterone Growth^2	0.664 (0.245) ♣	-0.437 (0.220) ♣	0.253 (0.438)	-0.394 (0.453)	1.028 (0.322) ♣	-0.015 (0.099)	0.534 (0.377) ♫	0.287 (2.156)	4.665 (6.403)	-1.181 (1.754)	2.404 (1.249) €
R Squared	0.19	0.23	0.16	0.14	0.20	0.47	0.15	0.21	0.26	0.26	0.19

Regression Omitting Hormones											
Height	0.625	-3.660	5.114	6.032	9.644	0.459	-1.119	21.492	80.704	-9.306	19.310
	(2.298)	(2.649) ♫	(3.174) ♫	(3.311) €	(3.147) ♣	(0.884)	(3.182)	(20.451)	(49.493) ♫	(18.129)	(7.409) ♣
R Squared	0.08	0.16	0.13	0.11	0.15	0.14	0.09	0.16	0.17	0.15	0.14
Sample Size	394	392	391	391	391	394	377	392	392	392	392
Growth Rates and Weight Regression Including Hormones											
Height	-0.621	-3.993	5.787	7.821	7.940	-1.182	-7.568	25.459	43.260	-39.788	14.207
	(2.274)	(2.797) ♫	(3.537) ♫	(3.626) ♣	(3.461) ♣	(0.910) ♫	(3.314) ♣	(24.48)	(57.337)	(21.452) €	(8.329) ♣
Weight	-0.001	-0.001	-0.002	-0.003	0.001	0.001	0.005	-0.009	0.002	0.015	0.004
	(0.001)	(0.001)	(0.002)	(0.002) ♫	(0.002)	(0.000) ♣	(0.002) ♣	(0.009)	(0.024)	(0.009) €	(0.005)
Testosterone Growth	0.275	0.171	0.156	0.287	-0.836	-0.038	0.527	0.380	1.131	0.985	-0.022
	(0.166) €	(0.165)	(0.265)	(0.269)	(0.235) ♣	(0.063)	(0.242) ♣	(1.288)	(4.016)	(1.364)	(0.824)
Testosterone Growth^2	0.695	-0.415	0.312	-0.323	0.998	-0.044	0.414	0.511	4.619	-1.541	2.296
	(0.228) ♣	(0.216) €	(0.419)	(0.425)	(0.333) ♣	(0.101)	(0.343)	(2.038)	(6.367)	(1.893)	(1.312) €
R Squared	0.19	0.23	0.17	0.15	0.20	0.48	0.18	0.21	0.26	0.27	0.19
Regression Omitting Hormones											
Height	1.639	-2.772	7.029	8.677	8.535	-0.267	-5.177	26.892	73.231	-23.879	16.096
	(2.530)	(2.798)	(3.522) ♣	(3.526) ♣	(3.345) ♣	(1.201)	(3.407) ♫	(23.26)	(56.761) ♫	(21.147)	(8.144) ♣
Weight	-0.001	-0.001	-0.002	-0.003	0.001	0.001	0.005	-0.007	0.010	0.019	0.004
	(0.001)	(0.001)	(0.002)	(0.002) ♫	(0.002)	(0.001)	(0.002) ♣	(0.010)	(0.027)	(0.010) €	(0.005)
R Squared	0.08	0.17	0.14	0.12	0.16	0.15	0.12	0.16	0.17	0.16	0.14
Sample Size	394	392	391	391	391	394	377	392	392	392	392
Using Anthropometric Changes with Hormone Growth Regression Including Hormones											
Height Change	-6.652	-12.461	-10.202	-9.104	-3.291	0.501	-7.765	48.785	-44.753	5.498	20.412
	(5.047) ♫	(5.963) ♣	(7.996)	(8.792)	(8.524)	(2.154)	(7.882)	(52.843)	(121.9)	(40.869)	(17.454)
Weight Change	-0.003	-0.005	-0.009	-0.006	0.002	0.002	0.008	-0.009	-0.001	0.024	-0.000
	(0.002) ♫	(0.003) €	(0.004) ♣	(0.004) ♫	(0.004)	(0.001) ♣	(0.003) ♣	(0.017)	(0.046)	(0.019)	(0.009)
Testosterone Growth	0.311	0.218	0.241	0.371	-0.826	-0.057	0.462	0.380	1.220	0.649	-0.112
	(0.163) €	(0.156) ♫	(0.260)	(0.269) ♫	(0.240) ♣	(0.065)	(0.252) €	(1.401)	(4.126)	(1.370)	(0.831)
Testosterone Growth^2	0.539	-0.697	0.099	-0.470	1.158	0.006	0.500	0.953	5.036	-1.227	2.883
	(0.231) ♣	(0.215) ♣	(0.398)	(0.425)	(0.345) ♣	(0.102)	(0.367) ♫	(2.331)	(6.654)	(1.909)	(1.329) ♣
R Squared	0.20	0.25	0.18	0.14	0.18	0.47	0.16	0.21	0.27	0.27	0.19
Regression Omitting Hormones											
Height Change	-4.582	-9.227	-10.056	-4.971	-7.099	-0.487	-13.113	5.757	-206.378	-82.807	9.237
	(4.190)	(5.495) €	(7.133) ♫	(7.906)	(8.019)	(2.286)	(7.434) €	(47.751)	(114.960) €	(38.340) ♣	(17.886)
Weight Change	-0.003	-0.006	-0.010	-0.008	0.001	0.001	0.007	-0.007	-0.005	0.026	-0.005
	(0.002) ♫	(0.003) ♣	(0.004) ♣	(0.004) ♣	(0.004)	(0.001)	(0.003) ♣	(0.017)	(0.046)	(0.018) ♫	(0.009)
R Squared	0.09	0.18	0.15	0.11	0.14	0.14	0.10	0.16	0.18	0.16	0.13
Sample Size	393	391	390	390	390	393	376	391	391	391	391

Note: Robust standard Errors in parentheses. Regressions include time indicators as well as family structure, education and income. Hormone regressions also account for time of reading and pubertal status. . ♣, €, ♫ denote significance at 5%, 10%, 20% level respectively.

Table 7: Effect of Initial Health Outcomes on Subsequent Hormone Levels and Anthropometric Measures

TESTOSTERONE							
Birth Weight	12.403 (8.703) ♫	15.519 (14.336)	16.128 (14.981)	16.227 (15.875)	17.648 (16.823)	18.025 (16.916)	17.494 (17.075)
Birth Week	-1.457 (2.163)	-1.873 (2.256)	-1.684 (2.403)	-0.750 (2.582)	-0.750 (2.631)	-0.963 (2.725)	-0.696 (2.714)
Birth Height of Body	N/A	0.730 (1.677)	0.802 (1.634)	0.733 (1.703)	0.690 (1.805)	0.616 (1.813)	0.667 (1.794)
Birth Head Circumference	N/A	-3.027 (3.597)	-3.064 (3.606)	-3.250 (3.757)	-3.557 (3.856)	-3.435 (3.860)	-3.512 (3.904)
Mother's Age at Birth	N/A	0.031 (1.112)	0.010 (1.115)	0.064 (1.221)	-0.043 (1.204)	-0.173 (1.212)	-0.033 (1.227)
Birth weight <2500g	N/A	N/A	4.965 (17.552)	11.275 (18.879)	12.547 (17.492)	12.165 (17.418)	12.480 (17.630)
Mother's Pre Natal Care	N/A	N/A	N/A	N/A	21.449 (9.223) ♣	21.136 (9.230) ♣	21.396 (9.215) ♣
Sickness at Birth	N/A	N/A	N/A	N/A	22.218 (13.746) ♫	20.670 (14.521) ♫	22.011 (13.665) ♫
Complications of Delivery	N/A	N/A	N/A	N/A	-3.213 (7.453)	-3.708 (7.354)	-3.258 (7.428)
APGAR Score 1 Min	N/A	N/A	N/A	N/A	N/A	1.217 (1.585)	-0.401 (2.909)
APGAR Score5 Min	N/A	N/A	N/A	1.116 (2.344)	2.729 (2.758)	N/A	3.212 (4.936)
Constant	71.150 (72.849)	141.879 (146.800)	130.237 (150.600)	90.829 (164.101)	67.057 (168.692)	92.886 (168.970)	63.761 (176.313)
Sample Size	626	583	583	532	532	537	532
R Squared	0.24	0.23	0.23	0.25	0.26	0.26	0.26
CORTISOL							
Birth Weight	-0.102 (0.063) ♫	-0.017 (0.071)	-0.043 (0.071)	-0.054 (0.076)	-0.051 (0.078)	-0.049 (0.078)	-0.039 (0.081)
Birth Week	0.032 (0.021) ♫	0.046 (0.023) ♣	0.036 (0.025) ♫	0.043 (0.030) ♫	0.041 (0.030) ♫	0.042 (0.030) ♫	0.038 (0.031)
Birth Height of Body	N/A	-0.025 (0.017) ♫	-0.028 (0.017) €	-0.030 (0.018) €	-0.029 (0.019) ♫	-0.026 (0.019) ♫	-0.028 (0.019) ♫
Birth Head Circumference	N/A	-0.010 (0.026)	-0.009 (0.026)	0.003 (0.027)	0.000 (0.027)	-0.003 (0.027)	-0.002 (0.028)
Mother's Age at Birth	N/A	0.006 (0.017)	0.007 (0.017)	0.009 (0.020)	0.009 (0.020)	0.011 (0.019)	0.008 (0.020)
Birth weight <2500g	N/A	N/A	-0.219 (0.152) ♫	-0.178 (0.181)	-0.196 (0.185)	-0.185 (0.193)	-0.191 (0.190)
Mother's Pre Natal Care	N/A	N/A	N/A	N/A	-0.086 (0.125)	-0.078 (0.123)	-0.083 (0.124)
Sickness at	N/A	N/A	N/A	N/A	0.086	0.110	0.099

Birth					(0.100)	(0.098)	(0.097)
Complications of Delivery	N/A	N/A	N/A	N/A	-0.029 (0.075)	-0.010 (0.076)	-0.024 (0.077)
APGAR Score 1 Min	N/A	N/A	N/A	0.010 (0.028)	N/A	0.016 (0.020)	0.032 (0.037)
APGAR Score5 Min	N/A	N/A	N/A	N/A	0.009 (0.031)	N/A	-0.031 (0.059)
Constant	0.177 (0.715)	0.845 (1.150)	1.412 (1.202)	0.734 (1.524)	0.944 (1.563)	0.803 (1.438)	1.188 (1.648)
Sample Size	365	344	344	312	312	316	312
R Squared	0.35	0.36	0.36	0.36	0.36	0.37	0.37
HEIGHT							
Birth Weight	4.781 (1.188) ♣	4.758 (1.555) ♣	4.551 (1.507) ♣	4.374 (1.479) ♣	4.670 (1.492) ♣	4.748 (1.522) ♣	4.587 (1.506) ♣
Birth Week	-0.970 (0.380) ♣	-1.113 (0.430) ♣	-1.177 (0.448) ♣	-0.746 (0.459) ♫	-0.801 (0.446) €	-0.868 (0.450) €	-0.772 (0.441) €
Birth Height of Body	N/A	0.253 (0.292)	0.229 (0.294)	0.318 (0.287)	0.348 (0.292)	0.302 (0.294)	0.335 (0.289)
Birth Head Circumference	N/A	-0.253 (0.597)	-0.237 (0.598)	-0.144 (0.556)	-0.248 (0.561)	-0.169 (0.567)	-0.222 (0.561)
Mother's Age at Birth	N/A	0.120 (0.170)	0.127 (0.169)	0.308 (0.161) €	0.299 (0.159) €	0.251 (0.165) ♫	0.305 (0.159) €
Birth weight <2500g	N/A	N/A	-1.643 (3.494)	1.292 (3.723)	1.362 (3.663)	1.211 (3.604)	1.326 (3.667)
Mother's Pre Natal Care	N/A	N/A	N/A	N/A	1.020 (1.660)	0.903 (1.691)	0.994 (1.662)
Sickness at Birth	N/A	N/A	N/A	N/A	5.218 (2.216) ♣	4.817 (2.030) ♣	5.116 (2.207) ♣
Complications of Delivery	N/A	N/A	N/A	N/A	-1.279 (1.277)	-1.589 (1.304)	-1.313 (1.283)
APGAR Score 1 Min	N/A	N/A	N/A	N/A	N/A	0.228 (0.313)	-0.230 (0.454)
APGAR Score5 Min	N/A	N/A	N/A	0.445 (0.453)	0.630 (0.418) ♫	N/A	0.907 (0.615) ♫
Constant	181.223 (12.986) ♣	179.915 (22.598) ♣	183.730 (24.215) ♣	151.326 (22.552) ♣	152.463 (22.868) ♣	159.732 (22.819) ♣	150.679 (22.715) ♣
Sample Size	636	594	594	541	541	546	541
R Squared	0.34	0.35	0.35	0.36	0.37	0.37	0.37

Note: Robust standard errors clustered at the individual level in parentheses. Regressions include year of measurement indicators. . ♣, €, ♫ denote significance at 5%, 10%, 20% level respectively.

Table 8: Effect of Kindergarten Teacher Rated Children Behavior on Subsequent Hormone Levels and Anthropometric Measures

Outcome	TESTOSTERONE	CORTISOL	HEIGHT
Teacher Rating of Aggression	1.254 (1.622)	-0.002 (0.030)	-0.114 (0.386)
Teacher Rating of Opposition	-1.910 (3.452)	-0.001 (0.063)	0.435 (0.758)
Teacher Rating of Anxious Behaviour	0.776 (1.211)	-0.004 (0.018)	-0.146 (0.253)
Teacher Rating of Pro-Social Behaviour	0.450 (0.589)	0.004 (0.014)	0.137 (0.119)
Teacher Rating of Inattention	-2.383 (1.621) ♫	-0.025 (0.023)	-0.218 (0.299)
Teacher Rating of Hyperactivity	-0.646 (2.600)	0.075 (0.064)	0.259 (0.660)
F Test of Joint Significance of Teacher Ratings	0.65 [0.6888]	0.81 [0.5604]	0.51 [0.7978]
R Squared	0.21	0.18	0.31
Number of Observations	696	419	726

Note: Robust standard errors clustered at the individual level in parentheses. Regressions include year of measurement indicators. . ♣, €, ♫ denote significance at 5%, 10%, 20% level respectively. Prob > F in square brackets.

Appendix Table 1: First Stage Regressions Explaining Parental Behavior for Testosterone Sample

	Parental Rules	Parental Supervision
Years of Mother Education	-0.032 (0.027)	-0.092 (0.030)♣
Years of Mother Education ^{2}	3.6*10E-4 (1.3*10E-3)	0.029 (0.014)♣
Years of Father Education	0.005 (0.022)	-0.074 (0.024)♣
Years of Father Education ^{2}	-0.004 (0.009)	0.031 (0.010)♣
Family Income	-0.121 (0.063)€	0.113 (0.069)€
Family Structure	-0.039 (0.008)♣	-0.043 (0.008)♣
Sensitization Contact Treatment Group	-0.032 (0.010)♣	-0.047 (0.011)♣
Kindergarten Assessment made Eligible for the Experiment	7.3*10E-3 (0.009)	0.020 (0.009)♣
Bimodal Prevention Treatment Group	-0.016 (0.011)€	-0.022 (0.012)€
Preschool Intervention	0.004 (0.003)♫	0.001 (0.004)
Kindergarten Teacher Rating of Aggression	0.004 (0.001)♣	-0.001 (0.001)
Kindergarten Teacher Rating of Opposition	-0.006 (0.002)♣	-0.000 (0.002)
Kindergarten Teacher Rating of Anxious Behaviour	0.001 (0.001)	0.004 (0.001)♣
Kindergarten Teacher Rating of Pro-Social Behaviour	-0.001 (0.000)♣	0.001 (0.000)♣
Kindergarten Teacher Rating of Inattention	0.002 (0.001)♣	-0.002 (0.001)€
Kindergarten Teacher Rating of Hyperactivity	-0.001 (0.002)	0.001 (0.002)
Constant	0.899 (0.022)♣	0.741 (0.024) ♣
Obs	715	715
R-squared	0.14	0.14

Note: Standard errors clustered at the individual level in parentheses. Regressions include year of measurement indicators and other factors related to hormone levels and time of readings. ♣, €, ♫ denote significance at 5%, 10%, 20% level respectively.