

Hormones and aggression in childhood and adolescence

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Abstract

This review is a survey on recent psychobiosocial studies on association between hormones and aggression/violence in children and adolescents, with a special focus on puberty, given the rapid changes in both hormones and behavior occurring during that developmental period. Since it cannot be assumed that all readers have much background knowledge, it inevitably begins with some comments about the concept and multifaceted nature of aggression, as well as with a brief reminding about hormone candidates to be linked to aggression during human development. Then, we finish off with the status of its knowledge in today's science, tackling in a systematic way with the main data published, hormone by hormone. The origin of the gender-based differences in aggression must lie in neuroendocrinological events occurring during prenatal life or early in postnatal life. A complex and indirect effect of testosterone on aggression is proposed. A low HPA axis activity seems associated with chronic aggressive and antisocial behaviors. It is also suggested that early adrenal androgens contribute to the onset and maintenance of persistent violent and antisocial behavior, and that it begins early in life and persists into adulthood, at least in young boys. There are also some studies suggesting an association between aggression and some pituitary hormones in children, even if present data are still far from being consistent. The hormone-aggression link during development thus is not consistently reported. There can be an indirect relation in three ways: hormones can be involved in the development of aggression as a cause, as a consequence, or even as a mediator. Psychosocial factors may influence the causation and progression of violence in children through hormonal action.

Keywords: Hormones; Aggression; Violence; Children; Adolescents

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1. Introduction

One of the biggest hurdles in the study of aggression and violence is the lack of a consensus on their definitions (Kavoussi, Armstead, & Coccaro, 1997). Although usage of both terms is recognized, there is much disagreement about their precise meaning and causes. They are used so broadly that it becomes virtually impossible to formulate a single and comprehensive definition. Under the general rubric of aggression or violence an *omnibus* term with a certain amount of ambiguity is subsumed, which consists of a large variety of meanings, related to several qualitatively distinct subtypes of behavior heterogeneous in nature. Even if they may be similar in appearance, each one is related to different factors, has separate genetic and neural control mechanisms, and is instigated by different external circumstances (see Andreu & Ramirez, in press; Ramírez, 1996, 1998, among others). Some authors tend to conceive aggression as a behavior based on biology, and violence as a social construction. Erich Fromm sees them as two antithetic biological concepts. Aggression is seen as a biological behavior, natural to all the animal world, adaptive, intentional and propositional, not always necessarily negative, but sometimes justifiable and beneficial, needed for the survival of the individual and the species, and always under the limits of the self-control. Violence, on the other hand, is considered a biological alteration, privacy of humans, malign, pathological and destructive, and consequently absolutely undesirable and reprobable, that should always be controlled and replaced by an alternative behavior (Gómez Jarabo, 1999). Another distinction is presented by Archer (1994, 2000), who understands by aggression the occurrence and frequency of acts, with no reference to their consequences; and by violence, solely the damaging consequences of aggressive acts. Many others try to include both within a *continuum*, rating violence as an extreme, harmful aggression, defined broadly like ‘the abusive or unjust exercise of power’ (Rivara, 2002) or as ‘hypertrophic aggression’ (Sanmartin, 2002). The conceptual differences between aggression and violence thus are still to be clarified (Ramírez, 2000a, 2000b). Considering this conceptual confusion in this paper, terms such as violence, aggression, or aggressivity will be used synonymously, for pragmatic and operational reasons.

This article will stress the importance of biology in the study of violence during developmental stages, but it does not mean that we conceptualize biology as a domain isolated from other ones. We are not slaves to our genes, nor slaves to our environment. Neglecting psychosocial factors in the causes of aggressive behavior would be as misleading as to focus on the individual’s biology without recognizing its inevitable interaction with other factors, such as cognition, emotion, or social context. A close knit community with stable families and effective policing, for example, may reduce levels of violence in antisocial prone adolescents. And, on the contrary, neglected and abused children are more prone toward antisocial behavior. A developmental perspective of aggression thus is based on the assumption that aggressive behavior is multidetermined and dynamic over the life span, and a product of a complex continuous interaction of the multiple psycho-bio-social changes. This holistic approach promotes a much needed focus on the plasticity of the child (Pribram & Ramírez, 1980, 1981, 1995; Stoff & Cairns, 1996). The social environment thus is linked to human behavior through our biology.

The most important general insight of recent years has been perhaps the recognition that life experience can shape brain chemistry in significant ways, and that experience and neurophysi-

ology form a seamless web. The neurobiological plasticity expressed in the functional organization of the nervous system is open to the input coming from the personal experiences, which can result in large, long-lasting and consequential change. Stressors, for example, appear to affect hormone concentrations in humans, even if these effects have received only minimal research attention. Integration of biological research with social-scientific studies thus can add to our understanding of how life experience influence interactions that involve or lead up to violence (for a longer discussion of this topic, see in this same Journal: [Book, Starzyk, & Quinsey, 2001](#)).

One of the many biological component systems that affect aggression, and with a very promising future given its extraordinary recent advances, is its relation to chemistry -neurotransmitters, neuromodulators, and hormones. Pharmacological and genetic studies have dramatically expanded the list of neurotransmitters, hormones, cytokines, enzymes, growth factors, and signaling molecules that influence aggression ([Nelson & Chiavegatto, 2001](#)). This neurochemical/neuropeptide/neuroendocrine ‘orchestra,’ as it has been elegantly described by [Eichelman and Hartwig \(1996\)](#), is played through many anatomical sites within an organism genetically prepared to function aggressively.

Although most of the experimental psychoendocrinological research is done in other animal species, given the many technical and ethical limitations and obstacles encountered in the direct investigation of aggressive behavior in our species ([Ramírez, 2000b; Ramírez & Brain, 1985](#)), human aggression is unique. We may have ‘inherited the biological basis’ for aggression in common with other species, but we have a unique capability for intelligence and learning, and this applies to all kinds of behavior, including the aggressive one. Humans may show overt aggression in the same situations as other animals, such as in competition over food, mates or dominance, but usually our own intelligence plus the social rules established by our culture allow a higher flexibility, preventing us from being violent in those circumstances. The motives for violence in humans are clearly more complex, having to do with self-image, reputation, and perceptions of ‘psychological’ harm ([Toch, 1984](#)).

This review is a systematic search of hormonal correlates to aggressive behavior in human infancy and adolescence. The main reason for much delimitation is because developmental processes may provide the common ground for understanding the processes of socio-biological integration, and reproductive transitions provide excellent periods of development in which to examine hormone-behavior relations. Age is as important as neglected individual-differences variable in aggression research. Only a handful of psychological studies had examined age differences ([O’Connor, Archer, & Wu, 2001](#)), until the recent ISRA World Conference on Aggression (Montreal, July 28–31, 2002), which has been focused precisely to this very topic: the developmental origins of aggressive behavior.

2. Method

2.1. Data sources

SCIENCEDIRECT and MEDLINE-derived Online reviews of bibliographies were systematically searched for articles published during the last 15 years related to hormones and

aggression in children and adolescents. Some pivotal earlier publications were also obtained and included in the review. Reference lists from selected and review articles were also examined.

2.2. Study and data selection

The abstracted data of each study will be presented, hormone by hormone, with a short discussion on their main findings and suggestions. A summary combining their results in relation to age, sex, and the type of aggression is also given.

3. Hormones associated to human development

There are some dozen hormones of particular importance in the control of human development: thyroxin (T_3) from thyroids; cortisol (CORT), and adrenal androgens (dehydroepiandrosterone [DHEA], dehydroepiandrosterone sulphate [DHEAS], and androstenedione [$\Delta 4$ -A]) from adrenal cortex; testosterone (T) from testes; estrogens (E) from ovaries; insulin from pancreas; and a series from pituitary: growth hormone (GH), thyroid-stimulating hormone (TSH), adrenocorticotrophin (ACTH), gonadotrophins (luteinizing hormone [LH] and follicle stimulating hormone [FSH]), prolactine, and vasopresine (ADH).

During puberty, dramatic changes occur in endocrine physiology, and specially in the hypothalamic–pituitary–gonadal (HPG) axis: the gonads secrete hormones in amounts sufficient to cause accelerated growth of the genital organs and the conspicuous appearance of secondary sexual characters. The most obvious changes are brought about by the rise of the secretor activity of gonadal and adrenal sexual hormones in interaction. Its time is usually assessed from the appearance of outward signs attributable to the action of hormones. But, there is a first event, immediately previous to the morphological changes: an increased secretion of the hypothalamic LHRH, followed by a gradually increase of the pituitary gonadotrophic hormones: FSH and LH. The peripheral increase of T and E with their characteristic events follows. Girls tend to pass through various stages of puberty at an earlier age than boys do (18–24 months earlier), beginning at approximately ages 9 for girls and 10 for boys. In males, in all except the 10-year-olds, there is a sharp rise in T across a 1-year period; in the 14-year-olds levels begin a *plateau*. In female, T shows little change in the 9-year-olds across 1 year; there is a rise in T in the 10- and 11-year-olds; and the changes in T becomes more variable in the 12-, 13-, and 14-year-olds because of menstrual cycle variations (Tanner, 1978).

For both boys and girls there are, however, wide individual differences in T in the same chronological age cohort during early adolescence. Many years ago, Schoenfeld (1943) observed that 4% of 10-year-old boys already showed pubescent signs, but 6% of 14-year-old boys showed none yet. And a similar study in girls (van't Land & de Hass, 1957) showed that, whereas 12% of 12-year-old girls had started to menstruate, another 12% of 15-year-old ones had still to experience menarche, with a range of 11–16.3 years of age (average: 13.5). These individual differences in hormonal levels at the same chronological age pose important

theoretical and methodological considerations. Genetic factors and timing of puberty as well as other environmental (climate, light, temperature, etc.), socioeconomical (nutrition, urban vs. rural, stress, etc.) and experiential factors (experiences of success and failure are differently reflected in competencies and adjustment) contribute to these individual differences in hormone levels at puberty. The methodological approach, used to control for individual differences in timing, includes age as a covariate in analyses when relations among hormones and behavior are examined (Nottelmann et al., 1987; Susman, Dorn, & Chrousos, 1991; Susman et al., 1987, 1985).

4. Hormones associated to aggression during human development

Only a few of these above mentioned hormones have been found to have some relationship with aggression during human development: gonadal and adrenal androgens, and perhaps and in minor degree also gonadotrophins, prolactine, and estrogens. Let us here attempt to review what has been reported in the scientific literature.

4.1. Gonadal hormones

4.1.1. Brief information about hormones during human development

The main androgen or male sex hormone is T. Its secretion rises in three periods of life: an early first peak in the fetus from about 11 postmenstrual weeks probably till birth, time when it falls rapidly (during this time it causes differentiation of the external genitalia and of the hypothalamus in an male type); a second peak about 2 months after birth lasting a few months (its function is still unknown); and a third very large peak at puberty (growth, maturation of genital organs, and appearance of secondary sex characters), first becoming apparent between 10 and 12 years of age, requiring a joint action with GH for its full effect on the adolescent growth spur. In humans, androgens have been described, albeit inconsistently, to play a role in the regulation of sexuality, aggression, cognition, emotion, and personality.

The female sex hormone is E. It begins to be secreted by the ovaries very early in life, long before puberty, at low levels during childhood, increasing sharply at puberty, and fluctuating regularly thereafter with the phase of the menstrual cycle. Contrary to what has been observed in the testes (a prenatal peak of T), there is no evidence of any hormonal action by the ovary of the fetus. They also have an important role on maturation of genital organs and appearance of secondary sex characters.

4.1.2. Report of the studies

Given the dramatic changes that occur in endocrine physiology at puberty, it is not surprising that increases in *testosterone* are hypothesized to be related to increases in aggression. This explains why T has been the most investigated hormone in its research. The wealth of evidence supporting the ability of T to facilitate aggressive behavior in a broad number of mammal species has led to wonder about its potential role in human aggression, expecting at least a positive correlation between both variables. These studies, however,

yielded equivocal results. Even if elevated circulating T levels have been reported in some antisocial youth, the T-aggression link is not consistently reported across studies in children and adolescents. Archer (1991) conducted three meta-analyses, including only five to six studies, and found a weak, positive relationship between T and aggression. Another recent meta-analysis based on 45 independent studies with 54 independent effect sizes re-examined this relationship (Book et al., 2001), and found a range of correlations from $-.28$ to $.71$. This mean weighted correlation ($r=.14$) corroborates Archer's finding of a weak positive relationship.

The first study to show the activational influences of circulating T and aggression in adolescents was done with 15–17-year-old boys. Aggression in response to hypothetical provocation was measured by self-reports. A direct and longitudinal effect of T on aggression was shown: a higher T level led to an increased readiness to respond to provocation, but T had no direct effect on unprovoked aggression (Olweus, Mattson, Schalling, & Low, 1980, 1988).

A second study designed to analyze puberty-related psycho-biological processes in 10–14-year-old girls examined, among other aspects, whether hormonal pubescent changes were more likely to be associated with negative affect, measuring self-reports of emotional states and a hormonal assessment in serum of LH, FSH, E, T, DHEAS, and CORT. Aggressive affect was negatively associated with DHEAS, but no relations with T were found (Brooks-Gunn & Warren, 1989; Warren & Brooks-Gunn, 1989).

A third study was a NIMH-NICHD collaboration that analyzed, among other aspects, the relations between serum levels of adrenal and gonadal hormones (LH, FSH, T, E, DHEA, DHEAS, $\Delta 4$ -A, and CORT) and mother-reported aggressive attributes in 56 boys and 52 girls, age 9–14 years. The general pattern of findings in boys only, but not in girls, was a higher level of delinquent behavior related to lower DHEAS and T/E₂ ratio, and a higher rebellious attitude related to higher levels of LH, lower levels of FSH, and higher levels of DHEA (Susman et al., 1987). Another aspect focused on was the expression of anger of young adolescents while interacting with their parents during problem-solving tasks. The expression of anger was related to a consistent relationships with $\Delta 4$ -A, which will be commented later, and to higher levels of hormones that increase at puberty, especially with regard to E in girls. The interpretation would be that young adolescent girls may be very sensitive to changes in E level, as is also hypothesized during the menopausal period (Inoff-Germain et al., 1988). From the same sample, adjustment problems were also associated with a hormone profile similar to that described for aggressive attributes: higher $\Delta 4$ -A and lower T levels or a lower T/E ratio (Nottelmann et al., 1987).

T levels could be a signal of social success rather than of physical aggression, as suggested first by Sapolsky (1991), examining experiential influences on T and CORT secretion in male baboons in natural environment, concluded that aggressiveness and social status were associated with higher T, while subordinates were with lower levels, and more recently in human adolescents by Schaal, Tremblay, Soussignan, and Susman (1996) in a study that analyzed the association of male pubescent T with social dominance and physical aggression. Boys perceived as socially dominant by unfamiliar peers, from age 6 to 12, were found to have concurrently higher levels of T at age 13 than boys perceived as less socially dominant.

In contrast, boys who had a history of high physical aggression, during the same age range, had lower T levels at age 13 compared with boys with no such a history. T levels therefore were positively associated with social success rather than with physical aggression. High T levels in adolescent boys may thus be regarded as a marker of social success in a given context, rather than of social maladjustment as suggested in previous studies. Adolescents may try to gain social status through dominance and leadership, using aggressive and assertive methods. If successful, T is expected to increase, and if unsuccessful, T would decrease because the negative status associated with failure.

Another question posed was whether chronically aggressive and impulsive behavior affects hormone concentrations in adolescents during their developmental transitions. Hormone determinations in serum and saliva and several self-report tests were applied to Caucasian rural adolescents, either experiencing transition to puberty (boys: mean age=12.7 and girls: mean age=11.9) and experiencing transition to pregnancy (girls: mean age=17.4). An analysis of hormone-behavior connections showed concurrent relationships, indicating individual differences in hormone concentration as possible influences on behaviors. Experience and behavior may also be implicated on hormone concentrations, changing the endocrine milieu. Subtle variations in timing of puberty related to experience may predispose some adolescents to aggression and violent behavior, and to other wrong coping strategies, such as heavy drinking. Also late maturing males experience difficulty in attaining dominance and peer popularity because their immature physical status (Susman, Worrall, Murowchick, Frobose, & Schwab, 1996).

A cross-cultural comparison within American white male adolescents (Cohen, Nisbett, Bowdle, & Schwarz, 1996) also revealed that norms characteristic of a 'culture of honor' manifested themselves not only in the cognitions, emotions, and behaviors, but also in the physiological reactions of the subjects. Whereas Northerners were relatively unaffected when insulted, Southerners were more upset (as shown by a rise in CORT levels), and more physiologically primed for aggression (as shown by a rise in T levels). BLOOD?

In another research, salivary T and CORT levels were also measured in 29 violent delinquents and 36 U.S. college students of a similar age. The delinquents had higher T levels but did not differ regarding CORT (Banks & Dabbs, 1996).

The first study to causally relate the administration of physiological doses of sex steroids to changes in aggressive behaviors in adolescents, focused on the role of sex steroids in the development of aggressive behaviors in hypogonadal adolescents (Finkelstein et al., 1997). Depo-testosterone (to 35 boys) or conjugated estrogens (to 14 girls) was administered in 3-month blocks alternating with placebo at three dose levels approximating early, middle and late pubertal amounts, and the Olweus Multifaceted Aggression Inventory was applied after each period. Results demonstrated significant hormonal effects on physical aggressive behaviors and aggressive impulses, but not on verbal aggressive behaviors nor on aggressive inhibitions in both boys and girls. The fact that physical aggression was affected whereas the verbal was not, could be explained at least partially by the changes in musculature also observed.

Until this last decade, however, this eventual link between serum T and aggression has not been investigated in younger children, before the time of puberty. Interactions between social

behavior of preschool 5-year-old children (with a special focus on aggressive behavior) and T were analyzed at the Basque Country University, in Spain, measuring their saliva hormonal levels. A positive relationship between T and aggression was found only in boys, but not in girls, in the context of 'social interactions' (playful aggression: giving and receiving threat/aggression, defense/avoidance), but not in the context of play (Ahedo, Cardas, Aizpiroz, Brain, & Sánchez-Martín, 2002; Sanchez-Martin et al., 2000). These findings confirm also in small boys the suggestion that circulating T play an important role in all social behavior and not only in aggression; and that the development of sex-typed behavioral differences is already expressed on early postnatal life.

In preschool children in a nursery situation, Corrine Hutt, at the University of Reading, has found that boys were overall more aggressive than girls; most aggressive acts tended to involve boys fighting with other boys. She argued that this sex difference in aggression was a consequence of perinatal hormone exposure in boys. But, measured serum T, sex hormone binding globulin (SHBG), DHEA, and DHEAS in 18 highly aggressive CD prepubertal boys, ages 4–10, hospitalized for violent or unmanageable behavior, their comparison with a group of age and race matched controls from the same demographic area, screened negative for aggressive problems. No significant differences were found between aggressive and non-aggressive children for T, SHBG, DHEA, DHEAS, or ratios of combinations of these variables (Constantino et al., 1993).

Another study, that Melissa Hines's group is still carrying out at the City University of London on 200 children aged three and a half, suggests some relation between T levels and kinds of play. Although it does not directly focus on aggression, but rather on gender role behavior, it is interesting enough to be mentioned here because it is focused more toward an organizational prenatal influence of hormones rather than the activational effects analyzed in the previously mentioned studies.¹ A 'masculine–feminine' score was compiled: the kinds of questions were whether they played with dolls or trucks, whether most of their friends were boys or girls, and whether they liked sport. Researchers noted a clear link between high T levels in a mother's womb and masculine behavior in girls. Girls exposed to higher doses of the male hormone were more likely to prefer toy cars to dolls, rough-and-tumble games to dressing up, and mud pies to tea parties. Conversely, the findings showed an association between low T in pregnancy and daughters who display typically 'girlie' behavior, such as dressing up in frocks and stealing their mother's make-up. However, social factors were also involved: tomboys tended to have older brothers and parents whose behavior was highly masculine. On the contrary, mothers' hormonal levels did not appear to have the same effect on boys, possibly because, since T levels are already high in unborn boys, small differences in the womb would have little extra effect. Boys may also be under greater social pressure to behave as boys should. Pregnant women with higher than average levels of the male hormone T in their blood thus have a greater chance of giving birth to a tomboy. Consequently, both

¹ The hormonal influence on behavior can be at least of two kinds: (a) activational, stemming from contemporaneous effects, or (b) organizational, referred to structural changes occurred during pre- or perinatal development; this early hormonal state would sensitize or desensitize the individual to hormonal circulation in adulthood (Leshner, 1978).

Hutt's and Hines' findings may suggest that the exposure to higher levels of androgens around 3 months in utero can be at least partially responsible for increased rough and tumble play observed in boys and on girls with congenital adrenal hyperplasia (CAH), characterized by behavioral masculinization (see Berenbaum's research in the next section).

A similar approach was followed by Frank Sulloway's research concerning birth order and rebelliousness. It has been suggested that rebelliousness among latter born children could be explained by higher T levels in aging mothers, rather than as an adaptation for sibling competition, even if the two explanations are not mutually exclusive. These higher T levels with age in women would not be in absolute terms, because an absolute increase seems oppositional to the finding of less T exposure in utero in latter born sons, but more accurately just higher T levels relative to decreasing E levels.

Finally, van Goozen, Matthys, Cohen-Kettenis, Thijssen, et al. (1998) in Utrecht studied the relationship between androgens and aggression in prepubescent boys who were diagnosed as suffering from severe aggression and antisocial conduct disorders (CD), measuring their T, $\Delta 4$ -A, and DHEAS levels. CD boys had significantly higher levels of DHEAS and marginally significantly higher levels of $\Delta 4$ -A; moreover, DHEAS levels were significantly positively correlated with the intensity of aggression. But there were no differences in T. These findings question the usefulness of T as biological marker for aggressivity in early childhood.

Saliva levels of T were also compared with behavioral measures among 45 boys aged 5–11 years, 25 from a psychiatric group with disruptive behavior disorders and 20 from a normal control group. In the overall sample, T was associated with withdrawal and aggression (especially among older boys) and low social involvement in activities (especially among younger boys), contrary to van Goozen's conclusions. T was also higher in the psychiatric than in the normal group, but only among the older boys, aged 9–11. Whether girls would show similar relations, and whether T levels in young children predict later development and behavior, remains to be determined (Chancea, Brown, Dabbs, & Caseya, 2000).

4.1.3. Summary and comments

Contrary to the consistent findings first reported in older adolescents (Olweus et al., 1980, 1988), the above mentioned studies and meta-analysis, as well as in other similar ones at pubertal and at a younger age (Granger, Weisz, McCracken, Kauneckism, & Ikeda, 1994; Scerbo & Kolko, 1994), yielded rather equivocal results, with a lack of links and less conclusive association between androgens and aggression. This may reflect the developmental maturational states of the different studies: T would reach levels consistent with the activational influences of hormones only at the late age stages. It may also indicate that T levels in adults are a consequence and not a cause of aggression (Brain & Susman, 1996).

In practice, the investigation of eventual correlations between gonadal androgens and aggression in prepuberal children did not start until last decade. Studies with 5-year-old children of both sexes found a relationship between T level in saliva and playful aggression in boys, but not in girls, suggesting that the presence of T in males may play some role in social behavior at early age. Several others, however, have found a significant higher T level in saliva of violent and CD boys only at 9/11 years of age, but not in earlier age. Another interesting finding is that pregnant mothers with high level of androgens in blood may be

responsible, at least partially, of having behavioral masculinized daughters, as it has been already observed at 3.5 years of age; but apparently sons of these mothers did not show any specific behavioral change. This possible causal behavioral effect of the gonadal hormones has also been observed in hypogonadal adolescents of both sexes: the administration of sexual steroids during 3 months produced an increase in physical aggression, but not in verbal aggression.

In adolescents of different age, several reports mention a concurrent relationship between higher levels of T in blood and in saliva and provoked aggression [but not with unprovoked one] in boys, but not in girls. Puberal girls, however, show a positive relationship between higher levels of E and anger.

T could have a complex and indirect facilitatory effect on aggression being sensitive to psycho-social environmental influences. And E would also facilitate conflicts in both men and women, mediatizing the influence of the T (Niehoff, 1999; Susman, Worrall et al., 1996). The findings of social stress and aggression accompanying changes in T and CORT [antisocial behavior is associated with lower gonadal steroids and higher adrenal androgen concentrations in adolescent] support the hypothesis that social experiences and contexts of development affect hormonal levels. Therefore, a two-way relationship between aggression and gonadal hormones might be suggested. Gonadal hormones, besides being only one of the multiple processes -biological, social, and cognitive, to influence aggression in children and adolescents, might also be a signal of aggression, or even more precisely perhaps, of social success.

4.2. Adrenal hormones

4.2.1. Brief information about hormones during human development

The major hormonal product of the limbic hypothalamus–pituitary–adrenal (HPA) system in humans is CORT, a glucocorticoid secreted in a pulsatile manner by adrenal cortex, and controlled by ACTH from pituitary. It is secreted during fetal life and childhood at the same rate as in adults, proportionally speaking to body size. It has an antiinflammatory, an antistress, and an antigrowth action (this last effect may be through its inhibition of somatomedin actions and perhaps the secretion of GH itself; Tanner, 1962).

The adrenal gland also secretes some closely related substances, called androgens because of their functions in most respects similar to T: namely DHEA, DHEAS, and $\Delta 4$ -A. They are largely responsible for some of the puberal changes and for the maintaining of some secondary sex characters. This is specially true in girls; in boys, all these things are done more effectively by T. Their rate of secretion is very low during childhood, but a marked increase takes place at puberty, to somewhat higher levels in boys than in girls. Thereafter, the amount declines and by ages 60–70 returns to prepuberal values.

4.2.2. Report of the studies

Low salivary CORT levels have been associated with persistence and early onset of aggression in normal school-aged boys. They triple the number of aggressive symptoms, and were named as most aggressive by peers three times as often as boys who had higher CORT

concentrations in saliva at either sampling time (McBurnett, Lahey, Rathouz, & Loeber, 2000). This same negative correlation between concentration of CORT in saliva and CD, has also been reported in preadolescent CD boys: children (9.6 years as average) with CD had lower levels of CORT in saliva than those without CD (McBurnett, Lahey, Capasso, & Loeber, 1996; Vaniukok et al., 1993).

Virkkunen (1985) also found that those habitually violent adult males who showed aggressive CD during childhood, excreted only about half the amount of free CORT compared to others. This suggests that CORT in childhood would be a risk factor for chronic aggression: children with very low levels of CORT were almost always highly aggressive. Contrary to the hypothesized inverse relationship between CORT secretion and aggressive behavior suggested by these previous results measuring saliva CORT levels, no significant difference was found between 7- and 11-year-old aggressive and nonaggressive boys with Attention Deficit Hyperactivity Disorder (ADHD) whose hormonal levels were measured in plasma (Schulz, Halperin, Newcorn, Sharma, & Gabriel, 1997).

On the contrary, under stressful circumstances, higher levels of adrenal androgens and lower E are secreted suggesting that stressful experiences may play a role in the development of the gonadal axis. Consequently, given that the best known HPA hormone involved in modulating adaptation to stress is CORT, in a psychological stress paradigm saliva CORT concentrations were positively related to conduct problems, but only when boys showed 'very high levels' of conduct problems over time (Lahey, McBurnett, Raine, Stouthamer-Loeber, & Loeber, 2002). High levels of CORT and larger increases of it from morning to afternoon have also been found in children with anxiety and depression (McBurnett et al., 1991), as well as in children with more immature social skills, more emotionally negative and with less self-control (Dettling, Gunnar, & Donzella, 1999; Dettling, Parker, Lane, Sebanc, & Gunnar, 2000). A higher adrenal androgen concentration thus would be an index of higher stress (Nottelmann et al., 1987; Sapolsky, 1991).

In an attempt to prove the hypothesis that social experiences affect hormonal levels, saliva samples of T and CORT were taken in disruptive children, aged 7–14 years (Scarpa & Kolko, 1996). Moderate positive relationships between T and staff-rated aggression, and between CORT and parent-rated aggressive responses to provocation, as if they would 'internalize' their abused experience, appeared in all disruptive children, regardless of age. A significant negative relationship was also found between CORT and staff-rated inattention/overactivity (Scerbo & Kolko, 1994).

Other studies in antisocial youth failed to find any link with CORT levels, though. In an above mentioned research, measuring salivary T and CORT levels in 36 college students and 29 delinquent participants of a similar age, T levels were higher in violent delinquent adolescents than in normal college students; but CORT levels did not differ in any of the samples (Banks & Dabbs, 1996; Kruesi et al., 1989; Targum et al., 1990).

Contextual and individual differences also play an important role in hormonal secretion. An already mentioned study with American male adolescents stressed how cultural differences could influence physiological reactions to insults. Southerners, heavily influenced by a characteristic 'culture of honor,' were more upset, as shown by a rise in CORT levels, and

more physiologically primed for aggression, as shown by a rise in T levels, whereas Northerners were relatively unaffected (Cohen et al., 1996).

Another two studies of Gunnar's group at Minneapolis considered the relations between temperament, social competence, and levels of a stress-sensitive hormone CORT in preschoolers, from birth to approximately 5 years of age. In both studies, salivary CORT was sampled daily for the initial weeks of school year (Group Formation period) and for several weeks later in the year (Familiar Group period). For each child, two measures of CORT activity were examined (separately for each period) based on the distribution of CORT levels across days: (a) median CORT (50th percentile) and (b) CORT reactivity (the difference between the 75th and 50th percentile). Median CORT was modestly stable across periods, but CORT reactivity was not. Children who showed high CORT reactivity (75th minus 50th percentile ≥ 0.10 $\mu\text{g/dl}$) during the Group Formation period but low-to-normal CORT reactivity during the Familiar Group period were outgoing, competent, and well liked by their peers. In contrast, children who changed from low/normal to high CORT reactivity and those who maintained high CORT reactivity from the Group Formation to Familiar Group period were affectively negative and solitary. Children who showed high median CORT during the Familiar Group period or over both periods scored lower on a measure of attentional and inhibitory control. Together, these accumulated findings suggest that relations among temperament, social competence, and neuroendocrine reactivity reflect both individual and contextual differences: children with negative emotional temperaments may be most likely to exhibit elevations in CORT under conditions of less than optimal care, whereas young children under neglectful and abusive care often evidenced reduced rather than increased CORT levels (Gunnar & Donzella, 2002; Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997).

Large individual differences were also remarked in another study by van Goozen, Matthys, Cohen-Kettenis, Gispen-de Wied, et al. (1998) with oppositional defiant disorder (ODD) prepubertal boys, finding that CORT levels were overall lower during stress (provocation and frustration).

Let us finally just mention a new promising approach, exploring associations among T and CORT and children's family relations and behavioral development. This ongoing study at Penn State University involves 400 families. Although still in its preliminary stages, it has already revealed that parents' and children's CORT levels may be linked to parenting behaviors in ways that in turn affect social and emotional development (Granger, unpublished communication; see also Kavoussi et al., 1997). A deeper knowledge of both hormones could hopefully give some light on the linkages between biology, behavior, and environments within the context of the family.

Aggression and antisocial behavior have been also found associated with higher *adrenal androgen* concentrations in adolescents. In the NIMH-NICHD collaboration already mentioned in the previous section on gonadal hormones, aggression attributes were examined in relation to a variety of endocrine changes at puberty. Adjustment problems were also associated with higher $\Delta 4\text{-A}$ and relatively lower sex steroid levels, a profile characteristic of later maturation (Nottelmann et al., 1987) and of response to stress. This may suggest that some individuals reflect a predisposition to heightened biological reactivity to environmental

challenges, which undoubtedly are plentiful during adolescence (Susman, Worrall et al., 1996).

Another aspect of the same research was the relation between endocrine changes and the expression of anger of young adolescents interacting with their parents during problem-solving tasks. For boys, the expression of anger was associated with higher levels of DHEA and lower levels of DHEAS. For girls, the expression of anger was related to higher levels of $\Delta 4$ -A. In brief, levels of hormones that increase at puberty were associated with adolescent expression of anger while interacting with their parents (Inoff-Germain et al., 1988).

A different study focused among other aspects on whether hormonal pubescent changes were more likely to be associated with negative affect. It included a hormonal assessment of LH, FSH, E, T, DHEAS and CORT on 10–14-year-old girls. Results showed an association of aggressive affect with DHEAS, but no relations with T were found (Brooks-Gunn & Warren, 1989; Warren & Brooks-Gunn, 1989).

The relationship between different degrees of aggressiveness and neurotransmitter-neuroendocrine responses to stress has also been analyzed in 30 male peripuberal adolescents. Plasma concentrations of norepinephrine (NE), epinephrine (EPI), ACTH, CORT, GH, PRL, and T were measured immediately before the beginning of some psychologically stressful tests and at their end 30 min later. A high-normal aggressiveness was associated with significantly higher basal concentrations of NE, ACTH, PRL, and T and with a significant increase of GH responses to the stressful stimuli (Gerra et al., 1998).

Berenbaum's group has published two interesting papers about the contribution of early androgens to variability in human aggression. One of them reported their research on CAH girls, who were administered CORT in the prenatal and early postnatal periods. The assessment of aggression by the Multidimensional Personality Questionnaire (MPQ) and parents rating, showed that females with CAH had higher aggression than control females, although the difference was significant only in adolescents and adults (Berenbaum & Resnick, 1997). In a second more recent study, it was observed that females with CAH due to 21-hydroxylase deficiency were masculinized and defeminized: they played more with boys' toys, were more likely to use aggression when provoked, and showed less interest in infants than normal ones. This sex-atypical behavior was significantly associated with degree of inferred prenatal, but not postnatal, androgen excess. This finding supports the idea that behavioral masculinization in girls with CAH results from high levels of androgens during fetal development and not in postnatal life. The fact that aggression was not consistently associated with indicators of prenatal or postnatal androgen excess, probably was due to the lack of reliability in its measurement (Berenbaum, 1999; Berenbaum, Duck, & Bryk, 2000).

van Goozen's team (2000) has also focused their research on the relationship between androgens and aggression in children with antisocial behavior. One study analyzed prepuberal boys who were diagnosed as suffering from severe aggression and antisocial behavior. CD boys had significantly higher levels of DHEAS and marginally significantly higher levels of $\Delta 4$ -A; but there were no differences in T. Moreover, DHEAS levels showed significantly positive correlation with the intensity of aggression. This suggests that adrenal androgen functioning plays an important role in the onset and maintenance of aggression in young boys. Another study, in 24 children with ODD, showed a specifically elevated adrenal

androgen (DHEAS). And similar results were observed in CD boys under 14 years: they had higher circulating levels of DHEAS correlated with 'disruptive behavior,' and of ACTH with restless-impulsive ratings (Dmitrieva, Oades, Hauffa, & Eggers, 2001). It is speculated that the mechanism could be a shift in balance of ACTH-beta-endorphin functioning in the hypothalamic–pituitary–adrenal axis due to early stress or genetic factors (van Goozen et al., 2000).

4.2.3. Summary and comments

Some above mentioned studies in normal (Inoff-Germain et al., 1988; Nottelmann et al., 1987; Susman et al., 1987) and abnormal (van Goozen, Matthys, Cohen-Kettenis, Thijssen et al., 1998, 2000) children of both sexes showed consistent relations between aggression and other negative attributes [anger, antisocial behavior, delinquency, rebelliousness, CD] with higher levels of hormones of adrenal origin, such as DHEA, DHEAS and $\Delta 4$ -A. One of these papers, however, related the expression of anger with lower serum levels of DHEAS in boys (Inoff-Germain et al., 1988). This association has been described not only in adolescents (15–17 years of age) but also in preadolescents (5–11 years of age). This suggests that early adrenal androgens contribute to the onset and maintenance of human aggression. Moreover, the consistent relationships of anger with $\Delta 4$ -A, a major source of androgens in females, indicate that androgens of adrenal origin, as opposed to gonadal origin, may play a role in females aggression.

The hypothesis that social experiences affect the function of both the HPG and HPA axes has been already suggested, after observing that antisocial behavior is associated with lower gonadal steroids and higher adrenal androgen concentrations in adolescent. Psychosocial environmental stressful experiences can affect HPA hormones, involved in modulating adaptation to stress, and in this way they may exert a major impact on gonadal hormonal excretion, playing a final role in aggression. This influence on the hormonal level shows individual and contextual differences (i.e., temperament and family), as it has already been observed in preadolescents (9–11 years of age).

Imbalances in the adrenal axes thus may have neurotropic repercussions in development: stress and aggression in adolescents may suppress the gonadal axis leading to alterations in timing of puberty, whereas social success during adolescence may have the opposite effect, leading to higher levels of gonadal hormones (Susman, Worrall et al., 1996).

The typical adrenocortical response to stress is a higher secretion of CORT, which in turn would moderate the T-aggression relationship. On the contrary, a low HPA axis activity seems associated with chronic aggressive and antisocial behaviors, that begin early in life (it has been found to be present already in 9.6 years old boys) and persist into adulthood. Presence in children of low CORT levels in saliva might be interpreted as an eventual risk factor for chronic aggression in adulthood. This correlation with severe and persistent aggression has been reported analyzing CORT levels in male children and adolescents, normal and CD ones. In adulthood CORT surges were also correlated negatively with evocations containing defensive elements and with rage.

In summary, there is an important, although indirect, role for HPA axis on the onset and maintenance, of persistent violent and antisocial behavior in young boys, and vice versa.

4.3. Pituitary hormones

4.3.1. Brief information about hormones during human development

The pituitary gland secretes two hormones, known as gonadotrophins, within the HPG axis: LH and FSH. (a) LH: in males, it is secreted in a pulsatile fashion; in females in a cyclic fashion, interacting with FSH to control the menstrual cycle; and (b) FSH: in males, it causes growth of sperm and the seminiferous tubules, with a consequent testicular enlargement; in females, the eggs grow under its influence (this explains why, in girls, FSH is especially high around 20–30 postmenstrual weeks, because much of the initial growth of eggs takes place then, during late fetal life). The activity of this gland depends on the age of the host rather than of the organ itself; its full activity is inhibited in immature individuals (Donovan & van der Werff ten Bosch, 1965).

Finally, just to mention another pituitary hormone, which association with aggression has also been described in humans and nonhuman primates: the PRL. PRL is necessary for the secretion of breast milk in the adult, but seems to play little part, if any, in the childhood. In boys, no change of its blood level is observed at puberty; but in girls, a small rise occurs in late puberty.

4.3.2. Report of the studies

In a study on the relations between adrenal and gonadal hormones and aggressive attributes in adolescents, the levels of *gonadotropins* were also measured in the already mentioned NIMH-NICHD sample (ages: 9–14 years). The hypothesis was that if gonadotropins mediate the effects of stress on T, one would expect to see lower levels of LH and FSH in adolescents with aggression problems, and higher ones in highly socially competent adolescents. Higher scores of rebellious attitude in boys related to lower levels of FSH, but unexpectedly also to higher levels of LH and of DHEA (Susman et al, 1987). From the same sample, Nottelmann et al. (1987) reported that adjustment problems were also associated with a similar hormone profile to the described for boys with aggressive attributes. And in another paper (Susman, Granger, Murowchick, Ponirakis, & Worrall, 1996) quite reverse results to the hypothesized are reported: boys with aggressive tendencies and a higher rebellious attitude were higher on FSH, contradicting the hypothesis that stressors suppress the gonadal axes.

An inverse correlation between *PRL* and aggression was observed in macaques (Botchin, Kaplan, Manuck & Mann, 1993), and both tendencies have been reported in adult humans. Sobrinho et al. (in press), studying neurovegetative responses to emotions elicited during a hypnoidal state, have just reported that rage had a marginally significant positive association with PRL surges. And PRL responses to serotonin agonists, such as d,l-fenfluramine or meta-chlorophenylpiperazine, (this response to serotonin agonists is an index for assessment of central nervous system serotonergic activity and responsivity), were also found to be positive correlated with indices of trait hostility and impulsiveness in patients with substance abuse (Fishbein, Lozovsky, & Jaffe, 1989; Handelsman et al., 1998). On the contrary, another PRL [D,L-FEN] challenge study conducted in mood and personality disordered patients, found that PRL responses were reduced in those patients with increased irritability, impulsive aggression, verbal hostility, and direct aggression (Coccaro et al., 1989), as well as in those with a

previous history of suicide attempt (Coccaro, 1996; Lopez-Ibor, Lana, & Saiz, 1990). A recent study in a nonpatient population reported basic neurobehavioral differences between sexes: whereas in men, peak PRL responses to fenfluramine correlated significantly with an interview-assessed life history of aggression, no significant relationships were observed across all women, although subanalyses restricted to postmenopausal subjects (in whom ovarian influences on PRL secretion may be mitigated because of diminished estrogen) showed a pattern of behavioral associations somewhat similar to that seen in men (Manuck et al., 1998).

Studies on this relationship in nonadult humans also reported mixed results. One reported no difference in PRL [_{D,L}-FEN] responses between aggressive CD children and adolescents compared to healthy subjects (Stoff et al., 1992). In another already mentioned study, dealing with different degrees of aggressiveness and neurotransmitter–neuroendocrine responses to stress in male peripuberal adolescents, high-normal aggressiveness was associated with significantly higher basal concentrations of PRL (Gerra et al., 1998).

4.3.3. Summary and comments

The association between aggression and some pituitary hormones, such as gonadotrophins and PRL has also been described in nonadult humans, but for the time being the reported results in humans are still mixed and confusing. This failure to clearly support hypothesis may reflect the reality that psychoendocrine processes are more complex than merely a one-to-one coordinate relation between a hormone and a behavior.

5. Final comments

There have been few systematic studies to explore the relationship between aggression and hormones. Most research on this topic has tried to uncover direct links between measures of biological status, such as levels of the hormone T and individuals' activities, such as their levels of aggression, risk-taking and nurturance. In addition, there have been no well-designed studies of the interaction between biology and an individual's environment in the genesis of aggressive behavior. Most studies of aggression in humans focus on individual different conditions, wrongly assuming that hormonal levels are stable over time of day, failing to consider periods of development in which they act, and forgetting that the biological and behavioral responses to androgens are context-dependent (Book et al., 2001; Rubinow & Schmidt, 1996; Susman & Shirtcliff, 2002). Some inconsistencies in results thus should be due to the different ages of samples. The assessment of the longitudinal stability of the hormonal levels has also failed. There are no longitudinal data on whether the status of being in the top 10% of T distribution in childhood is predictive of being in the top 10% of T distribution in adulthood (Constantino et al., 1993; Susman, Worrall et al., 1996).

There are also disputes about eventual differences between the results obtained according to the methods employed: *in saliva* or *in serum*. Some authors point out that the focus on *serum* hormonal level may be a significant factor in these mixed results, because it does not recognize that intracellular events constitute critical steps in the production of hormonal

effects, including behavioral facilitation (Simon, McKenna, Lu, Cologer, & Clifford, 1996). According to them, circulating hormonal levels may not be the appropriate analysis with respect to physiologically relevant behavioral effects; for instance, a product of T metabolism might be the critical molecule in mediating aggression and hormones (Susman, Granger et al., 1996). As a matter of fact, the hypothesized inverse relationship between CORT secretion and aggression, suggested by results obtained measuring saliva CORT levels, was not found between aggressive and nonaggressive in 7–11-year-old boys with ADHD, whose hormonal levels were measured in *serum* (Schulz et al., 1997).

We also want to stress that the link between hormones and aggression shows a reciprocal and circular interplay between both. In the development of aggression there are involved psychosocial factors, linked to the metabolic and physiological pathways, and with genetic characteristics. Yet we tend to think of aggression as the effect of our biology—i.e., hormonal influence on aggression, whereas it can also be a causal component, influencing hormones. For example, one's own aggression creates stress which in turn can cause changes in adrenal steroids (Grisso, 1996); or, also, the antisocial behavior may influence the activity of the HPG axis (McEwen, 1992). Aggression, as well as other adjustment problems, may suppress the gonadal axis (Susman, Worrall et al., 1996). Hormones, likewise, may be considered potential causes, consequences, and mediators of aggression. For example, behavioral experiences and sociocultural context lead to endocrine changes that, in turn, influence the occurrence of aggression (Andreu, García-Bonacho, Esquifino, & Ramirez, 2001).

T is the hormone most studied in association with aggression in adolescents, but recent results are less conclusive than the first findings (Olweus et al., 1980, 1988) suggested. For instance, one paper (Sanchez-Martin et al., 2000) concluded that T could be a useful biological marker for serious aggression (and behavioral patterns reflecting different levels of sociability) in preschool boys; but another one (Schaal et al., 1996), after revealing that T was positively associated with social success rather than with physical aggression, suggested that T level could be a marker of social success in a given context, rather than of social maladjustment. Perhaps, the identification of biological laboratory markers is not a clinically useful strategy at this time for a biological substrate so complex as the one for aggression (Schulz et al., 1997). This inconsistency has led to the conclusion that gonadal hormones are only one of a myriad of influences on aggression in adolescents. For example, they may reflect the developmental maturational status: in older adolescents, T may have reached a level consistent with the activational influences of hormones.

This suggests that T may have a relationship with sexually dimorphic behaviors, in particular with the expression of aggression, usually different in boys and in girls (Harrisa, 1999; Ramírez, 1978). Until around 10 years of age, the typical way of solving conflict situations is common to both sexes: open and direct physical or verbal aggression. A couple of years later, with the arrival of puberty, strategies become quite characteristic of each sex. Girls stop shouting and hitting and interchanging strikes; direct physical aggression begins to be substituted by indirect emotional aggression, i.e., hidden methods are preferred to direct encounters: ostracism, contempt, gossiping, etc. On the contrary, adolescent males seem to be more impatient and irritable; they tend towards risk-taking behavior, unable to understand

danger, and cravings for thrills and glory.² With maturation a general increase in the use of indirect aggression results, and in adulthood the prevalent type becomes a more subtle form of aggression, known as ‘social’ aggression (Björkqvist, Österman, & Kaukianien, 1992; Owens, Slee, & Shute, 2002; Vaillancourt et al., 2002). Thus both sexes are equally aggressive, but they express it in different ways: for instance, whereas men may express their hostility at work by shouting at the secretaries, women rather tend to do it indirectly, spreading rumors, ‘forgetting’ important tasks, and so on.

There is a close relationship therefore between the developmental onset of steroid hormones and the onset of aggression and violence in adolescent boys, with rapid changes in both hormones and behavior (Susman, Granger et al., 1996). But it does not mean that sex differences arise just at the time of sexual maturation; they begin much earlier in life. The findings of the Basque University’s group suggest that the development of sex-typed behavioral differences is correlated with circulating T, and it is already expressed on early postnatal life, as soon as infants have appropriate motor abilities for interacting. Berenbaum and Hines’ findings also suggest that the origin of these gender-based differences in aggression must lie in neuroendocrinological events occurring during prenatal life, such as the exposure to higher levels of androgens around 3 months in utero. This produces different dispositions in boys and girls, which begin to be shown as soon as children are able to interact, and become more accentuated by age (Archer, 2002).

There is also no reason to expect a one-to-one relation between the increase of T and that of aggression at puberty, and even less a direct hormonal effect, because T is affected by other endocrine systems and is aromatized to other hormones. Also it may not be the active substance implicated in aggression. Brain and Susman (1996) offer a developmental explanation of these inconsistencies, suggesting that T levels may be a consequence and not a cause of aggression in adults.

Even more, if some hormones can affect aggression and violence, they do not directly cause them. T, for example, does not directly put people to fight or to be violent; this only occurs in response to some provoking environmental stimuli. But having a particular hormonal state may predispose the individual to be more or less aggressive when exposed to those stimuli.

The increase of T at puberty might indirectly interact with the psycho-social context (cognitive processes, emotions, family and peer configuration, personal life experiences, etc.) in both ways. For example, winners and losers show different hormonal patterns (Rose, 1980). Watching one’s heroes win or lose has different physiological consequences, including changes in the production of endocrine hormones, that extend beyond changes in

² Over thousands of generations, males seem to have been bred to be thrillingly, gloriously, and expendably stupid at adolescence. Various communities cause their young men to endure a startling and often gory array of harassing rituals and trials in order to become acceptable adults. In his autobiography, Nelson Mandela says that only after his tribal initiation at the age of 15 did he feel ready to assume the chieftaincy he inherited. Many of us have been witnesses of young men physically abused by fraternity brothers during hazing and initiations. Often, only when they have made their bones in some grim initiatory expedition are young men able to contemplate the next steps of courtship and marriage. We have to consider however that these rights *de passage*, characteristically of youngsters, have also a positive side: the channeling of those forces that are coming at puberty.

mood and self-esteem. Basking in reflected glory, in which individuals increase their self-esteem by identifying with successful others, is usually regarded as a cognitive process that can affect behavior. It may also involve physiological processes, as has been presented in a study of changes in T levels among male fans watching their favorite sports teams win or lose, either directly or on television (Bernhardt, Dabbs, Fielden, & Lutter, 1998). Participants provided saliva T samples before and after the contest. In both situations, mean T level increased in the fans of winning teams and decreased in the fans of losing teams. Adolescents thus may try to gain social status through dominance and leadership, via aggressive and assertive behavior. If successful in these pursuits, T is expected to increase. If unsuccessful, T is expected to decrease, because of their overall stress-related aggression problem. It is further expected that the longer the duration of social stress the lower the concentration of gonadal steroids.

Assuming that T and CORT are two hormones which hold promise to shed light on linkages between biology, behavior and environments within the context of the family, an important next step would be to examine the influence of both on family dynamics and subsequent child development, and how family relationships and experiences in turn affect the way that biological factors manifest themselves. In one family, for example, aggressive impulses may be moderated or channeled into constructive domains, while in another, aggressive acts may be unchecked and destructive (Booth, McHale, Crouter, & Granger, *in press*). Stressors thus appear to affect hormone concentrations in humans, even if these effects have received only minimal research attention.

Despite the progress of the last few years, the links between hormones and childhood violence remains woefully understudied. There is a need for more research into the comprehension of how biology works on the development of our behavior. It would provide physicians and psychologists with increased understanding and viable treatment potential for the violently aggressive patient (Strefling, 1990). This will require models that incorporate cellular aspects of steroid hormone action, including metabolism, chemical balance/imbalance, receptor function, and gene regulation. Our present knowledge on this topic is only in its nascent stage, but it will expand. And we hope that it will help to restore to individuals more control of their own destinies, with a consequent better choice and freedom.

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