Introduction

Since the early 1970s psychoendocrinological research on sexually dimorphic behaviour has focused on testosterone, qualitatively the most potent androgen in males (Rommerts 1998), which is also present in females at approximately one-tenth of the male serum concentration (Longcope 1986).

Testosterone does not cause behavioural changes per se; it can only alter the probability that particular behaviour will occur in the presence of a particular stimulus. Sex hormones can influence regions of the central nervous system (CNS) which contain hormone receptors. This interaction of a hormone with its receptor begins a series of cellular events that lead to a genomic response wherein the hormone acts directly or indirectly to activate genes that regulate protein synthesis (e.g. Chalepakis et al. 1990, Genazzani et al. 1992, McEwen 1992, Bixo et al. 1995).

Two decisive phases have been identified in the effects of testosterone on brain structures and consequently on behaviour. During fetal and neonatal life, relatively high concentrations of testosterone are thought to influence brain development by organizing the undifferentiated brain in a sex-specific manner. It has been shown, from studies primarily of rodents, but also of primates and other mammals, that the hypothalamus, the hippocampus, the preoptic-septal region and the limbic system are important target areas for sex steroid action (e.g. Hutchinson 1991, McEwen 1992, Collea & Hines 1995).

These brain structures and hence the corresponding behavioural repertoires are then thought to be activated at the beginning of puberty when the production of sex hormones increases (Schulkin 1993).

In humans, however, hormonal influences on behaviour are much less potent than in animals. Differences in behaviour are considered to result mainly from a combination of intrapsychic, social and cultural factors.

General methodological issues

Hormonal influences on human behaviour are difficult to prove as pertinent research has to rely predominantly on correlational studies of endogenous hormone levels and behaviour which cannot ascertain hormonal influences. Some knowledge derives from clinical studies on individuals who have been exposed to atypical levels of hormones during some developmental period of their lives, so-called ‘experiments of nature’.

Only on very few occasions can scientists ethically manipulate hormone levels in humans in order to examine subsequent effects on brain and behaviour. But even from these studies on hormone substitution one cannot always draw firm conclusions regarding a particular hormonal–behavioural relationship. From double-blind, placebo-controlled studies it may be possible to conclude that a particular hormone is the metabolic agent associated with behaviour. However, such studies are rare exceptions in human behavioural endocrinology. When interpreting psychoendocrinological findings, these caveats should be borne in mind.

The interaction between sex hormones and behaviour is complex. If the relationship is to be investigated, endogenous and genetic factors influencing the gonadal system, e.g. pulsatile patterns, circadian variations, circannual rhythms, age-related changes and body constitution have to be considered in the study design (Christiansen 1999). Moreover, a person’s androgen level and his or her behaviour are mutually dependent; sex hormones can affect behaviour, and behaviour can alter sex hormone levels. Behaviour influencing androgen levels, especially testosterone, has been reported for sexual behaviour, alcohol consumption, nutrition, physical exercise and psychic and psychosomatic stress.

The general concept that behaviour can feed back to hormone levels was first described with regard to sexual behaviour in an often-cited publication. A man working on an island attributed his increased beard growth immediately prior to and during his visits to his girlfriend on the mainland to elevated androgen levels induced by sexual anticipation and sexual activity (Anonymous 1970). Since then, numerous empirical studies have dealt with effects of sexual behaviour on testosterone levels.

It could be demonstrated that almost any sexual behaviour can significantly alter sex hormone levels; however,
cognitive factors and emotional involvement of the sub-
jects produced mixed results. The majority of data on
eugonadal men refer to effects on ejaculation. Orgasmic
frequency in males – whether through sexual intercourse
or masturbation – correlated positively with free, biologi-
cally active testosterone, serum testosterone or dihydrotest-
osterone (DHT) (Kraemer et al. 1976, Monti et al. 1977,
Brown et al. 1978, Knussmann et al. 1986, Dabbs &

Endocrine effects of erotic stimulation were also
investigated. A significant increase in testosterone levels
was found in men after more or less accidental sexual
stimulation through people, erotic pictures and movies
during the 24 h before blood sampling (Christiansen et al.
1984). Even closer correlations were found in controlled
laboratory experiments with men watching erotic movies,
while showing sexually neutral films had no effect upon
testosterone secretion (Hellhammer et al. 1985, Carani

Up to now, very little attention has been paid to
behavioural-androgenic effects in women. Dabbs &
Mohammed (1992) measured salivary testosterone levels
in women after sexual intercourse and detected a signi-
ficant increase in testosterone compared with a baseline
value. Samples taken in the evenings without preceding
coitus did not show such an increase.

In general, alcohol consumption leads to a significant
decrease in testosterone and DHT levels in males, regard-
less of whether beer or wine is consumed. The shorter the
time elapsed after the last drink, the greater the decrease of
androgen levels. Even 10–20 h after drinking, especially if
one suffers from a severe hangover, testosterone levels can
still be significantly suppressed (reviewed in Christiansen
1999).

Nutrition and diet have the potential to affect testos-
terone production and metabolism. A decrease in plasma
testosterone can be observed in men on a low vegetarian
diet or protein–calorie malnutrition. Depressed plasma
testosterone levels also consistently occur as a result of
obesity, due to the elevated metabolism of testosterone
in fat tissue and a decrease in binding capacity of the
sex hormone-binding globulin (SHBG) (overview in
Christiansen 1999).

Effects of physical exercise upon the male and female
reproductive system show some similarities, especially
with regard to testosterone, when intrinsic gender
differences in the endocrine system are acknowledged
(Shangold 1984, Hackney 1989). Despite their athletic
appearance, male athletes have lower androgen levels than
unselected men in a resting state. Testosterone concen-
trations of trained subjects were only 60–85% of age-
matched untrained men, although no difference in mean
plasma SHBG capacity could be detected between trained
and untrained men (Kuoppasalmi 1980). Acute effects of
submaximal, prolonged exercise in marathon runners or
cross-country skiers resemble hormonal changes found in
endurance-trained men during the resting state. A highly
significant decline in testosterone concentrations com-
pared with pre-competition baselines was observed (Arce
while young sportswomen had significantly increased
testosterone levels after a long-distance run (De Crée et al.
1990). Regardless of the kind of sport, maximal or
submaximal exercise (5–30 min) normally results in sig-
nificant increases in testosterone levels in males which are
independent of luteinizing hormone (LH) and follicle-
stimulating hormone secretion (Cumming et al. 1986).

The apparent disagreement between the effects of sub-
maximal, prolonged exercise and 5–30 min exercise on
testosterone levels in males are explained by the early
(non-LH dependent) rise in testosterone concentrations
(Adlercreutz et al. 1976) and a decreased metabolism of
testosterone due to a drop in hepatic blood flow (Cadoux-
Hudson et al. 1985).

Stress responses of the pituitary–gonadal axis are not as
well known as the activation of the pituitary–adrenal axis
with the subsequent release of cortisol, which is considered
as one of the major components of the physiological stress
response. However, stress-induced responses of the
pituitary–gonadal axis altering testosterone levels are
impressive in their sensitivity to various psychosomatic
and psychic stressors, e.g. combat training, surgery with
anaesthesia, driving heavy-good vehicles, routine flight
missions in fighter-type aircraft as pilot or crew member,
and situations eliciting psychic stress such as examinations,
loss of close friends and relatives, dissatisfaction and
boredom (Christiansen et al. 1985, reviewed in
Christiansen 1999). Even the anticipation of a stressful
event, an end-of-term examination at university, can lead
to a sex–typical reaction of the pituitary–gonadal axis: a
significant decline of testosterone levels in males and an
increase in testosterone in females (Christiansen & Hars
1995). At a glance, this seems to be a contradictory result.
But from the view-point of evolutionary biology, it
makes sense. In males, spermatogenesis is testosterone-
dependent; in females, high levels of androgens are
associated with anovulatory menstrual cycles. Thus, both
reactions suppress fertility in order to increase the survival
of the individual and its family or group who are living
under threatening or unfavourable, so-called stressful
circumstances, which could be hazardous for pregnancies
and infants, and the raising of children.

Influence of androgen on human behaviour

Sexual behaviour

It has long been recognized that androgens play a critical
role in human male sexual behaviour, although it can be
profoundly influenced by intrapsychic, social, somatic and
cultural factors. It is quite obvious that the general pattern
of age-dependent rise and decline of androgen levels in
Men correspond to average levels of male sexual activity throughout the life-cycle. Before puberty, boys do not engage in sexual activity outside the context of play. After puberty, when the testes begin to secrete androgens, sex drive and the motivation to seek sexual contact become powerful and are overtly expressed. When blood levels of testosterone, especially free testosterone, diminish as men age, this mirrors their usually declining sexual interest, arousability and potency.

The physiological range of testosterone lies between 3 and 12 ng/ml, which is higher than necessary to maintain normal sexual functions. Testosterone levels found to be critical for sexual functions in males lie below or around 3 ng/ml, and they show a clear intersubject variation. Thus, there exists no generally critical testosterone value at which androgen-related sexual behaviour definitely stops.

Besides evidence from non-human primates and clinical case reports on effects of castration in human males, studies of hypogonadal men on androgen-replacement therapy provide convincing evidence of the essential role of androgens in some aspects of male sexual behaviour. In patients with induced or spontaneous hypogonadism, both pathological withdrawal and reintroduction of exogenous androgens affected the frequency of sexual interest and fantasies, sexual arousal and desire, spontaneous nocturnal or morning erections, ejaculation, sexual activities with and without a partner, and orgasms through coitus or masturbation (Salminies et al. 1982, Bancroft 1984, Gooren 1987, Carani et al. 1990b).

There is only limited evidence on the effects of testosterone administration to eugonadal men with or without sexual problems. In a controlled study of eugonadal men with diminished sexual desire, injections of testosterone produced a significant increase in sexual interest when compared with placebo-injected men (O’Carroll & Bancroft 1984). But in most of the men studied the increase in sexual interest was not translated into an improvement of their sexual relationship – perhaps because psychological problems with their partner had not been resolved with hormonal treatment alone.

When supraphysiological doses of testosterone, used as potential hormonal male contraceptive agents, were administered to healthy volunteers, this resulted in a significant increase in psychosexual stimulation or arousal during testosterone substitution. But there was no change in sexual activity or spontaneous erections (Anderson et al. 1992, Bagatell et al. 1994).

This led to the conclusion that androgens are only beneficial to sexual activities in those men whose endogenous levels are abnormally low. However, one cannot be certain on this point because with increasing levels of endogenous androgen it becomes more difficult to manipulate circulating levels with exogenous hormones. The homeostatic mechanisms are powerful and the more testosterone is administered, the more the individual’s own supply is suppressed or the metabolic clearance rate is increased. Failures to produce any effect on a man’s sex life other than on sexual interest and desire therefore may not be due to ineffective androgens, but rather a result of their failure to alter hormone levels significantly.

In several studies a significant relationship between endogenous androgen levels in normal males and sexual behaviour was observed: the degree of sexual interest, frequency of nocturnal and morning erections and orgasmic frequency varies significantly with endogenous androgen levels (Knussmann et al. 1986, Mantzoros et al. 1995, Nilsson et al. 1995). However, there exists a great interindividual variability of behavioural responses to hormones. In a longitudinal study (Knussmann et al. 1986) on young healthy volunteers, predominantly significantly positive intraindividual correlations of testosterone and orgasmic frequency were found. But some men showed significant negative or insignificant correlations, and this could explain contradictory results from other pertinent studies on testosterone levels and frequency of orgasms.

Less obvious and difficult to infer from everyday observation is the role of testosterone in female sexual behaviour. Physiological testosterone levels in women, which are one-tenth of those in the normal male and to which males are unresponsive, were, for a long time, thought to be irrelevant. Thus, the idea that androgens could have enhancing effects on female sexual desire and arousal received little attention until testosterone was used to treat oophorectomized women.

A variety of models have been employed to test the relationship between testosterone and sexuality in women. Because plasma testosterone levels peak around the time of ovulation, one investigational strategy has involved monitoring changes in several aspects of sexual behaviour at different points during the menstrual cycle. As plasma levels of oestradiol also reach their highest point at the ovulatory phase, this research design makes it difficult to prove that testosterone alone induces the increase in sexual behaviour during the mid-cycle portion of the menstrual cycle observed in some studies.

But several well-controlled correlational studies measuring endogenous concentrations of testosterone in women found evidence of an androgenic enhancement of sexual behaviour: less sexual avoidance, more sexual gratification, sexual thoughts, initiation of sexual activity, higher levels of sexual interest and desire, more vasocongestive responses to erotic films; increased frequency of masturbation and coitus, and a higher number of sexual partners (Persky et al. 1978, 1982, Schreiner-Engel et al. 1981, Morris et al. 1987, Alexander & Sherwin 1993, Cashdan 1995).

The most powerful design for the study of the specificity of testosterone influence involves hormone-replacement therapy in women who are oophorectomized. Some studies have shown – without contradictory evidence – that administration of testosterone, either alone or in addition to an oestrogen-replacement regimen, is more
effective than oestrogens alone or a placebo. In particular, an increase in sexual desire and fantasies was elicited, but also in sexual arousal and in coital or orgasmic frequency (Sherwin et al. 1985, Sherwin & Gelfand 1987).

Although there is converging evidence from correlational and experimental investigations that testosterone enhances male and female sexual behaviour the underlying behaviour mechanism is not known. Testosterone might have direct effects on cognitive behaviour, e.g. influence the awareness of sexual cues, but it is also suggested that testosterone may act peripherally to enhance sexual pleasure and, thereby increase sexual desire and even sexual activity, circumstances and partner permitting.

Cognitive abilities

Although there is no difference in performance in modern standardized tests of intelligence between females and males, sex differences in certain cognitive tasks have been reported for years. According to recent meta-analyses the differences have been decreasing since the 1970s; however, they remain significant (Hyde & Linn 1988, Hyde et al. 1990). On average, females excel at verbal tasks, whereas males outperform females on visual–spatial abilities (map reading, mental rotation and manipulation). The significant differences in abilities are slight, and there is more variation within each sex than between the sexes.

It is generally accepted that among many other sources of variance which contribute to the sex differences in cognition, sex hormones, especially androgens, play a critical role in sex-typical cognitive functioning as well as in interindividual differences within the sexes. Evidence of a connection between sex hormones and cognitive functioning came first from studies of men and women with endocrine dysfunction: phenotypic women with Turner’s syndrome, kwashiorkor-induced endocrine dysfunction in boys, men with hypogonadotrophic hypogonadism and children with congenital adrenal hyperplasia. Direct manipulation of steroid hormone levels supports the conclusion that androgens play a role in cognition. Administration of testosterone to castrated males, eunuchs, and older males improved their attention and alertness as long as the androgen treatment lasted. Either testosterone, oestradiol, or a combination of both hormones produced a significant rise in concentration and speed in solving simple arithmetic problems in a group of males with severe mental exhaustion. Similar results were reported for poorly androgenized male adolescents. Positive effects of testosterone treatment were also observed when normal, ageing men were given testosterone to enhance sexual function; as a side-effect they showed improved performance on visual–spatial tests (reviewed in Christiansen 1999). A similar result was obtained in female-to-male transsexuals who received high doses of testosterone in preparation for sex therapy. Their spatial skills improved dramatically and their verbal skills declined considerably within 3 months (Van Goozen et al. 1994b). Sherwin (1988) demonstrated in a double-blind, cross-over, placebo-controlled study that oophorectomized women substituted with testosterone or a combination of oestradiol and testosterone showed stability on short- and long-term memory and perceptual speed, typically the most prominent deficits in menopausal women.

A few studies investigating endogenous androgens detected a curvilinear relationship; high levels of testosterone in females but low androgen levels in males were associated with superior performance in visual–spatial tasks (Gouchie & Kimura 1991). However, the majority of the data show a positively linear androgen–cognitive relationship in males as well. Testosterone and DHT concentrations correlated significantly and positively with visual–spatial orientation and negatively with verbal tests. Correspondingly, a more ‘masculine’ cognitive pattern (superior skills on spatial tests as compared with verbal tests) is positively correlated with androgen concentrations in healthy males (Christiansen & Knusmann 1987a). The evidence from men living in Western cultures was validated by a study on Kung San hunter-gatherers and Kavango farmers from Namibia (southern Africa), who lived mainly at the subsistence level. The African data yielded the same hormonal–cognitive pattern as was found in Western samples: testosterone showed the greatest number of significant relationships, a positive one with visual– or tactual–spatial tasks and a negative association with verbal tests (Christiansen 1993).

Although it is reasonable to conclude from pertinent studies that testosterone plays a role in cognitive functioning throughout life, from the prenatal period till old age, any causal model will have to recognize the reciprocal effects that environment and androgen have on each other.

Aggression and mood

The relevance or irrelevance of testosterone for human aggression has received enormous public attention, usually culminating in highly emotional discussions based on misinterpretations of scientific research. Since 1972, many empirical studies on criminals have shown that testosterone is generally positively associated with a history of violent crime and aggressive or violent behaviour in prison. These findings encouraged many people to conclude that castration of highly aggressive males would keep them from further killing or injuring innocent people, raping women and abusing children, and would finally turn them into well-adjusted members of society. This statement is quite often heard after a spectacular case and has been seriously discussed as a solution to the problem. Regardless of the method of castration – surgical removal of testes or chemical castration with anti-androgens – it cannot solve the problem of unprovoked violent crimes.
In humans, testosterone cannot illicit violence, it can only alter the probability that aggression is shown in a particular situation under a specific combination of external and internal cues.

Moreover, results in psychoendocrinological research are based on statistical evaluation of data collected from a group of persons, the sample. Even if findings suggest that testosterone is related to antisocial and offending behaviour, conclusions can only be drawn with a certain probability for the sample and its universe. We cannot predict future aggressive behaviour for any individual subject. A significant positive correlation of testosterone with violence in the study group does not correspond necessarily with a high degree of violence in all individuals of the sample. Even if for the majority of probands a positive relationship of testosterone and aggression were found, there are also individuals with relatively low testosterone levels and a high degree of aggression in the same study group.

Evidence of androgenic influence on human aggression comes from investigations of prenatal testosterone exposure, endogenous androgen levels, and from testosterone substitution in males and females. Animal studies generally indicate that the presence of androgens in early life is important in establishing a biological readiness for future aggressive behaviour. Much of the pertinent research on psychological effects of sex hormones in human studies consists of naturally occurring syndromes which result from either spontaneous endocrine excess or deficiency during fetal and early postnatal life or prenatal sex hormone treatment of the pregnant mother. Overall, the studies show a slight but significant effect of exposure to testosterone and progesterone with androgenic potential: an increase in physical aggressiveness and intense energy expenditure (e.g. vigorous play) but not in verbal aggression during childhood years. The positive effects of early exposure to sex hormones are significant for both boys and girls, yet these influences seem subtle (reviewed in Christiansen 1999). It was suggested that organizing effects may only be detected clearly after puberty in the presence of adult sex hormone levels. This has been tested in a study by Yalom et al. (1973), and indeed they found stronger correlations of prenatal anti-androgen exposure with aggressive behaviour in adolescents after reaching puberty.

In adults, aggressive behaviour is predominantly positively correlated with endogenous testosterone levels although the aggressive act is generally removed from hormone assessment. The hormonal effect is now referred to as activational as opposed to organizational in early life. A meta-analysis of pertinent studies employing common measures of aggression showed a weak, significantly positive correlation between testosterone and trait measures of aggression (self-reported by using questionnaires) and a stronger positive correlation with behavioural measures (Archer 1991).

In the beginning, a number of studies were carried out in prison where usually some of the inmates are highly aggressive. Here, the researchers most likely expected to ascertain a significant relationship between current testosterone levels and aggression and they actually did (Table 1).


Investigations of female prison inmates confirmed the results obtained from male offenders. Even the low testosterone concentrations in women seem to exert an influence, but only on unprovoked violence (Dabbs et al. 1988).

In a group of traditionally living Kung San hunter-gathers (so-called bushmen) from the Kalahari desert in Namibia we found that within a subgroup of physically aggressive San men violent behaviour correlated significantly and positively with free testosterone and DHT levels. As the physically aggressive men also exhibited higher mean values in body measurements of robustness of the face and trunk this finding may point to a possible pathway of indirect androgen action on human aggression, in addition to the widely accepted influence of testosterone on aggressive behaviour via its action on specific sites in the CNS (Christiansen & Winkler 1992).

Aggressive behaviour which was not a punishable offence also showed significant correlations with androgens in men and women. During experimentally controlled alcohol intake, aggressively predisposed students were more dominant in a discussion and had higher free testosterone levels than non-aggressively predisposed students (Lindman et al. 1987). In male hockey players the pre-play testosterone levels correlated positively with reactive aggression during the tournament (Scaramella & Brown 1978). In pubescent boys, their peers’ and mothers’ ratings of aggressive behaviour correlated positively with testosterone and several other androgens (Olweus et al. 1980, Susman et al. 1987). Male and female patients in a clinic for nervous diseases showed more destructive aggression with higher levels of testosterone (Kedenburg 1977, Ehlers et al. 1980).

The evidence for testosterone influence on aggressive behaviour is not always significant, but still positive. Neither normal males (Aromäki et al. 1999), pubescent boys (Susman et al. 1987) nor rapists (Rada et al. 1983) exhibited a significantly positive testosterone-aggression relationship. Men who had been arrested for wife-battering under the influence of alcohol did not have significantly higher testosterone levels than a control group comprising non-violent public-house patrons with a similar alcohol consumption (Lindman et al. 1992).

Although correlational data suggest that aggressive behaviour in men and women are related to current, endogenous testosterone levels, they cannot prove a cause-effect relationship. In addition to studies on prenatal hormone treatment, research on the effects of testosterone intake in the adult female and male can possibly answer the question whether aggression is actually testosterone-dependent (Table 2).

Anabolic-androgenic steroid (AAS) abuse in men and women (Strauss et al. 1983, Bahrke et al. 1990, 1992, Perry et al. 1990, Brower et al. 1991), controlled AAS substitution in sportsmen (Hannan et al. 1991), testosterone-replacement therapy for hypogonadal adolescent or adult males (Skakkebaek et al. 1981, Finkelstein et al. 1997), in surgically menopausal females (Sherwin & Gelfand 1985) and in female-to-male transsexuals (Van Goozen et al. 1994a), and exogenous testosterone as a potential hormonal male contraceptive (Anderson et al. 1992, Bagatell et al. 1994) can result in increased irritability and aggressiveness, but not necessarily.

Increased aggressiveness can occur, even more so in high-dose AAS users. But to claim that aggression in AAS users is only testosterone-dependent is too simple. Factors such as unstable personality may be the source of willingness to abuse steroids, as well as of aggressiveness. It was observed that irritability was slightly increased in many male steroid users but that only in a few, who were premorbid, might steroid use have been sufficient ‘to push them over the edge’ and contribute to irrational or violent behaviour. Greater risks of steroid abuse come from the association of high testosterone levels with emotional well-being.

Significantly positive correlations of endogenous testosterone levels in men of all ages with elation, joyfulness and sense of well-being have been observed and negative relationships with depression, sadness and anxiety (Wang et al. 1996, Nieschlag & Behre 1997, reviewed in Christiansen 1999, Barrett-Connor et al. 1999, Grinspoon et al. 2000). Research on anabolic steroid users support these findings. Members of body-building studios using

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Table 1 Endogenous testosterone and aggression in women and men (references are given in the text)

<table>
<thead>
<tr>
<th>Relationship</th>
<th>Males</th>
<th>Females</th>
</tr>
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<tbody>
<tr>
<td>Significant positive</td>
<td></td>
<td></td>
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<tr>
<td>Insignificant positive</td>
<td></td>
<td></td>
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<tr>
<td>Significant negative</td>
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</tbody>
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<table>
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<tr>
<th>Self-reported measures of aggression</th>
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<tbody>
<tr>
<td>Males</td>
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<tr>
<td>Prison inmates</td>
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<tr>
<td>Adolescent males</td>
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<tr>
<td>Adult males</td>
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<td>College students</td>
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<td>University students</td>
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<td>Normal males</td>
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<tr>
<td>Psychiatric patients</td>
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<td>Pubescent boys</td>
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<td>Hockey players</td>
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<tr>
<td>Pubescent boys</td>
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<tr>
<td>Prison inmates</td>
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<tr>
<td>Normal males</td>
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<tr>
<td>Psychiatric patients</td>
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<tr>
<td>Pubescent boys</td>
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<tr>
<td>Prison inmates</td>
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</tbody>
</table>
steroids were contacted and offered a cash payment to engage in confidential interviews about their steroid use (Pope & Katz 1988, 1989). One-third of the individuals were reported to be manic or near manic. Most symptoms subsided when anabolic steroid use was discontinued.

Perry et al. (1990) investigated 20 competitive and non-competitive weight- lifters who consistently practised anabolic steroid abuse in cycles lasting 7–14 weeks and found similar results. Accompanying the changes in physical parameters, 70% of the men experienced becoming depressed more frequently while cycling. Clinical syndromes of depression were noted in 40–50% of the subjects, including low energy levels and excessive worrying. According to Kashkin & Kleber (1989), anabolic steroids might have direct reward properties. Increasing numbers of unexpected suicides in previously non-depressed young men who abruptly stopped using anabolic steroids have been reported. In dependent AAS users withdrawal of the drug resulted in depressed mood and fatigue (Brower et al. 1989, 1991). Thus, withdrawal symptoms manifested by anabolic steroid abusers and the symptoms of postpartum depression in women may result from a common underlying cause, dependence upon elevated steroid hormone levels.

In women, both low and elevated androgen levels seem to be associated with depressive mood in adolescent girls (Paikoff et al. 1991), adult females (Hartmann et al. 1996), and in women with premenstrual dysphoria (Eriksson et al. 1992). On the other hand, women with abnormally low androgen levels after oophorectomy reported significantly less depression and anxiety during testosterone substitution reaching normal female serum concentrations (Sherwin & Gelfand 1985). In a large group of perimenopausal women, those with still relatively high endogenous dehydroepiandrosterone sulphate (DHEA-S) levels felt less dysphoric and had a higher degree of emotional well-being in comparison with women with low DHEA-S concentrations (Christiansen & Hars 2000). These findings point to a negative effect of hormonal changes from low to high levels (e.g. puberty, premenstrual dysphoria) or withdrawal from normal androgen levels (postpartum, perimenopause, castration).

To understand the complexity of the relationship between sex hormones and aggression one further aspect has to be considered. Testosterone and aggression seem to be mutually dependent. In addition to sex hormone influences on human aggression, several studies have shown that assertive or aggressive behaviour followed by a rise in status leads to an increase in testosterone levels (Mazur & Lamb 1980, Elias 1981, Gladue et al. 1989, McCaul et al. 1992, Gonzales-Bono et al. 2000). Personal merit may produce different testosterone responses in winners and losers. In a basketball match, the objective personal contribution to the outcome of the game was positively related to testosterone changes, suggesting that personal merit is a factor that could explain part of the variability observed in the competition-induced testosterone response of the players (Gonzalez-Bono et al. 1999). The experience of winning and of a rise in status even seems to maintain an already elevated level, sustaining the winner’s activation and readiness to enter subsequent competitions for higher status. Testosterone levels in tennis players rose 15 min before the next match if the individual had won the previous match and probably anticipated winning again (Booth et al. 1989). The experience of winning and of a rise in status seemed to produce a rise in testosterone, or to maintain an already elevated level, sustaining the winner’s activation and readiness to enter subsequent competitions for higher status. Mazur’s (1985) ‘biosocial theory of status’ incorporates these findings by hypothesizing a feedback loop between an individual’s testosterone level and his or her assertiveness in attempting to achieve or maintain interpersonal status or dominance rank. This feedback loop may account for winning and

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**Table 2 Testosterone substitution and aggression in women and men (references given in the text)**

<table>
<thead>
<tr>
<th>Relationship</th>
<th>Samples</th>
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<tbody>
<tr>
<td>Significant positive</td>
<td>Males: Weight-trained athletes (AAS)</td>
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<tr>
<td></td>
<td>Weight-lifters (AAS)</td>
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<tr>
<td></td>
<td>Hypogonadal adolescents</td>
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<tr>
<td></td>
<td>Body-builder (AAS)</td>
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<tr>
<td></td>
<td>Male-to-female transsexuals</td>
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<tr>
<td></td>
<td>Healthy males under controlled AAS (short-distance runner, football player, wrestler, body-builder, weight-lifter)</td>
</tr>
<tr>
<td>Insignificant positive</td>
<td>Males: Hypogonadal males</td>
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<tr>
<td></td>
<td>Weight-lifters (AAS)</td>
</tr>
<tr>
<td></td>
<td>Normal males (testosterone for contraceptive use)</td>
</tr>
<tr>
<td>Insignificant negative</td>
<td>Males: Healthy males</td>
</tr>
<tr>
<td></td>
<td>College students</td>
</tr>
</tbody>
</table>
losing ‘streaks’ because each win reinforces a high testosterone level, which in turn reinforces further assertiveness or aggression.

This model of reciprocal effects between sex hormones and environment, although being more complex than simple hormonal or experiential factors, does not fully explain the variation in aggressive behaviour between individuals. Much work still remains to be done before we will have reliable predictors of aggressive behaviour.

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