

Testosterone, Sexual Offense Recidivism, and Treatment Effect Among Adult Male Sex Offenders

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The relationship between serum testosterone and sexual violence was examined in a sample of 501 convicted adult male sex offenders attending an intensive in-hospital group psychotherapy treatment program. It was found that men with higher testosterone tended to have committed the most invasive sexual crimes ($p < .001$, two-tailed). Further, a positive partial correlation (controlling for age) between testosterone and sexual offense recidivism over a lengthy follow-up period (mean = 8.9 years) was found. When the sample was separated into one group that completed treatment and one group that did not, an important ameliorating treatment effect was observed. Although controlling for age, serum testosterone remained significantly predictive of sexual recidivism for the treatment noncompleter group ($p < .05$, two-tailed). For those who completed treatment testosterone was no longer predictive of sexual reoffense ($p > .05$, two-tailed). Among convicted sex offenders, higher serum testosterone appears to be associated with greater likelihood of further sexual violence. Effective therapy, however, appears able to intercede in the influence of testosterone on sexually deviant behavior. It is suggested that serum testosterone may be an informative static risk factor and completion of intensive treatment should be accorded significance in future actuarially based risk prediction instruments.

KEY WORDS: testosterone; sex offenders; recidivism; treatment effect.

Androgens are steroid hormones that mediate sexual development and sexuality. The most relevant hormone in this regard is testosterone, along with its active metabolite dihydrotestosterone (DHT). Testosterone production is initiated when gonadotropin-releasing hormone (GnRH) is secreted from the hypothalamus. In

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postpubertal males, GnRH is secreted in pulsatile fashion approximately every 2 hr (Conn & Crowley, 1991). GnRH stimulates the anterior pituitary gland to release luteinizing hormone (LH) and follicle-stimulating hormone (FSH). These two hormones then stimulate testosterone production by the Leydig or interstitial cells in the testes (Conn & Crowley, 1991; Griffin & Wilson, 1992). Smaller amounts of testosterone are also produced by the adrenal glands (Mazur & Booth, 1998). Typically, testosterone is approximately 98% bound, not only to sex-hormone-binding globulin (SHBG) mainly but also to albumin (Brown, 1999).

Testosterone has a diverse set of effects upon and throughout development: in utero, in childhood, through puberty, and into adulthood (Harris, 1999; Mazur & Booth, 1998). The effects of the androgens are mediated through androgen receptors that are found in a variety of tissues (Keller, Ershler, & Chang, 1996). After a peak in puberty and early adulthood, serum testosterone concentrations tend to fall gradually with age, especially after age 40. It has been suggested that prenatal and perinatal testosterone levels have a major influence upon the organization and structure of androgen receptors. In adulthood then, testosterone is thought to influence behavior through interaction and activation of these preexisting structures (Mazur & Booth, 1998). There is also evidence that these receptors can be active in the absence of the steroid hormone itself (Auger, 2004), further complicating the original receptor theory (Baulieu et al., 1971).

Although age is a known factor affecting serum testosterone, other environmental factors have been shown to have an influence. These factors include stress, success, failure (Gladue, Boechler, & McCaul, 1989; Mazur & Lamb, 1980; Thompson, Dabbs, & Frady, 1990), diet (Aldercreutz, 1990), and sexual activity (Kraemer et al., 1976). However, it is noteworthy that, in general, the intraindividual variance is less than the interindividual variance in these serum levels (Couwenbergs, Knussmann, & Christiansen, 1986). That is, genetic, or at least constitutional factors seem to account for much more of the variance than environmental factors.

To date, the associations with behavioral phenomena (e.g., aggression) have been correlated more with total serum testosterone values than with free testosterone measures (Pugeat et al., 1988). According to the free hormone hypothesis (Ekins, 1990; Mendel, 1989), it should be the unbound portion of the hormone that is biologically (physiologically) active. Insofar as testosterone is concerned, however, there is some doubt about this theory and evidence is mounting that the bound portion of the hormone may also be physiologically active. It is possible that these hormonal complexes may have transport mechanisms of their own (Pardridge, 1991; Rosner, 1990).

There is significant evidence of testosterone's influence on both aggression and sex drive in males (Ellis, 1986; McManus & Bryden, 1991). For example, low testosterone has been implicated in erectile dysfunction (Tsumijima et al., 2003). In a study of prisoners, testosterone among socially dominant inmates was found

to be higher than among other inmates, and higher again among those inmates identified as chronically aggressive (Ehrenkranz, Bliss, & Sheard, 1974; Mazur & Booth, 1998). Rada, Laws, and Kellner (1976) reported that rapists who committed crimes with a greater degree of violence had higher testosterone than rapists who used less violence.

Archer (1991) conducted a meta-analysis on the literature examining the interaction between testosterone and aggression; an overall effect of $r = .38$ was obtained for the relationship between testosterone and ratings of aggressiveness by others. Archer, Biring, and Wu (1998) have also reported more recently, a mean effect size of $d = 0.40$ ($r = .20$) in a review of 18 studies comparing student samples with aggression prone groups. Even after removing 3 outlier studies with the largest reported effect sizes, the association between testosterone and aggression remained modest but highly significant ($d = 0.28$, $r = .14$; Archer et al., 1998). Book, Starzyk, and Quinsey (2001) reported a similar finding in their own meta-analysis. That is, the overall correlation between testosterone and aggression was modest and significant ($r = .14$). Archer, Graham-Kevan, and Davies (in press), however, reanalyzed the Book et al. data and arrived at an effect which was smaller ($r = .08$), but retained statistical significance.

It has been reported that there are differences in testosterone levels in some different groups. For example it has been demonstrated that there are inter-racial differences in total serum testosterone between North America Indians (includes mixed racial North American Indians) and Caucasians (Brooks & Reddon, 1996; Studer, Reddon, & Siminoski, 1997). Among North American adult males, Blacks reportedly have significantly higher testosterone levels than Whites (Ellis & Nyborg, 1992). Men with higher testosterone are less likely to marry, and if married, are more likely to divorce (Mazur & Booth, 1998). Finally, lower socio-economic status has been suggested as a possible factor in encouraging antisocial behavioral expression associated with higher testosterone among males (Dabbs & Morris, 1990).

Given the evidence that testosterone has some association with aggression, and differences in testosterone levels exist between some groups, the authors decided to test two hypotheses. First, that high serum testosterone is correlated with severity of offense and second, that testosterone level is correlated with sexual offense recidivism among a known sex offender sample.

METHOD

Participants

The participants in this study consisted of 520 voluntary admissions to the Phoenix Program, an in-patient sex offender treatment program located in western

Canada (Studer, Reddon, Roper, & Estrada, 1996). All patients were adult males who had been convicted of sexual offenses of various types (i.e., adult victims, child victims, intra- and extrafamilial victims, exhibitionists). Briefly, the program is comprehensive, intensive, and relies almost exclusively on group psychotherapy as the treatment modality. Types of group therapy include psychoeducational, cognitive behavioral, and general interpersonal groups on the basis of the principles outlined by Yalom (1995). The offenses were deemed “serious” enough by the judiciary to warrant periods of incarceration.

Serum testosterone levels were collected as part of a routine battery of endocrine testing. Samples were always drawn in the early morning and usually obtained within the first week of admission. Also included in the battery were FSH, LH, SHBG, as well as albumin, for completeness. Samples were then assayed using commercially available kits. Because of the unavailability of testosterone data, 14 cases were dropped from the sample.

Antiandrogenic pharmacotherapy is sometimes used in treating sex offenders. Because these drugs inhibit testosterone availability, four patients who entered the treatment program on these drugs were also dropped from the sample. Of the patients comprising the final sample, none were on any such medication at the time of the initial blood work, nor were any patients on these medications for prolonged periods of time during or posthospitalization to the best of the authors’ knowledge. One patient was excluded because of HIV-positive status because it was unclear how this individual’s compromised health may have affected testosterone levels. The final sample consisted of 501 patients.

Procedure

The first hypothesis stated there would be a positive relationship between serum testosterone levels and the degree of sexual violence exhibited by sexual offenders. To test this, a correlation was obtained between testosterone levels and offense severity. Offense severity was assigned according to the most intrusive assault known to have been committed by the patient. This may have been an assault known or unknown to authorities. Severity codes ranged from 1 (*non-contact*) to 6 (*severe violence*) and have been used in a previous examination of patients from this program (see Aylwin et al., 2000). For this portion of the analysis, there was no rationale for controlling for age, as the offenses were historical and the rating was based on the most severe offense regardless of the offender’s age.

The second hypothesis stated that there would be a positive relationship between testosterone levels and sexual offense recidivism. In order to provide the most complete test of this hypothesis, the data were examined from two perspectives. In the first part of the analysis, the absolute value for serum testosterone was used. As some might argue that there is a range of normal testosterone values,

the dichotomy of “normal” versus “above normal” might be most relevant. The sample was therefore reexamined after being divided into one group within the normal range and another who had high testosterone. There is slight variation on what is considered the upper limit of the normal range. To be fully inclusive, the range for “normal” serum testosterone for this study was set at 8.0–29.9 nmol/L. “Above normal” testosterone was considered to be ≥ 30.0 nmol/L. Data was coded dichotomously (0 = normal, 1 = high). Three cases were dropped in the dichotomous recoding due to testosterone levels that were below the normal range. None of these three individuals completed treatment.

Recidivism was defined as a conviction for a postdischarge sexual offense. As part of ongoing program evaluation, reoffending is tracked through the Canadian Police Information Centre (CPIC) to determine sexual recidivism. The mean length of time from discharge from the in-patient program to CPIC follow-up was 106.7 months (range: 6.0–188.0 months, $SD = 49.5$). Of note, the length of the follow-up period is very substantial and long enough to capture a great majority of those offenders who may be prone to reoffend.

As both recidivism and testosterone levels amongst sexual offenders are related to age, it was important to control for this variable in the part of our study which involved recidivism. Consequently, to control for age, partial correlations were computed to determine the strength of relationship between testosterone and recidivism. To investigate the influence of treatment efficacy on this relationship, the sample was subsequently reexamined on the basis of treatment completion or noncompletion.

RESULTS

In testing the first hypothesis, there was a significant positive correlation between historical (past) offense severity and serum testosterone ($r_{499} = .186$, $p < .0005$, two-tailed; 95% confidence interval $.100 < .186 < .269$). When testosterone was coded as a dichotomous variable, the correlation between this variable and maximum historical offense severity was similar ($r_{496} = .170$, $p < .0005$, two-tailed; 95% confidence interval $.083 < .170 < .254$).

Table I shows the results of a comparison between treatment completers and treatment noncompleters. In most ways the groups were comparable. Similar proportions of each group had testosterone in the high range ($p = .926$), and similar proportions were of North American Indian (includes mixed racial North American Indians) heritage ($p = .131$). The groups were also comparable with regards to mean age ($p = .521$) and number of prior sex offenses ($p = .473$). There were two notable differences between the groups. The treated group had, overall, higher testosterone ($p = .041$, two-tailed; $d = 0.18$), and more serious/invasive sexual offenses ($p = .002$, two-tailed; $d = 0.28$). Although neither of these effect sizes are large they are nonetheless real differences (i.e., statistically significant).

Table I. Comparison of 501 Treatment Completers and Noncompleters of the Phoenix Program

Variable	Treatment completers (<i>n</i> = 270)			Treatment noncompleters (<i>n</i> = 231)			
	<i>n</i> (%)	<i>M</i>	<i>SD</i>	<i>n</i> (%)	<i>M</i>	<i>SD</i>	<i>p</i>
Testosterone (nmol/L)	—	21.96	6.43	—	20.69	7.40	.041*
Portion with high testosterone	32 (11.9%)	—	—	28 (12.1%)	—	—	.926
Portion N.A. Indian ^a	40 (14.8%)	—	—	46 (19.9%)	—	—	.131
No. prior sex offenses	—	.46	1.15	—	.53	1.30	.521
Age (years)	—	35.4	8.80	—	36.0	11.25	.473
Offense severity ^b	—	3.97	1.22	—	3.62	1.25	.002**

Note. Means compared using independent samples *t*-test; percentages were compared using a phi correlation coefficient (significance identical to Pearson's chi square).

^aDenotes North American Indian (includes mixed racial North American Indian).

^bBased on severity codes from Aylwin et al., 2000.

p* < .05, two-tailed. *p* < .01, two-tailed.

When the pooled sample of treatment completers and noncompleters was examined, the percentage of patients with serum testosterone 30.0 nmol/L or above was 14.5%. In the general population, a rate of 2.5% would be expected (T. Higgins, personal communication, September 22, 2004). Only three patients (0.60%) had testosterone levels below the normal range. There was no difference in the proportional rates of testosterone in the high range between completers and noncompleters (*p* = .926).

For the combined group, while controlling for age, it was observed that sexual recidivism was demonstrably higher in those with higher serum testosterone. There was a significant partial correlation between serum testosterone when coded as a continuous variable, and sexual recidivism ($r_{498} = .113$, *p* = .012, two-tailed; 95% confidence interval .026 < .113 < .199). When testosterone was coded dichotomously (i.e., those who had elevated testosterone above the normal range versus those in the normal range) the partial correlation remained significant ($r_{495} = .095$, *p* = .035, two-tailed; 95% confidence interval .007 < .095 < .181).

When the group was divided by treatment completion or noncompletion, the predictive value (i.e., partial correlation, controlling for age) of serum testosterone in predicting sexual offense recidivism was significant for noncompleters. This was the case whether testosterone was coded continuously ($r_{228} = .168$, *p* = .011, two-tailed; 95% confidence interval .040 < .168 < .291) or dichotomously, ($r_{225} = .174$, *p* = .009, two-tailed; 95% confidence interval .045 < .174 < .297) (Table II).

Remarkably, the significance of testosterone in predicting sexual offense recidivism while controlling for age among those who completed treatment was absent. Again, this was found whether testosterone was coded as a continuous ($r_{267} = .065$, *p* = .285, two-tailed; 95% confidence interval $-.055 < .065 < .183$) or a dichotomous variable ($r_{267} = .013$, *p* = .828, two-tailed; 95% confidence interval $-.107 < .013 < .132$).

Table II. Partial Correlations Between Serum Testosterone and Sexual Recidivism^a after Controlling for Age Among Adult Male Sex Offenders (*N* = 501) Attending In-patient Treatment at the Phoenix Program

	Combined group (<i>n</i> = 501)		Completers (<i>n</i> = 270)		Noncompleters (<i>n</i> = 231)	
	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>
Testosterone ^b (continuous)	.113	.012*	.065	.285	.168	.011*
Testosterone ^c (normal-high)	.095	.035*	.013	.828	.174	.009**

^aBased on mean follow-up of 108.3 months (range: 6.0–188.0 months, *SD* = 48.7 months).

^bTestosterone coded as a continuous variable.

^cTestosterone dichotomously coded 0 = normal range, 1 = higher than 29.9 nmol/L, three noncompleters of treatment dropped from recoding as they fell below the normal range.

p* < .05, two-tailed. *p* < .01, two-tailed.

A review of possible relationships between other endocrine measures and sexual offense recidivism revealed no significant findings. Specifically, correlations with recidivism for completers, noncompleters, and for the pooled group were not significant for LH, FSH, or SHBG.

DISCUSSION

The findings of this study are important for three different reasons. Firstly, the correlation between serum testosterone values and offense severity was statistically significant (i.e., non-null). This lends further support to the hypothesis that testosterone is related to severity of expressed aggression associated with sexual offending. This clinically derived measure specifically assessed aggression during sexual offending as opposed to either dimension in isolation. That is to say, testosterone levels seem to be related not only to sex drive and aggression as separate entities, but also to sexual aggression.

Secondly, serum testosterone level was a more robust predictor of sexual offense recidivism than the authors had anticipated. The correlation was clear and statistically significant for the noncompleters of treatment as well as the pooled sample. To date this measure has not been used in risk prediction algorithms, but may be worth considering in that light. According to recent research undertaken to develop actuarial measures of likelihood to recidivate, static/historic variables account for the greatest degree of variance in predicting reoffense. None of the established risk factors has been shown to be overwhelmingly predictive by themselves, but in combination, some degree of accurate prediction has been demonstrated (Hanson, 1997, 2000). The strength of correlation reported here between testosterone and sexual recidivism ($r_{498} = .113$ for the overall group) while controlling for age, is at least as strong as some of the currently accepted static risk factors (Hanson & Bussière, 1998). In fact, for those who did not complete

treatment, this relationship was more robust ($r_{228} = .168$) and proved to have a larger correlational value than for almost all the static factors reported by Hanson and Bussière (1998; i.e., any stranger victim, early onset, any prior offenses, age, never married, any nonrelated victims, any male victims, diverse sex crimes, and antisocial personality).

Thirdly, and far more importantly from a treatment perspective, the effect of the “static” (or more accurately, less mutable) variable of serum testosterone, is dramatically lessened with treatment. Although the serum testosterone itself is likely not affected by group-therapy treatment per se, the relevance of this variable decreases with that therapy. There is a growing body of research demonstrating that physiological/biological events occur in response to psychotherapeutic treatment (e.g., Brody et al., 2001; Furmark et al., 2002; Goldapple et al., 2004; Wykes et al., 2002). The theoretical possibility does therefore exist, that serum testosterone levels themselves may be influenced by group therapy. To date, studies showing physiological changes because of psychotherapy have not addressed the permanency of those changes. Nevertheless, it is possible that successful treatment diminishes serum testosterone. Another plausible interpretation is that the antisocial effects of testosterone in sex offenders are redirected with treatment into more prosocial noncriminogenic activities. Of course, both of these two possibilities could be correct.

This study, in conjunction with a previous finding that the static variable of prior sex offenses becomes less meaningful in risk prediction after treatment (Studer & Reddon, 1998), reveals the positive effects of some kinds of treatment in mitigating sexual offender recidivism. These types of findings are particularly important in this era of evidence-based practice. This is even more impressive when it can be shown that treatment completers are not selected on the basis of such static factors. If we were inadvertently selecting individuals more likely to succeed, the differences (i.e., higher mean testosterone and more severe offenses) would be expected to be in the other direction. The present findings indicate that both serum testosterone levels and completion of an intensive group psychotherapy treatment program warrant closer evaluation as potentially important factors for inclusion in future actuarial prediction of reoffense among sex offenders.

One limitation to the study is the reliance on commercially available kits and blood assays that were conducted by a third party laboratory. However, all assays were conducted by a single clinical chemistry lab that was the accepted provider for all hospitals and clinics in the region. A second limitation, in retrospect, was that serial serum testosterone testing during and following treatment was not conducted. This would have helped to clarify whether behavioral changes were mediated through reductions in serum testosterone levels.

It is unclear whether all comprehensive and intensive group psychotherapy treatment programs would show a similar result. This poses a possible limitation to the generalizability of these findings. Our program is unique in several ways,

perhaps, primarily in its intensity (i.e., group therapy for 35–40 hr per week over typically 10–13 months). We postulate that consistent behavioral changes, which demonstrate a cognitive, overlay, affective restraint, and personal insight into impulses and drives, could account for the amelioration of the predictive influence on recidivism. Perhaps, after all, Plato was correct when he suggested that reason can rule man's passions.

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REFERENCES

- Aldercreutz, H. (1990). Western diet and western diseases: Some hormonal and biochemical mechanisms and associations. *Scandinavian Journal of Clinical and Laboratory Investigation*, *50*, 3–23.
- Archer, J. (1991). The influence of testosterone on human aggression. *British Journal of Psychology*, *82*, 1–28.
- Archer, J., Biring, S. S., & Wu, F. C. W. (1998). The association between testosterone and aggression among young men: Empirical findings and a meta-analysis. *Aggressive Behavior*, *24*, 411–420.
- Archer, J., Graham-Kevan, N., & Davies, M. (in press). Testosterone and aggression: A reanalysis of Book, Starzyk, and Quinsey's (2001) study. *Aggression and Violent Behavior*.
- Auger, A. P. (2004). Steroid receptor control of reproductive behavior. *Hormones and Behavior*, *45*, 168–172.
- Aylwin, A. S., Clelland, S. R., Kirkby, L. K., Reddon, J. R., Studer, L. H., & Johnston, J. (2000). Sexual offense severity and victim gender preference: A comparison of adolescent and adult sex offenders. *International Journal of Law and Psychiatry*, *23*, 113–124.
- Baulieu, E. E., Alberga, A., Jung, I., Lebeau, M. C., Mercier-Bodard, C., Milgrom, E., et al. (1971). Metabolism and protein binding of sex steroids in target organs: An approach to the mechanism of hormone action. *Recent Progress in Hormone Research*, *27*, 351–412.
- Book, A. S., Starzyk, K. B., & Quinsey, V. L. (2001). The relationship between testosterone and aggression: A meta-analysis. *Aggression and Violent Behavior*, *6*, 579–599.
- Brody, A. L., Saxena, S., Stoessel, P., Gillies, L. A., Fairbanks, L. A., Alborzian, S., et al. (2001). Regional brain metabolic change in patients with major depression treated with either paroxetine or interpersonal therapy: Preliminary findings. *Archives of General Psychiatry*, *58*, 631–640.
- Brooks, J. H., & Reddon, J. R. (1996). Serum testosterone in violent and non-violent young offenders. *Journal of Clinical Psychology*, *52*, 475–483.
- Brown, T. R. (1999). Androgen metabolism and action. In C. Wang (Ed.), *Male reproductive function* (pp. 65–84). Boston: Kluwer.
- Couwenbergs, C., Knussmann, R., & Christiansen, K. (1986). Comparisons of the intra- and inter-individual variation in sex hormone levels of men. *Annals of Human Biology*, *13*, 63–72.
- Conn, P. M., & Crowley, W. F., Jr. (1991). Gonadotropin-releasing hormone and its analogues. *New England Journal of Medicine*, *324*, 93–103.
- Dabbs, J. M., Jr., & Morris, R. (1990). Testosterone, social class, and antisocial behavior in a sample of 4,462 men. *Psychological Science*, *1*, 209–211.
- Ehrenkranz, J., Bliss, E., & Sheard, M. (1974). Plasma testosterone: Correlation with aggressive behavior and social dominance in man. *Psychosomatic Medicine*, *36*, 469–475.

- Elkins, R. (1990). Measurement of free hormones in blood. *Endocrine Reviews*, *11*, 5–46.
- Ellis, L. (1986). Evidence of neuroandrogenic etiology of sex roles from a combined analysis of human, nonhuman primate and nonprimate mammalian studies. *Personality and Individual Differences*, *7*, 519–552.
- Ellis, L., & Nyborg, J. (1992). Racial/ethnic variations in male testosterone levels. *Steroids*, *57*, 72–75.
- Furmark, T., Tillfors, M., Marteinsdottir, I., Fischer, H., Pissioti, A., Langstrom, B., et al. (2002). Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive behavioral therapy. *Archives of General Psychiatry*, *59*, 425–433.
- Gladue, B. A., Boechler, M., & McCaul, K. D. (1989). Hormonal response to competition in human males. *Aggressive Behavior*, *15*, 409–422.
- Goldapple, K., Segal, Z., Garson, C., Lau, M., Bieling, P., Kennedy, S., et al. (2004). Modulation of cortical-limbic pathways in major depression. Treatment specific effects of cognitive behavior therapy. *Archives of General Psychiatry*, *61*, 34–41.
- Griffin, J. E., & Wilson, J. D. (1992). Disorders of the testes and male reproductive tract. In J. D. Wilson & D. W. Foster (Eds.), *Williams textbook of endocrinology* (8th ed., pp. 799–852). London: Saunders.
- Hanson, R. K. (1997). *The development of a brief actuarial risk scale for sexual offense recidivism* (User Report No. 97-04). Ottawa, Ontario, Canada: Department of the Solicitor General of Canada.
- Hanson, R. K. (2000). *Risk assessment*. Beaverton, OR: Association for the Treatment of Sexual Abusers.
- Hanson, R. K., & Bussière, M. T. (1998). Predicting relapse: A meta-analysis of sexual offender recidivism studies. *Journal of Consulting and Clinical Psychology*, *66*, 348–362.
- Harris, J. A. (1999). Review and methodological considerations in research on testosterone and aggression. *Aggression and Violent Behavior*, *4*, 273–291.
- Keller, E. T., Ershler, W. B., & Chang, C. (1996). The androgen receptor: A mediator of diverse responses. *Frontiers in Bioscience*, *1*, 59–71.
- Kraemer, H. C., Becker, H., Brodie, H., Doering, C., Moos, R., & Hamburg, D. (1976). Orgasmic frequency and plasma testosterone levels in normal males. *Archives of Sexual Behavior*, *2*, 125–132.
- Mazur, A., & Booth, A. (1998). Testosterone and dominance in men. *Behavioral and Brain Sciences*, *21*, 353–397.
- Mazur, A., & Lamb, T. (1980). Testosterone, status, and mood in human males. *Hormones and Behavior*, *14*, 236–246.
- McManus, I. C., & Bryden, M. P. (1991). Geschwind's theory of cerebral lateralization: Developing a formal, causal model. *Psychological Bulletin*, *110*, 237–253.
- Mendel, C. M. (1989). The free hormone hypothesis: A physiologically based mathematical model. *Endocrine Reviews*, *10*, 232–274.
- Pardridge, W. (1991). Transport of thyroid hormones into tissues in vivo. In S. Wu (Ed.), *Thyroid hormone metabolism* (pp. 123–143). London: Blackwell.
- Pugeat, M., Garrel, D., Estour, B., Lejeune, H., Kurzer, M. S., Tourniaire, J., et al. (1988). Sex steroid-binding protein in nonendocrine diseases. *Annals of the New York Academy of Sciences*, *538*, 235–247.
- Rada, R., Laws, D., & Kellner, R. (1976). Plasma testosterone levels in the rapist. *Psychosomatic Medicine*, *38*, 257–268.
- Rosner, W. (1990). The functions of corticosteroid-binding globulin and sex-hormone binding globulin: Recent advances. *Endocrine Reviews*, *11*, 80–91.
- Studer, L. H., & Reddon, J. R. (1998). Treatment may change risk prediction. *Sexual Abuse: A Journal of Research and Treatment*, *10*, 175–181.
- Studer, L. H., Reddon, J. R., Roper, V., & Estrada, L. (1996). Phoenix: An in-hospital treatment program for sex offenders. *Journal of Offender Rehabilitation*, *23*, 91–97.
- Studer, L. H., Reddon, J. R., & Siminoski, K. G. (1997). Serum testosterone in adult sex offenders: A comparison between Caucasians and North American Indians. *Journal of Clinical Psychology*, *53*, 375–385.
- Thompson, W. M., Dabbs, J. M., Jr., & Frady, R. L. (1990). Changes in saliva testosterone levels during a 90-day shock incarceration program. *Criminal Justice and Behavior*, *17*, 246–252.

- Tsujimura, A., Matsumiya, K., Matsuoka, Y., Takahashi, T., Koga, M., Iwasa, A., et al. (2003). Bioavailable testosterone with age and erectile dysfunction. *Journal of Urology*, *170*, 2345–2347.
- Wykes, T., Brammer, M., Mellers, J., Bray, P., Reeder, C., Williams, C., et al. (2002). Effects on the brain of a psychological treatment: Cognitive remediation therapy: Functional magnetic resonance imaging in schizophrenia. *British Journal of Psychiatry*, *181*, 144–152.
- Yalom, I. D. (1995). *The theory and practice of group psychotherapy* (4th ed.). New York: Basic Books.