

The Role of Central and Peripheral Hormones in Sexual and Violent Recidivism in Sex Offenders

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Hormonal factors are important in multifactorial theories of sexual offending. The relationship between hormones and aggression in nonhumans is well established, but the putative effect in humans is more complex, and the direction of the effect is usually unclear. In this study, a large sample ($N = 771$) of adult male sex offenders was assessed between 1982 and 1996. Gonadotrophic (follicle-stimulating hormone and luteinizing hormone) and androgen hormone (total and free testosterone; T) levels were assessed at Time 1, along with indicators of sex drive and hostility. Individuals were observed up to 20 years in the community, with an average time at risk of 10.9 years (SD 4.6). Gonadotrophic hormones correlated positively with self-reported hostility and were better predictors of recidivism than was T (area under the curve (AUC), 0.58–0.63). Self-reported hostility emerged as a partial mediator of this relationship between gonadotrophic hormones and recidivism. These results point to a potentially new area of investigation for hormones and sexual aggression.

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Neurobiological factors are considered important in multifactorial theories of sexual offending, and androgenic hormones have been specifically implicated in the elicitation of both sexual and aggressive behaviors.^{1,2} Testosterone (T) is the primary circulating androgen in men. Its production is initiated when gonadotrophin-releasing hormone is secreted from the hypothalamus. T is mostly bound to plasma proteins in the blood, with approximately two percent being in an unbound (free) form. It has been suggested that the free form of T is physiologically the most active,³ but the bound portion may also have effects.⁴ In males, T levels rise in utero and again dramatically at puberty. T has both organizational

and activational effects on physical development, mood, and behavior in males.⁵

Androgens and Aggression

Androgens such as T are related to male aggression across vertebrate species.^{6–8} Animal research studies have consistently shown that aggression appears at the onset of gonadal puberty,^{7,9} is reduced by castration, and can be restored by treatment with exogenous androgens.^{10,11} The ethological evidence suggests that androgens influence aggression in humans.

The relationship between androgens and sexual activity and aggression in humans is more complex than that demonstrated in vertebrate species, because of cognitive, emotional, social, and contextual factors.⁷ In a meta-analysis,¹² the authors examined this question by reviewing 45 studies of human participants (total $n = 9,760$); the results demonstrated a correlation of 0.14 between T and aggression. A reanalysis of these data after correcting for methodological problems, such as the inclusion of overlapping studies, resulted in a lesser correlation,¹³ but a further analysis by the original authors, correcting for new errors in the reanalysis found that the effect was retained ($r = 0.13$).¹⁴

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Androgens and Sexual Behavior

T is a major hormone associated with sexual motivation and behavior.⁵ Low levels of T can result from hypogonadism (primary or secondary) and are associated with decreased sexual interest and function. T replacement therapy can result in demonstrable improvement in the symptoms of hypogonadism.¹⁵ Experimental research with humans and other mammalian species shows that surgical castration or antiandrogen drug treatment reduces sexual interest and behavior, but does not necessarily negate the ability to engage in sexual activities.¹⁶

Androgens and Sexual Offending

Given the relationships between androgens and both aggressive and sexual behavior, many comprehensive theories of sexual offending have incorporated hormonal factors.¹⁷ However, there is surprisingly little evidence of this putative causal role.¹⁸ The results of a castration and antiandrogen study suggest that reducing androgens can decrease sexual recidivism among identified offenders, but it was not a randomized clinical trial.¹⁶ Several studies, despite their methodological limitations, have shown an effect of antiandrogen treatment on libido and sexual behavior.^{19–22}

Several studies have reported that violent sex offenders have higher levels of androgens than do non-violent comparison groups^{23–25} and that levels correlate positively with both prior violence and the severity of sexual aggression in sex offenders.^{25–27} A limitation of these studies, however, is that the direction of any effect is not clear (e.g., whether testosterone preceded or followed sexual aggression). An exception is a recent study, in which Studer et al.⁴ found a modest but statistically significant association between serum T levels and subsequent sexual offending in a sample of 501 adult male sex offenders with a mean follow-up of 8.9 years.

It is unclear why testosterone levels can predict sexual offending years in the future. One possibility is that it increases aggression, including sexual aggression. Another possibility is that it increases the frequency of sexual behavior, which then raises the risk of sexual aggression (as a result of more opportunity, for example). One way of determining which explanation is more apt is to identify mediators of the association between testosterone and sexual offending. For example, finding that the relationship is bet-

ter explained by hostility than by sex drive suggests that the testosterone-aggression link is more relevant, whereas findings emphasizing sex drive as a mediator suggest that the testosterone-sexual behavior link is more relevant.

Gonadotrophins

Testosterone blood levels have a circadian fluctuation, with the highest levels occurring in the early morning. In healthy men, low T results in an increase in hypothalamic gonadotropin-releasing hormone (GnRH) that in turn stimulates an increase in two gonadotropic hormones: follicle-stimulating hormone (FSH) and luteinizing hormone (LH). FSH and LH are peptide hormones that are produced in the anterior pituitary: FSH stimulates the Sertoli cells in the testes and is therefore associated with spermatogenesis and fertility, whereas LH stimulates the Leydig cells in the testes to produce T.

Although there has been considerable research on individual sex hormones in isolation, there have been comparatively fewer studies examining interactions between LH, FSH, and T. One study²⁸ reported a spike in LH following a GnRH injection in men with pedophilia, but not in a group of men who did not have pedophilia. The finding was replicated in a study of men with pedophilia compared with a control group of men with no known paraphilic interests. Compared with the control group, the men with pedophilia showed greater increases in LH (but not FSH) than did the controls after injection of GnRH.²⁹

There have been relatively few studies examining the relationships of FSH and LH with subsequent aggressive and sexual behavior. In an early investigation using a small sample of adult males (N = 28), Mendelson et al.³⁰ found that violent men had higher LH levels than nonviolent men had, as shown in postmortem blood samples. The relationships found for androgens in previous research may have masked a more central role for LH or FSH in androgen regulation.

Present Study

The present study was designed to extend previous research concerning the relationship between gonadotrophic and androgen hormone levels and subsequent long-term sexual and violent recidivism in a large sample of adult male sex offenders. This study

was based on a retrospective chart review of assessments conducted by the Sexual Behaviors Clinic. Distinctive features of this study include a large sample size; a long follow-up period; measurement of both total and free T, in addition to both primary pituitary gonadotrophins (LH and FSH); and examination of hostility and sex drive as potential mediators of any association between hormones and sexual or violent recidivism.

On the basis of previous findings, we hypothesized the following: T, FSH, and LH would correlate positively with the violence of the index offense and the intrusiveness of the index sexual offense, self-reported hostility, and sex drive; T, FSH, and LH would correlate positively with sexual reoffending, even many years in the future; and both hostility and sex drive would partially mediate the relationship between hormones and recidivism.

Method

Participants

The present sample was based on a retrospective chart review of adult males (18 years of age or older at the time of the index offense) who had been convicted of a sexual offense. Only participants who had hormone test results in their medical records were included in the present study.

Participants were 771 adult men who had been assessed between 1982 and 1996 at a university-affiliated forensic sexual behaviors clinic. The majority of the participants were referred via the criminal justice system (e.g., judge or defense, 66%), followed by their treating physician (18%); five percent were self-referred. In the total sample, 278 (36.1%) were classified as intrafamilial offenders against children, 190 (24.6%) were classified as extrafamilial offenders against children, 172 (22.3%) were exhibitionists, 72 (9.3%) were rapists of adult women, and 59 (7.7%) had heterogeneous victim types.

The average age of the sample was 36.7 years (SD 11.9; range, 18–78) and approximately 41 percent of the participants reported having ever been married or having lived in a common-law relationship. Twenty-six percent had charges or convictions for previous sexual offenses, 39 percent had previous violent (including sexual) offenses, and 50 percent had prior criminal offenses of any kind. The majority of the sample was assessed at pretrial (36%), followed by those assessed at presentencing (13%). Approxi-

mately 11 percent were on probation at the time of assessment. Approximately 93 percent of all participants assessed at the clinic consented to their results being used for research purposes. This research study was approved by the Research Ethics Board of the Royal Ottawa Health Care Group.

Measures

Biochemical Assay Methods

Fasting blood samples were generally drawn to assay hormone levels between 8 and 11 a.m., to limit the effect of diurnal variations in circulating T. Total or free T, FSH, and LH were measured using commercially available radioimmunoassay kits at an independent laboratory. All hormone levels were provided by the same laboratory, which has modified its method of estimating hormone levels over the years, according to standard laboratory protocols. In 1994, the clinic transitioned to measuring free T, rather than total T. Thirty-seven participants received tests measuring both free and total T; the remainder of the participants had only either free T (post-1994) or total T (pre-1994).

Hostility

The Buss-Durkee Hostility Inventory³¹ was developed to measure various forms of aggression and hostility and is composed of 75 true-false statements. The measure includes seven subscales (assault, indirect hostility, irritability, negativism, resentment, suspicion, and verbal hostility) and an overall measure of hostility. The measure has been shown to have adequate test-retest reliability for each subscale and overall score and good internal consistency, discriminant validity, and convergent validity.^{32,33} Among sex offenders, BDHI scores for rapists have been significantly higher than those for nonoffending control subjects.^{25,34}

Index Offense Features

Offense features included two behaviorally anchored scales measuring the violence of the index offense and the intrusiveness of the index sexual offense. The violence of the index offense was rated on a 10-point scale: no force or violence, 0; threat of assault with no weapon, 1; threat of assault with a weapon, 2; minor injury with no weapon, 3; minor injury with a weapon, 4; severe beating with no weapon, 5; severe beating with a weapon, 6; potential homicide (the injuries could have caused death without successful medical intervention), 7; homicide, 8;

and homicide with postmortem mutilation, 9. Intrusiveness was rated on a six-point scale: no sexual intrusiveness, 0; verbal threat, 1; attempt, 2; touching, 3; penetration, 4; and sexual assault with excessive violence, 5. These behaviorally anchored scales have shown adequate discriminant validity and predictive validity in previous investigations of sex offenders.^{35,36} Inter-rater reliability for the behaviorally anchored scales was not available, as offense features were rated only by the evaluating psychiatrist.

Sex Drive

Sex drive was assessed with the Derogatis Sexual Functioning Inventory (DSFI) and a quantifiable index of sexual frequency (total sexual outlet (TSO), described later). The DSFI³⁷ is a self-report measure with 10 subscales assessing different dimensions of sexual functioning. Among these scales, sex drive is a composite summary of sexual interest that is expressed across five behavioral domains: sexual intercourse, masturbation, kissing and petting, sexual fantasy, and ideal frequency of sexual intercourse. The scale has shown adequate internal consistency and test-retest reliability.^{37,38} Tang *et al.*³⁹ reported marginal internal consistency coefficients for the sex drive subscale ($\alpha = 0.61$).

TSO is defined as the number of orgasms achieved through any combination of methods (e.g., intercourse, masturbation) during a specific week.⁴⁰ Several large-scale epidemiological studies have utilized TSO as a behavioral indicator of sex drive.^{41,42}

Recidivism

Recidivism data were obtained in 2002 from a national database (CPIC; Canadian Police Information Centre) of criminal arrests and convictions maintained by the Royal Canadian Mounted Police. Recidivism was defined as a new offense (charge or conviction) that occurred after the index conviction, regardless of when these offenses occurred during the follow-up period. In contrast, analyses involving fixed follow-up periods (see results below) included only the first incident of recidivism, rather than all incidents of recidivism.

The follow-up period and opportunity to reoffend began at the latest of three possible dates pertaining to the index offense: date of conviction, date of assessment, or date of release if incarcerated. Eleven participants died during the follow-up period; their opportunity to reoffend ended on the day

of death, if this date was reported in the CPIC records.

Recidivism outcomes included sexual recidivism, defined as any charge or conviction for a sexual offense, whether it involved physical contact with a victim (sexual assault) or not (indecent exposure, i.e., exhibitionism), and violent (including sexual) recidivism, defined as any charge or conviction for a nonsexually violent or sexual offense. The categories are not mutually exclusive and were statistically dependent ($\phi = 0.75$), such that all participants who sexually reoffended were coded as violent recidivists, and most (63%) individuals who reoffended violently were coded as sexual recidivists.

This method of coding recidivism is consistent with some prior recidivism studies.^{32,43} Sexual recidivism includes noncontact offenses that may have been a prelude to contact sexual offending (e.g., the Canadian Criminal Code includes an offense (invitation to sexual touching) that results from approaching a child sexually, even if no physical contact has taken place); violent recidivism captures all violent offenses, including sexual offenses that were pleaded down to nonsexually violent charges (e.g., an attempted rape that resulted in a conviction for assault); or sexually motivated offenses that resulted in nonsexually violent charges (e.g., sexually motivated homicide that was charged as first-degree murder⁴⁴).

Statistical Analyses

Several effect size indicators were reported in the present investigation. The simplest indicator representing the magnitude of effect in the present study was a correlation coefficient (Pearson's r). It is generally considered that correlation values of 0.1, 0.3, and 0.5, represent small, medium, and large effects, respectively. Post hoc comparisons on the continuous variables utilized Cohen's d ; corresponding values for small, medium, and large effects are 0.2, 0.5, and 0.8.⁴⁵

The area under the curve (AUC) of the receiver operating characteristic (ROC) was used to examine the predictive accuracy of the hormonal variables, as this statistic is less affected by recidivism base rates or selection ratios.⁴⁶ AUC values, which can range from 0 to 1, can be interpreted as the probability that a randomly selected recidivist has higher hormonal levels than a randomly selected nonrecidivist. A value of 1 represents perfect prediction, while a value of 0.5 indicates chance prediction. For descriptive

purposes, minimum AUC values of 0.56, 0.64, and 0.71 are described as small, medium, and large, respectively.⁴⁷ To detect significant differences between two AUC values, we determined the critical ratio *z*, the ratio of a difference score to the standard error of the difference score, using formulas provided by Hanley and McNeil.⁴⁸

Several mediation analyses were conducted with LH and FSH as independent variables; sexual recidivism as well as violent (including sexual) recidivism served as the dependent variables. Initially, three potential mediators were included, represented by the measures for sex drive, total sexual outlet, and self-reported hostility. The procedure used for the mediation analyses is based on the method described by Baron and Kenny.⁴⁹ However, while the regression equations used are based on Baron and Kenny, modifications and additional equations were used to account for multiple mediators, and transformations were used, since recidivism was treated as a dichotomous variable. The use of transformations is discussed in MacKinnon and Dwyer⁵⁰ and Winship and Mare.⁵¹ An example of SAS (Statistical Analysis Software) code for a three-mediator model given by Tabora et al.⁵² was studied before the writing of the code used here, and all regressions were performed in SAS. The presence or absence of mediation was assessed by application of the criteria given by Baron and Kenny.⁴⁹ Point estimates of the indirect effect were obtained by using the modified product-of-coefficients method discussed in MacKinnon and Dwyer,⁵⁰ and corresponding asymmetric 95 percent

confidence intervals were obtained using the program PRODCLIN.⁵³ This procedure and the resulting estimates are described as the *M*-test of indirect effects by Williams and MacKinnon.⁵⁴

Results

The follow-up period ranged from 1 to 20 years, with a mean of 10.9 years (SD 4.6). The percentage of men who reoffended sexually and violently over the duration of the follow-up period was 17.8 and 28 percent, respectively.

The mean levels of total T and free T in the sample were 21.08 nmol/L (SD 7.17) and 50.48 pmol/L (SD 17.38), respectively. The mean levels of FSH and LH were 8.19 IU/L (SD 6.95) and 9.65 IU/L (SD 6.99). All of these values are within normal limits.⁵² Total T, free T, and FSH levels did not distinguish any type of sex offender. However, there was a significant main effect for LH level and type of sex offender ($F(4,714) = 2.58, p < .05$). Tukey's least significant difference post hoc analyses revealed that intrafamilial child molesters had lower LH levels than did extrafamilial child molesters ($d = 0.21$; 95% CI, (0.02 to 0.40) and exhibitionists ($d = 0.28$; 95% CI, 0.08 to 0.48).

Pearson correlation coefficients were calculated to examine the relationships among hormone levels, self-reported hostility and aggression, the intrusiveness of the index sexual offense, the violence of the index offense, and indicators of sex drive. Correlation coefficients are displayed in Table 1; 95 percent

Table 1 Intercorrelation Matrix for Hormone Levels, Hostility/Aggression, and Hypersexuality

	T-test	F-test	FSH	LH	BDHI	Violence of Index Offense	Sexual Intrusiveness	Sex Drive	TSO
T-test (nmol/L)	—	0.21 (37)	0.08 (607)	0.25* (622)	-0.01 (618)	0.08 (566)	0.03 (531)	0.05 (616)	0.04 (611)
F-test (pmol/L)		—	-0.14 (114)	-0.06 (112)	0.18 (116)	0.20† (104)	-0.06 (109)	0.24† (114)	0.18 (112)
FSH (IU/L)			—	0.66* (681)	0.12‡ (692)	0.02 (631)	0.06 (604)	0.03 (690)	0.04 (687)
LH (IU/L)				—	0.20* (690)	0.13‡ (634)	0.13‡ (599)	0.02 (688)	0.07 (683)
BDHI					—	0.17‡ (637)	0.09* (606)	0.23* (726)	0.19* (721)
Violence of index offense						—	0.37† (602)	0.14‡ (635)	0.01 (633)
Sexual intrusiveness							—	-0.01 (604)	-0.08† (603)
Sex drive (DSFI)								—	0.40* (721)
TSO									—

Sample sizes are included in parentheses and vary because of differing numbers of subjects with missing information. T-test, total testosterone; F-test, free testosterone; FSH, follicle stimulating hormone; LH, luteinizing hormone; BDHI, Buss-Durkee hostility inventory; DSFI, Derogatis sexual functioning inventory; TSO, total sexual outlet.

* $p < .001$.

† $p < .05$.

‡ $p < .01$.

Table 2 Predictive Accuracy of Gonadotrophic and Androgen Hormone Levels

Hormone	n	Type of Recidivism			
		Sexual		Violent (Including Sexual)	
		AUC	95% CI	AUC	95% CI
Total T* (nmol/L)	649	0.50	0.44–0.56	0.52	0.47–0.57
Free T† (pmol/L)	120	0.55	0.40–0.70	0.53	0.40–0.67
FSH (IU/L)	723	0.59*	0.54–0.64	0.58*	0.53–0.62
LH (IU/L)	719	0.63†	0.58–0.68	0.63†	0.58–0.67

Total T, total testosterone; free T, free testosterone; FSH, follicle stimulating hormone; LH, luteinizing hormone; CI, confidence interval.

* $p < .01$.

† $p < .001$.

confidence limits for r were determined using Fisher's r - z transformation and are shown in the text in parentheses. Only correlations significant at the $p < .01$ level are reported in the text to correct for Type I errors resulting from numerous associations being examined in the analysis.

Total T correlated significantly and positively with LH (0.18, 0.32), and FSH correlated significantly and positively with LH (0.62, 0.70) and self-reported hostility (0.05, 0.19). LH associated positively with self-reported hostility (0.13, 0.27), the violence of the index offense (0.05, 0.21), and the intrusiveness of the index sexual offense (0.05, 0.21). The violence of the index offense associated positively and significantly with the intrusiveness of the index sexual offense (0.30, 0.44). Self-reported hostility associated positively with the violence of the index offense (0.09, 0.24). Finally, with regard to hypersexuality, the DSFI positively and significantly correlated with TSO (0.33, 0.46), self-reported hostility (0.16, 0.30), and the violence of the index offense (0.06, 0.22). TSO was also significantly associated with self-reported hostility (0.12, 0.26).

Predictive Validity

Table 2 presents the AUC values and 95 percent confidence intervals for the hormone levels in relation to sexual and violent (including sexual) recidivism. Results indicated that LH and FSH were significantly associated with recidivism outcomes, whereas total T and free T were unrelated to recidivism. T levels were unrelated to recidivism, even after we categorized individuals into comparative degrees of low versus high androgen levels.

When comparing ROC indices using the formula provided by Hanley and McNeil,⁴⁸ LH was signifi-

cantly more accurate than total T in predicting sexual recidivism ($n = 622$; $z = 3.47$) and violent recidivism ($n = 622$; $z = 3.46$). LH was also more accurate than FSH in predicting violent recidivism ($n = 681$; $z = 2.87$). Finally, FSH was more accurate in predicting sexual recidivism when compared with total T ($n = 607$; $z = 2.20$).

It has been suggested that clinicians may be particularly concerned with assessing risk within relatively short, set periods.^{55,56} Predictive accuracy can also be evaluated by using fixed follow-up periods; this method creates an equal length of follow-up for each offender. We analyzed recidivism data as it pertains to two- and five-year fixed follow-up periods. We considered only offenders who had at least two years or five years of opportunity to reoffend (depending on the specific analysis), with recidivists being those individuals who reoffended within that specified period. Individuals who reoffended after the relevant interval were counted as nonrecidivists.

When a fixed two-year follow-up period was used, the AUC value for LH in predicting violent (including sexual) recidivism was slightly smaller, but remained statistically significant ($n = 662$; ROC = 0.61; 95% CI, 0.53–0.69). No other significant associations were evident. When a fixed five-year follow-up period was used, the AUC for LH in predicting sexual recidivism was slightly higher than the rate produced when an open-ended time frame was used and it was statistically significant ($n = 663$; ROC = 0.66; 95% CI, 0.59–0.73).

Putative Mediators Between Hormones and Recidivism

We evaluated whether sex drive, TSO, and self-reported hostility mediated the effects of LH and FSH on long-term recidivism. With regard to violent (including sexual) recidivism, we found that the relationship of LH with recidivism was mediated by self-reported hostility. Results from a logistic regression showed that LH was a significant predictor of violent recidivism ($p < .0001$). Next, in OLS (ordinary least squares) regressions, LH was significant in predicting total sexual outlet ($p = .04$) and self-reported hostility ($p < .0001$), but not sex drive ($p = .62$). Finally, in a logistic regression with the explanatory and all mediating variables, the only significant potential mediator was self-reported hostility ($p < .0001$); also, as required with potential mediators in the model, the absolute value of the estimated effect

of LH decreased, from 0.047 to 0.038. Concluding that self-reported hostility was the only mediator, regression analyses were conducted again with only self-reported hostility included as a mediator. Using these results yielded a point estimate of 0.0065 and a 95 percent confidence interval of 0.0033 to 0.0104 for the indirect effect of LH.

In a similar analysis for FSH, results from a logistic regression showed that FSH approached significance in the prediction of violent recidivism ($p = .0507$). In OLS regressions, FSH was not significant in predicting sex drive ($p = .48$) or total sexual outlet ($p = .24$), but it was significant in predicting self-reported hostility ($p = .003$). In the final logistic regression, self-reported hostility was the only potential mediator that was significant ($p < .0001$), and the FSH coefficient estimate for this regression was .016, compared with 0.022 for the regression with FSH only. Again, we concluded that self-reported hostility was the only mediator, and an appropriate second set of regression equations was obtained. From the results, the point estimate of the indirect effect size was 0.0043 and the 95 percent confidence interval produced by PRODCLIN was 0.0016 to 0.0077. FSH was no longer significant in the model that included self-reported hostility ($p = .151$). The terms perfect and complete mediation are used in the literature⁴⁹ to refer to situations in which controlling for the mediator results in loss of significance of the effect of the explanatory variables. However, given the borderline p value for FSH in the initial regression of violent recidivism, the interpretation of the change observed here is less clear.

Finally, we were interested in examining the potential mediators of sexual recidivism. We found that the relationship of LH with sexual recidivism was also mediated by self-reported hostility. Results of a logistic regression showed that LH was a significant predictor of sexual recidivism ($p = .0002$). Next, in OLS regression, LH was significant in predicting the potential mediator ($p < .0001$). Finally, in a logistic regression of sexual recidivism against both LH and self-reported hostility, the latter measure was significant ($p = .0052$); also, LH remained significant ($p = .0025$) and, as required, the absolute value of the coefficient estimate for LH decreased, from 0.0453 to 0.039. Thus, self-reported hostility, as captured by the BDHI, is a partial mediator of the effect of LH on sexual recidivism. The point estimate of the indirect effect size was 0.0029, and

the 95 percent confidence interval obtained with PRODCLIN was 0.0008 to 0.0053. Application of the M-test indicates that the mediated effect was significant.

In similar analysis with FSH as the independent variable, results of a logistic regression showed that this hormone was a significant predictor of sexual recidivism ($p = .0324$). Also, in an OLS regression of self-reported hostility on FSH, FSH was significant in predicting the potential mediator ($p = .0011$). Finally, in a logistic regression of sexual recidivism against both FSH and the potential mediator, self-reported hostility was significant ($p = .0014$); FSH was no longer significant ($p = .0693$), and the absolute value of the FSH coefficient estimate decreased from 0.0259 to 0.0232, with adjustment for the mediator. These results indicate the presence of complete mediation by self-reported hostility of the effect of FSH on sexual recidivism. The point estimate of the indirect effect size was 0.0031, and the 95 percent confidence interval produced by PRODCLIN was 0.0009 to 0.0061. Again, application of the M-test indicates that the mediated effect is significant.

Discussion

In the present study, we were primarily interested in the associations of androgen and gonadotrophin levels with self-reported hostility and sex drive, offense features, and long-term recidivism in a sample of male sex offenders. Our study was unique, in that we assessed several hormones and putative mediators and observed the individuals for up to 20 years after release into the community. This study represents, to our knowledge, one of the longest follow-up studies of sex offenders in the hormones literature.

We first hypothesized that androgens, particularly testosterone, would correlate with sex drive, hostility, the violence of the index offense, and the intrusiveness of the index sexual offense. Of the sex hormones assayed in this study, only free T exhibited significant associations (with the violence of the index offense and with self-reported sex drive) but these effect sizes failed to meet our more conservative approach to account for Type I error. Nevertheless, these findings suggest that total T is not sufficiently sensitive, which is consistent with the view that most total T is biologically inactive. LH and FSH, in contrast, correlated significantly and positively with self-reported hostility, and LH was associated with both the vio-

lence of the index offense and the intrusiveness of the index sexual offense.

Given the fact that androgens correlate with subsequent recidivism in sex offenders,⁴ we also hypothesized that androgen and gonadotrophin levels would correlate positively with long-term sexual and violent reoffending in our sample. This hypothesis was partially supported: only FSH and LH, but not T, were associated with long-term recidivism. Although some studies have demonstrated a positive association between peripheral hormones and sexual aggression,³⁰ most studies⁴ have shown that T is a more sensitive marker for subsequent aggression.

It was surprising that the gonadotrophins assayed in the present sample were better predictors of long-term sexual or violent recidivism than were the androgens. It is possible that, because LH has a narrower normal range and is a precursor hormone, it may be a more sensitive marker of neuroendocrine functioning than T. This does not explain why T is usually found to be a more sensitive marker for subsequent aggression. Another possibility is that some men have a breakdown in the mechanisms that regulate LH levels. This phenomenon most likely results in a failure of the hypothalamic pituitary axis to downregulate production of LH when T levels rise, and it may cause sex offenders to show elevated LH and T levels or to show elevated LH levels, independent of their T levels. The results of this study are consistent with the second interpretation. Studies to replicate this finding and to further investigate the reasons for the phenomenon are recommended.

We also demonstrated that self-reported hostility was an important mediator between LH (and possibly FSH) and sexual and violent recidivism, suggesting that the link between hormones and aggression is more germane than the link between hormones and sexual behavior. Comprehensive theories of sexual offending^{2,57} include numerous distal and proximal factors that interact in both a cumulative and synergistic manner to increase the likelihood of sexual aggression. The confluence model⁵⁷ highlights the role of hostility and other negative aspects of masculinity as partial contributors to sexual aggression against peers or adults.⁵⁷ Indeed, the fact that the sex hormone levels (total T, free T, LH, and FSH) in the present study were all within normal limits supports the multifactorial nature of sexual aggression. Our results do not support the idea that abnormal hormone levels explain sexual offending; instead, rela-

tively high levels of LH and FSH must interact with other factors to increase the likelihood of sexual aggression.

Limitations

A major limitation of this study is its retrospective design. This drawback is typical of sex offender studies in which long-term recidivism is one of the primary outcome measures. We could not control for extraneous factors, such as the effects of psychological or pharmacological treatment during the follow-up period as well as we might have been able to in a prospective design. Another problem with our retrospective design is that we were unable to account for hormone levels or our putative mediators after the initial assessment (i.e., at any point during the follow-up period). A second limitation pertains to the lack of a control group with which to compare our samples' hormone levels. We were therefore unable to compare the levels obtained in this sample with clinically meaningful groups, such as community participants and other offender groups (e.g., non-sexually violent offenders). The laboratory results were reported relative to test norms, however. Finally, there was a substantial portion of missing data across the variables that primarily resulted from the lengthy period over which assessments were conducted, the transition of the clinic to measuring free T during the study period, and the retrospective nature of the study.

Future Directions

The results of this study suggest that T (total or free) is not related to risk of sexual reoffense. In contrast, LH correlated significantly with sexual and violent recidivism. This result requires replication, however, as the relationship was relatively small, and this is the first study we are aware of to show that LH rather than T can predict long-term recidivism. If confirmed, this finding suggests a new area of investigation in the neurobiology of sexual offending. One possibility is that sexual offending, at least among some individuals, is related to disruption in the modulation of LH or the interactions between LH and T. This hypothesis could be further explored through clinical laboratory studies in which variation in both LH and T is tracked prospectively. It would be valuable for our theoretical explanations of sexual offending if this work were integrated with animal and human work on the effects of LH on aggressive

and sexual behavior. The animal literature is much less clear about the role of LH in these behavioral domains, compared with what is known about T.

Our main finding may also be relevant to understanding the effects of different drugs on sexual offending. Cyproterone acetate (Androcur) blocks intracellular testosterone throughout the body's receptors and thereby decreases testosterone's effects (but with feedback effects on LH secretion), as well as reducing total testosterone. Leuprolide acetate (Lupron) is a GnRH agonist that paradoxically inhibits LH and FSH release and thereby reduces T. Perhaps the key target is LH production rather than T production, and thus drugs that selectively target LH may produce larger effects than those that selectively target T.

An interesting question to examine is whether hormone levels can add to the moderate predictive accuracy provided by existing actuarial risk measures for sex offenders. Seto⁵⁸ suggested that only information from new risk factors or methods different from traditional reviews of personal history can add to the prediction provided by established measures. Although the correlation between LH and recidivism is small, it may still represent unique predictive variance because it is a novel risk factor obtained through a novel method (laboratory assay). We were not able to test this idea in the current study because we did not have actuarial risk scores for all the participants. This may be an interesting avenue for future research.

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References

1. Kafka MP: The monoamine hypothesis for the pathophysiology of paraphilic disorders: an update. *Ann N Y Acad Sci* 989:86–94, 2003
2. Ward T, Beech A: An integrated theory of sexual offending. *Aggress Violent Behav* 11:44–63, 2006
3. Mendel CM: The free hormone hypothesis: a physiologically based mathematical model. *Endocr Rev* 10:232–74, 1989
4. Studer LH, Aylwin AS, Reddon JR: Testosterone, sexual offence recidivism, and treatment effect among adult male sex offenders. *Sex Abuse* 17:171–81, 2005
5. Grubin D: Medical models and interventions in sexual deviance. Edited by Laws DR, O'Donohue WT. *Sexual Deviance: Theory, Assessment, and Treatment* (ed 2). New York: The Guildford Press, 2008, pp 594–610
6. Archer J: *The Behavioural Biology of Aggression*. Cambridge, UK: Cambridge University Press, 1988

7. Archer J: The influence of testosterone on human aggression. *Br J Psychol* 82:1–28, 1991
8. Scotti M-A, Belén J, Jackson J, et al: The role of androgens in the mediation of seasonal territorial aggression in male Siberian hamsters (*Phodopus sungorus*). *Physiol Behav* 95:633–40, 2008
9. Beatty WW: Gonadal hormones and sex differences in nonreproductive behaviors, in *Handbook of Neurobiology* (vol 11). Edited by Gerall AA, Moltz H, Ward IL. New York: Plenum Press, 1992, pp 85–128
10. Harris JA: Review and methodological considerations in research on testosterone and aggression. *Aggress Violent Behav* 4:273–91, 1999
11. Vom Saal F: Models of early hormonal effects on intrasex aggression in mice, in *Hormones and Aggressive Behavior*. Edited by Svare B. New York: Plenum, 1983, pp 197–222
12. Book AS, Starzyk KB, Quinsey VL: The relationship between testosterone and aggression: a meta-analysis. *Aggress Violent Behav* 6:579–99, 2001
13. Archer J, Graham-Kevan N, Davies M: Testosterone and aggression: a reanalysis of Book, Starzyk, and Quinsey's (2001) study. *Aggress Violent Behav* 10:241–61, 2005
14. Book AS, Quinsey VL: Re-examining the issues: a response to Archer et al. *Aggress Violent Behav* 10:637–46, 2005
15. Rizvi SJ, Kennedy SH, Ravindran LN, et al: The relationship between testosterone and sexual function in depressed and healthy men. *J Sex Med* 7:816–25, 2010
16. Gijs I, Gooren L: Hormonal and psychopharmacological interventions in the treatment of paraphilias: an update. *J Sex Res* 33:273–90, 1996
17. Ward T, Polaschek DLL, Beech AR: *Theories of Sexual Offending*. West Sussex, UK: John Wiley & Sons, Ltd., 2006
18. Fedoroff JP, Moran B: Myths and misconceptions about sex offenders. *Can J Hum Sex* 6:263–76, 1997
19. Fedoroff JP, Wisner-Carlson R, Dean S, et al: Metyroxyprogesterone acetate in the treatment of paraphilic sexual disorders: rate or relapse in paraphilic men treated in psychotherapy for at least five years with or without metoxyprogesterone acetate. *J Offender Rehabil* 18:109–23, 1992
20. Hucker S, Langevin R, Bain J: A double-blind trial of sex drive reducing medication in pedophiles. *Ann Sex Res* 1:227–42, 1988
21. Bradford JM: The treatment of sexual deviation using a pharmacological approach. *J Sex Res* 37:248–57, 2000
22. Maletzky BM, Tolan A, McFarland B: The Oregon Depo-Provera Program: a five year follow up. *Sex Abuse* 18:303–16, 2006
23. Brooks J, Reddon J: Serum testosterone in violent and nonviolent young offenders. *J Clin Psychol* 52:475–83, 1996
24. Giotakos O, Markianos M, Vaidakis N, et al: Aggression, impulsivity, plasma sex hormones, and biogenic amine turnover in a forensic population of rapists. *J Sex Marital Ther* 29:215–25, 2003
25. Rada RT, Laws DR, Kellner R: Plasma testosterone levels in the rapist. *Psychosom Med* 38:257–68, 1976
26. Dabbs J, Carr T, Frady R, et al: Testosterone, crime, and misbehavior among 692 male prison inmates. *Pers Individ Diff* 18:627–33, 1995
27. Kreuz L, Rose R: Assessment of aggressive behavior and plasma testosterone in a young criminal population. *Psychosom Med* 34:321–32, 1972
28. Gaffney GR, Berlin FS: Is there a hypothalamic-pituitary-gonadal dysfunction in paedophilia?—a pilot study. *Br J Psychiatry* 145: 657–60, 1984
29. Bain J, Langevin R, Hucker S, et al: Sex hormones in pedophiles, I: baseline values of six hormones; II: the gonadotropin releasing hormone test. *Ann Sex Res* 1:443–54, 1988

30. Mendelson JH, Dietz PE, Ellingboe J: Postmortem plasma luteinizing hormone levels and antemortem violence. *Pharmacol Biochem Behav* 17:1–3, 1982
31. Buss AH, Durkee A: An inventory for assessing different kinds of hostility. *J Consult Psychol* 21:343–49, 1957
32. Kingston DA: The offence progression in sexual offenders: an examination of the self-regulation model of the offence process. Unpublished doctoral dissertation, University of Ottawa, Ottawa, Ontario Canada, 2010
33. Tremblay PF, Ewart LA: The Buss and Perry aggression questionnaire and its relations to values, the Big Five, provoking hypothetical situations, alcohol consumption patterns, and alcohol expectancies. *Pers Indiv Differ* 38:337–46, 2005
34. Firestone P, Bradford JM, McCoy M, et al: Recidivism factors in convicted rapists. *J Am Acad Psychiatry Law* 26:185–200, 1998
35. Kingston DA, Firestone P, Moulden HM, et al: The utility of the diagnosis of pedophilia: a comparison of various classification procedures. *Arch Sexual Behav* 36:423–36, 2007
36. Kingston DA, Seto MC, Firestone P, et al: Comparing indicators of sexual sadism as predictors of recidivism among sexual offenders. *J Consult Clin Psychol* 78:574–84, 2010
37. Derogatis LR: Psychological assessment of psychosexual functioning. *Psychiatr Clin North Am* 3:113–31, 1980
38. Cooper J, Cernovsky ZZ, Colussi K: Some clinical and psychometric characteristics of primary and secondary premature ejaculation. *J Sex Marital Ther* 19:276–88, 1993
39. Tang CS, Lai FD, Phil M, et al: Assessment of sexual functioning for Chinese college students. *Arch Sexual Behav* 26:79–90, 1997
40. Kinsey AC, Pomeroy WB, Martin CE: *Sexual Behavior in the Human Male*. Philadelphia: W. B. Saunders, 1948
41. Atwood JD, Gagnon J: Masturbatory behavior in college youth. *J Sex Educ Ther* 13:35–42, 1987
42. Laumann EO, Gagnon JH, Michael RT, et al: *The Social Organization of Sexuality: Sexual Practices in the United States*. Chicago: University of Chicago, 1994
43. Kingston DA, Fedoroff P, Firestone P, et al: Pornography use and sexual aggression: the impact of frequency and type of pornography use on recidivism among sexual offenders. *Aggress Behav* 34:341–51, 2008
44. Rice ME, Harris GT, Lang C, et al: Violent sex offenses: how are they best measured from official records? *Law Hum Behav* 30:525–41, 2006
45. Cohen J: A power primer. *Psychol Bull* 112:155–9, 1992
46. Swets JA: Indices of discrimination or diagnostic accuracy: their ROCs and implied models. *Psychol Bull* 99:100–17, 1986
47. Rice ME, Harris GT: Comparing effect sizes in follow-up studies: ROC area, Cohen's *d* and *r*. *Law Hum Behav* 29:615–20, 2005
48. Hanley JA, McNeil BJ: A method of comparing the areas under receiver operating characteristic curves derived from the same cases. *Radiology* 148:839–43, 1983
49. Baron RM, Kenny DA: The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol* 51:1173–82, 1986
50. MacKinnon DP, Dwyer JH: Estimating mediated effects in prevention studies. *Eval Res* 17:144–58, 1993
51. Winship C, Mare RD: Structural equations and path analysis for discrete data. *Am J Sociol* 89:54–110, 1983
52. Taborga DP, Cheong JW, MacKinnon DP: Technical Assistance Report: Mediation Analysis. Research in Prevention Laboratory. National Institute on Drug Abuse Grant 5 R01 DA09757-04. October 18, 2000. Available at http://www.public.asu.edu/~davidpm/ripl/Mediation_Analysis.PDF. Accessed October 10, 2012
53. MacKinnon DP, Fritz MS, Williams J, et al: Distribution of the product confidence limits for the indirect effect: program PRODCLIN. *Behav Res Methods* 39:384–9, 2007
54. Williams J, MacKinnon DP: Resampling and distribution of product methods for testing indirect effects in complex models. *Struct Equ Modeling* 15:23–51, 2008
55. Bengtson S, Långström N: Unguided clinical and actuarial assessment of re-offending risk: a direct comparison with sex offenders in Denmark. *Sex Abuse* 19:135–53, 2007
56. Harris GT, Rice ME, Quinsey VL, et al: A multisite comparison of actuarial risk instruments for sex offenders. *Psychol Assess* 15: 413–25, 2003
57. Malamuth NM: Criminal and noncriminal sexual aggressors: integrating psychopathy in a hierarchical-mediational confluence model, in *Sexually Coercive Behavior: Understanding and Management*. Edited by Prentky RA, Janus ES, Seto MC. New York: Annals of the New York Academy of Sciences, 2003, pp 35–58
58. Seto MC: Is more better?—combining actuarial risk scales to predict recidivism among adult sex offenders. *Psychol Assess* 17: 156–67, 2005