

Sex and Context: Hormones and Primate Sexual Motivation

Kim Wallen

*Department of Psychology and The Yerkes Regional Primate Research Center,
Emory University, Atlanta, Georgia 30322*

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Gonadal hormones regulate the ability to copulate in most mammalian species, but not in primates because copulatory ability has been emancipated from hormonal control. Instead, gonadal hormones primarily influence sexual motivation. This separation of mating ability from hormonally modulated mating interest allows social experience and context to powerfully influence the expression of sexual behavior in nonhuman primates, both developmentally and in adulthood. For example, male rhesus monkeys mount males and females equally as juveniles, but mount females almost exclusively as adults. Having ejaculated with a female better predicted this transition to female mounting partners than did increased pubertal testosterone (T). It is proposed that increased pubertal T stimulates male sexual motivation, increasing the male's probability of sexual experience with females, ultimately producing a sexual preference for females. Eliminating T in adulthood reduces male sexual motivation in both humans and rhesus monkeys, but does not eliminate the capacity to engage in sex. In male rhesus monkeys the effects of reduced androgens on sexual behavior vary with social status and sexual experience. Human sexual behavior also varies with hormonal state, social context, and cultural conventions. Ovarian hormones influence female sexual desire, but the specific sexual behaviors engaged in are affected by perceived pregnancy risk, suggesting that cognition plays an important role in human sexual behavior. How the physical capacity to mate became emancipated from hormonal regulation in primates is not understood. This emancipation, however, increases the importance of motivational systems and results in primate sexual behavior being strongly influenced by social context.

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In most mammalian species gonadal hormones regulate both the ability to copulate and interest in cop-

ulating (Wallen, 1990). Females from a variety of species, such as the guinea pig (Young, 1937), have an imperforate vagina, except when they are fertile, limiting copulation to a small portion of their ovarian cycle. Similarly, females of all laboratory rodent species display an arching of the back, lordosis, which is under ovarian hormonal control and which serves to make the female's vagina accessible to the male's penis (Diakow, 1974; Pfaff, Diakow, Montgomery and Jenkins, 1978). Without this hormonally regulated spinal reflex, mating in rodents would not be physically possible. Similarly, many male mammals require gonadal steroids to produce a penile erection sufficiently rigid for sexual intercourse. Furthermore, in some species, gonadal androgens maintain sensory papillae on the glans penis that are required for the male to produce an erection and ejaculate (Beach and Levinson, 1950; Phoenix, Copenhaver, and Brenner, 1976). Although there has been no systematic investigation of the species distribution of these hormonally regulated physical adaptations essential to sexual performance, they have been reported in almost every nonprimate species studied. Such adaptations both assure that mating behavior is tightly coupled to fertility and limit sexual behavior solely to reproductive contexts. In contrast, in most species of primate, including humans, the capacity to copulate has been emancipated from hormonal control (Miller, 1931; Wallen, 1990). The exception is the prosimian primates, the lemurs, galagos, and lorises, where many species have vaginal membranes that disappear only when the female is fertile (Doyle, 1974). In all other primates there is no evidence of hormonally regulated physical adaptations that regulate the capacity to engage in sexual intercourse. Female primates display no behavior analogous to lordosis and male primates, including humans, are capable of producing complete erections

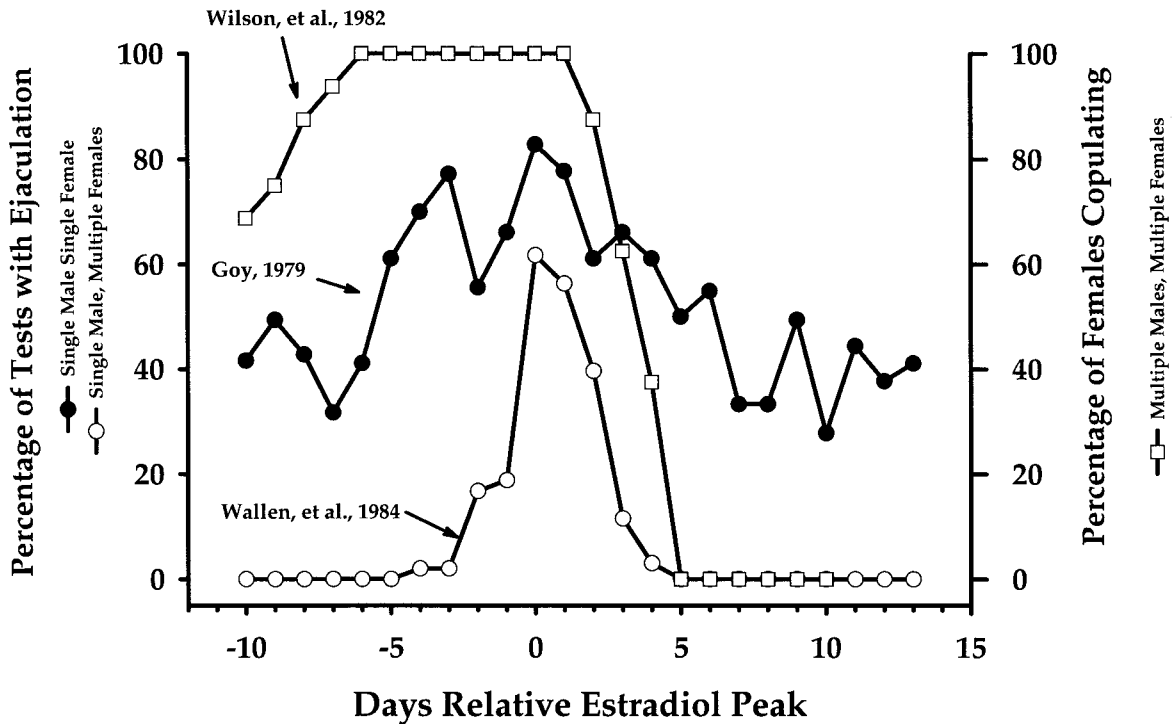


FIG. 1. The occurrence of copulation in rhesus monkeys studied under three different social contexts. Sexual behavior varied with the female's ovarian cycle, being highest around the time of the estradiol peak, but the specific relationship varied with social context. When tested in pairs without other conspecifics present, sexual behavior showed the weakest relationship to the female's cycle (figure adapted using data from Goy, 1979; Wallen *et al.*, 1984; and Wilson *et al.*, 1982).

in the absence of measurable levels of androgen (Bancroft and Wu, 1983; Kwan *et al.*, 1983). Instead, gonadal hormones primarily influence the motivation to copulate (Wallen, 1990, 1995, 1999). This separation of the ability to mate from sexual motivation allows social experience and social context to powerfully influence the expression of sexual behavior in nonhuman primates, both developmentally and in adulthood.

SOCIAL CONTEXT AND MONKEY SEXUAL BEHAVIOR

The relationship between social context and the influence of the female's ovarian cycle on sexual behavior is illustrated in Fig. 1. The occurrence of mating in relation to the female's midcycle estradiol peak varied when observed under three different social contexts: a single male and a single female (Goy, 1979), a group consisting of a single male with multiple females (Wallen *et al.*, 1984), and a group with multiple males and females (Wilson, Gordon, and Collins, 1982). When a single male and female were tested together in

a small laboratory cage on every day of the female's ovarian cycle, some mating occurred on every day, with an elevation around the time of ovulation. In contrast, when multiple females were present with the male in a larger testing area, mating was limited to the fertile period of the female's cycle. When multiple males and females were present, much more mating occurred during the follicular phase of the female's cycle, but completely ceased during the luteal phase. Thus the female's hormonal condition affected the occurrence of sexual behavior more profoundly under multiple than single female conditions. This relationship appears to be a consequence of the social structure of the testing situation and not other methodological differences between the studies. For example, the amount of observation time in the studies varied from 12 to 60 min, which could have altered the amount of sexual behavior observed. However, the most frequent occurrence of sexual behavior throughout the female's cycle was seen in the 12-min pair tests, where some sexual behavior occurred at every day of the female's cycle. In contrast, even though subjects were observed for 30–60 min in studies with multiple fe-

males, sexual behavior occurred in much more discrete periods and ceased completely during the luteal phase. Thus, the opportunity to engage in sex had little apparent impact on how the female's cycle affected the occurrence of sexual behavior. While other contextual factors, such as the amount of available space (Wallen, 1982), affect rhesus monkey sexual behavior, social composition, particularly multiple females in the group, strongly influences the extent to which copulation is tightly coupled to the female's hormonal condition.

WHY DOES SOCIAL CONTEXT AFFECT THE RELATIONSHIP BETWEEN FEMALE HORMONES AND BEHAVIOR?

Since rhesus monkeys are capable of engaging in sex at any time, variations in the occurrence of sex must result from factors other than the capacity to engage in sex. One important influence on sexual behavior is the potential social consequence of engaging in sex. This influence is readily apparent in humans, who rarely engage in sexual behavior in public because of legal and social sanctions. Similar principles operate in rhesus monkeys due to the potentially socially destabilizing effects of sex on social relations in groups (Wallen and Tannenbaum, 1997). Thus sexual behavior should occur more frequently when the social consequences of mating are nonexistent or benign. For example, the relatively impoverished environment of the laboratory testing cage offers a male-female pair few behavioral alternatives to mating. In addition, the pair test eliminates mate selection and sexual competition, and it has no external social structure to destabilize. Together these conditions result in mating that has minimal social consequences and other factors, such as the compatibility of the investigator-selected pair (Goy, 1979), become more important than female hormonal state. In contrast, a group with a single male and multiple females exaggerates sexual competition and females, particularly low-ranking females, that consort with the male receive mild to intense aggression from other group females (Wallen and Tannenbaum, 1997). Thus because sex in a group setting involves significant social risk (Wallen, 1999), females only interact with males when they are highly sexually motivated. Further support for the notion that sexual competition and social risk influence sexual behavior is seen in the strikingly greater period of follicular mating in groups with multiple

males (Wilson *et al.*, 1982) than single males, which likely reflects decreased competition between females for sexual access to a male. In this view, hormonally modulated sexual motivation is a mechanism that increases the likelihood that sexual behavior will occur, even in the face of unpleasant social consequences. Because the same hormones that produce increased sexual desire also make the female fertile, this mechanism couples mating behavior with fertility in a complex social environment. When the pressure of social consequences are not present, the relationship between hormonal changes and mating behavior becomes much less pronounced and may not exist at all.

The longer follicular period of mating seen in the multiple male and female group most likely reflects the presence of multiple males, but it might also result from the number of adolescent females present among the 15 females studied by Wilson and colleagues. Adolescent females, unlike sexually experienced females, show prolonged follicular phases that are also accompanied by a longer period of follicular mating (Wilson and Gordon, 1980). Thus to understand how hormones affect sexual behavior it is important to understand the effect of the social context, but also the developmental stage of primates as well. We have recently investigated this issue in regard to male sexual maturation.

DEVELOPMENT OF SEXUAL BEHAVIOR: PUBERTAL CHANGES IN MALE PREFERENCE FOR MOUNTING PARTNERS

Beginning as early as the first 3 months of life, juvenile male rhesus monkeys engage in mounting behavior that is similar in form to that of adult male monkeys (Wallen, 1996). This juvenile mounting is displayed almost equally to male and female partners and continues throughout their prepubertal development, a period of gonadal inactivity (Wallen, 2000). However, as adults, males rarely mount other males and mount females almost exclusively. We were interested in what accounts for this transition in the sex of male mounting partners and what relationship, if any, it bears to hormonal changes that occur at puberty. We investigated the relationship between the sex of partner that males mount as juveniles to the sex of partner they mount at puberty by observing the mounting behavior of a group of 19 males at 1.5 years, when the

males were juveniles, and again at 3.5 years, when the males were just beginning to go through puberty.

Methods

Subjects were embedded in two 75- to 100-member age-graded social groups containing multiple males and females with their relatives and young. Subjects were born in these social groups and remained in their natal group throughout the course of the study. These males were part of a study of the effects of neonatal testosterone suppression on behavioral and neuroendocrine development. The neonatal treatments have been previously described (Mann *et al.*, 1994, 1998) and have not been found to affect prepubertal mounting behavior (Wallen, Maestriperi, and Mann, 1995; Nevison, Brown and Dixson, 1997). Subsequent analysis of the pubertal maturation of these subjects found that social rank, but not neonatal androgen manipulations, affected the timing of puberty (Mann *et al.*, 1998). Thus, for this behavioral analysis all subjects were combined regardless of neonatal treatment. Data collection methods for juvenile mounting are described elsewhere (Wallen *et al.*, 1995), but consisted of 100 h of focal observations on all males where any mounts displayed by that male were recorded. When the males were an average of 3.5 years old, we observed the sexual behavior of the males during an average of 55 h of scans distributed over the breeding season months from September through January. The methodology for sexual behavior observations is described elsewhere (Zehr *et al.*, 2000). For the purposes of this paper, ejaculation refers to the display of the ejaculatory reflex which is characterized by a sudden pause in intromitted thrusting followed by a spasmodic tensing of the pelvis and legs. This reflex may not have been accompanied by the expulsion of semen because some males had not gone through endocrine puberty. Behavioral data from both juvenile and peripubertal samples were converted to hourly rates to correct for differences in individual observation time. Changes in mounting were analyzed using *T* tests or ANOVA procedures (SPSS version 10 for Windows) with developmental stage, sex of partner, ejaculatory status, and hormonal status as factors in different analyses. When more than two means were compared, a *posthoc* least significant differences (LSD) procedure was used (SPSS version 10 for Windows). Weekly blood samples were collected on the males and assayed for testosterone to determine gonadal activation. The testosterone assay and the hormonal changes in these animals have been previously de-

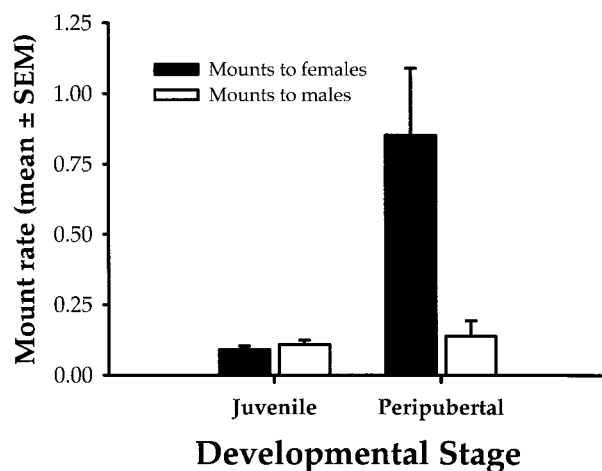


FIG. 2. Mount rate to male and female partners by juvenile and peripubertal group-living rhesus monkeys. The peripubertal increase in mounting occurred almost exclusively to female partners.

scribed (Mann *et al.*, 1998). All protocols were approved by an institutional animal use and care committee and were in accord with the *National Institutes of Health Guide for the Care and Use of Laboratory Animals*.

Results and Discussion

Fifteen of the 19 males showed pubertal increases in testosterone (T) during the observation period, as indicated by serum T levels above 1 ng/ml. Peripubertal males also showed a significantly higher rate of mounting than when they were juveniles (juvenile rate, 0.20 ± 0.02 mounts/h; peripubertal rate, 0.99 ± 0.23 mounts/h; $t = 3.30$, $df = 36$, $P = 0.002$). This fivefold increase in mounting rate was almost completely accounted for by the much higher mounting rate to female than to male partners, as shown in Fig. 2. Overall, there was no significant peripubertal change in mount rates to males, whereas mount rates to females increased dramatically. The change was even more marked for the subset of 11 of 19 males who ejaculated at some point during the period of observations at 3.5 years of age. These males almost exclusively mounted females, directing more than 95% of their mounts, on average, toward females. Figure 3 illustrates the significant difference in mounts to females between those males who were seen to ejaculate and those who were never observed ejaculating ($F_{1,17} = 6.5$, $P = 0.021$). The rate at which ejaculating males mounted females was significantly different from all other mount rates (P 's = 0.003 to

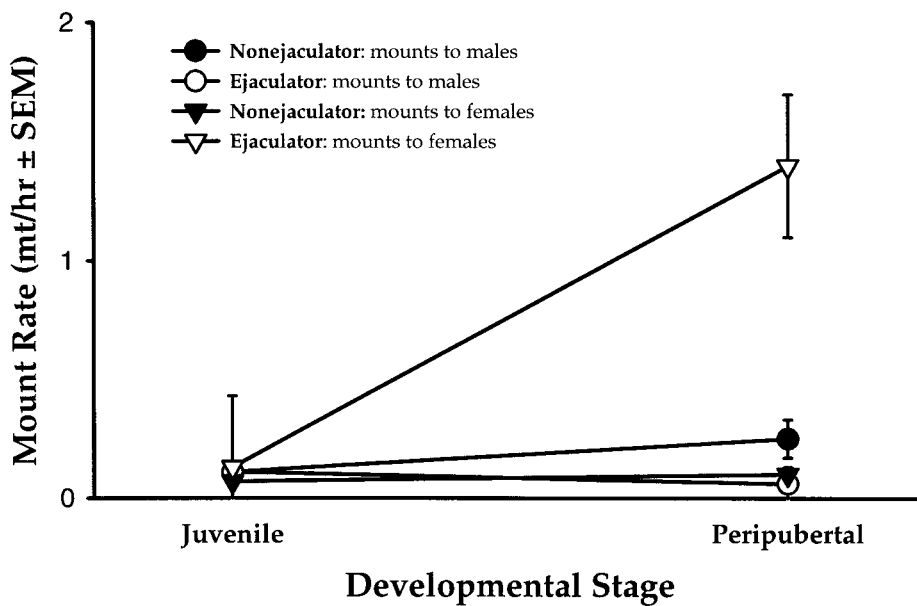


FIG. 3. Comparison of mount rates to male and female partners by group-living peripubertal male rhesus monkeys who were seen to ejaculate with a female (“ejaculators”) or not (“nonejaculators”) during the period of behavioral observations. Only ejaculators increased their mounting of female partners. The mounting of peripubertal nonejaculators was indistinguishable from their pattern of juvenile mounting.

0.005, LSD). The nonejaculating males had a higher peripubertal rate of mounting males than they did as juveniles; however, peripubertal ejaculatory status did not significantly affect male mount rate ($F_{1,17} = 3.2$, $P = 0.092$).

Since all subjects lived continuously in their social group we cannot rule out that “nonejaculator” males may have ejaculated when we were not observing them. However, it is likely that males we observed to ejaculate were the ones that did so routinely, allowing us to capture this behavior during our limited observations. Thus the marked difference in behavior seen between these two subsets of males may reflect high and low sexual activity or may actually reflect a difference between males that have experienced ejaculation and those who have not. In either case, it is clear that the sex of the partner changes markedly at puberty and appears to be related to the sexual experience of the males.

The gonadal function of the males contributed to, but did not fully account for, the change in mounting behavior at 3.5 years of age. While 10 of 15 males with elevated T displayed increased mounting of females and were observed to ejaculate, 5 males with elevated T were never seen to ejaculate and did not show a peripubertal increase in mounting of females. Furthermore, 1 of the 4 males that showed no increase in T during the study period displayed the ejaculatory re-

flex and also showed increased mounting of female partners. Figure 4 illustrates that the proportion of mounts displayed to male partners was best accounted for by whether a male was observed to ejaculate ($F_{1,15} = 17.6$, $P = 0.001$), with ejaculators displaying a much lower percentage of mounts to males. Whether a male showed increased T did not significantly predict the proportion of mounts displayed to males ($F_{1,15} = 0.004$, $P = 0.948$).

Whether this finding primarily reflects an effect of social experience on sexual behavior or is evidence that very low levels of T are sufficient to initiate adult copulation remains to be resolved. Data from adult male rhesus monkeys, however, demonstrate that the occurrence of male mounting behavior does not require androgens, but that androgens regulate the frequency of mounting and what contexts it is displayed in. The propensity to mount is organized by prenatal androgens (Wallen, 1996), but mounting itself is not under activational hormonal control (Goy and McEwen, 1980; Wallen, 1996). However, suppressing adult testicular function for a week significantly reduced the rate of male mounting (Wallen *et al.*, 1991), but did not completely eliminate mounting. This finding is consistent with the fact that juvenile males routinely mount while they are producing little or no T, but increase their mounting significantly peripubertally. Androgens do not produce mounting behavior;

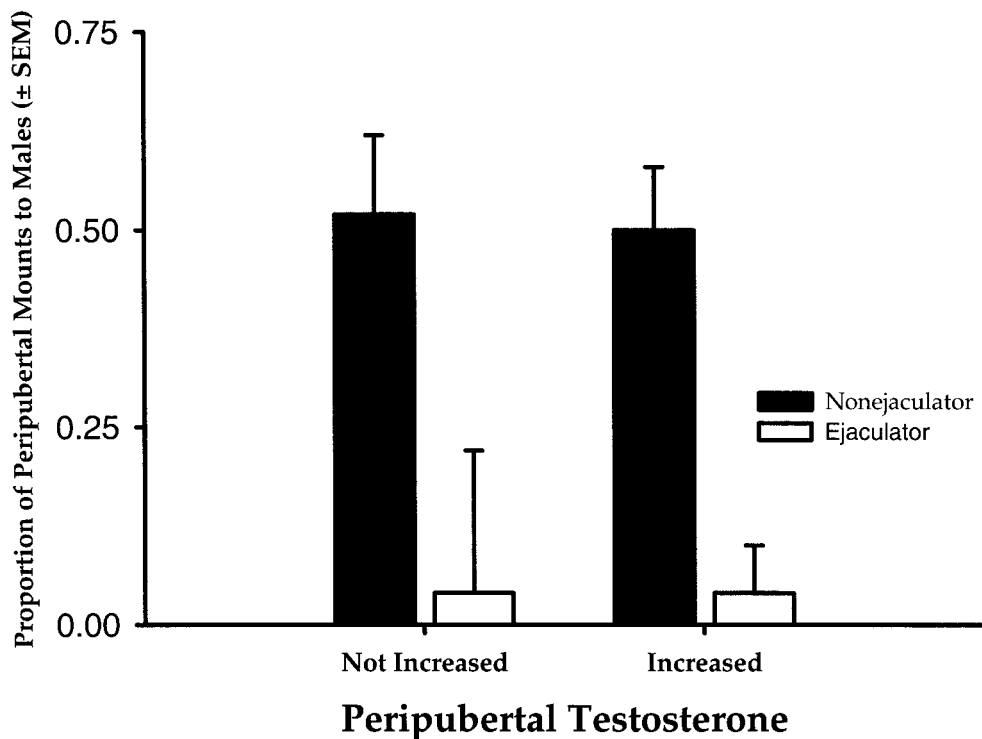


FIG. 4. Comparison of the proportion of peripubertal mounts to male partners for male ejaculators and nonejaculators in relation to whether the male had increased testosterone. Ejaculators displayed a significantly lower proportion of mounting to males than females, whether or not their testosterone was increased.

instead, they increase the probability that mounting will be elicited in specific contexts and to specific stimuli.

In this regard, the transition from equal mounting of male and female partners to almost exclusively mounting females seems likely to result from an interaction between the male's hormonal state and his social experience. Our results show that this transition can occur without a substantial rise in T if the male has sexual experience with a female. For most males the pubertal testosterone increase is the most likely impetus for this social transition. Testosterone increases penis size, and presumably tactile sensitivity, and it also increases sexual motivation. These hormonally induced changes produce increased male mounting and with it the likelihood that the male will achieve intromission. Intromission in turn increases the likelihood that a male will ejaculate. The rewarding properties of these sexual milestones increase male attraction and interest in female, rather than male, sexual partners. While testosterone stimulates these changes in the male, they also make the male more attractive to females, resulting in his becoming the target of female sexual solicitations. This active pursuit of the male by

sexually active females also increases the likelihood that males will mount females and not males. Thus the combination of a propensity for females to find sexually mature males attractive and to sexually solicit them, even if they are not particularly responsive (Zehr, Maestripieri, and Wallen, 1998), and the pubertal changes induced in the male by pubertal testosterone channel the male's behavior toward adult heterosexuality. It is clear, however, that testosterone alone is not sufficient, nor is it necessarily a prerequisite for this transition.

The best predictor of when a male will express a partner preference for females is whether the male has ejaculated with a female, independently of whether he has elevated testosterone. Males who have undergone endocrine puberty, but were never observed to ejaculate with a female, mounted males and females at comparable frequencies, whereas males who had not experienced increased pubertal T, but had ejaculated with females, showed a marked preference for female partners. To be sure, most males experienced both increased T and ejaculation with females together, but it is apparent that it is possible, though less common, to have one change without the other. Thus the tran-

sition to adult male heterosexual mating appears to require specific sexual experience that is typically triggered by the increased sexual motivation produced by pubertal androgens. This interaction between hormonally modulated sexual motivation increasing the likelihood of specific social experience and sexual reward from females results in a preference for females as mounting partners. It is not clear just how labile this preference is. The fact that postpubertal rhesus monkey males never have been reported to preferentially mount males when they have access to sexually active females (Wallen and Parsons, 1997) suggests that pubertal testosterone may also activate an attraction to females as sexual partners that increases the likelihood of a heterosexual transition. Exactly when and how pubertal testosterone affects these psychological end points remains to be elucidated.

SOCIAL CONTEXT AND HORMONAL INFLUENCES ON ADULT MALE SEXUAL BEHAVIOR

Other evidence of the interaction between social context and sexual motivation comes from studies of adult male rhesus monkeys, which are seasonal breeders and undergo an annual cycle of testicular regression and recrudescence (Gordon, Rose, and Bernstein, 1976). Sexual activity within rhesus groups covaries with changes in male testicular function, ceasing when testicular activity ends during the nonbreeding season and increasing with the onset of testicular function with the breeding season (Gordon *et al.*, 1976). Females also undergo a similar seasonal cycle of gonadal function (Gordon, 1981; Wilson *et al.*, 1982); thus the lack of sexual activity during the nonbreeding season may reflect the effect of gonadal inactivity on males, females, or both. Studies of heterosexual pairs of rhesus monkeys have demonstrated that castration gradually reduces, but does not eliminate, male sexual activity (Michael and Wilson, 1974; Phoenix, Slob, and Goy, 1973; Phoenix, 1978). Evidence that testicular androgen activates male sexual behavior, independently of female sexual activity, and that this activation is affected by social context comes from studies that suppressed male testicular function in social groups of rhesus monkeys during the breeding season, when group females are sexually active.

A single 15 mg/kg dose of a gonadotropin releasing hormone antagonist, antide, given to seven adult males embedded in a large heterosexual rhesus mon-

key group, produced castrate levels of androgen within 24 h after treatment. Male sexual behavior declined significantly after 1 week of suppressed testicular androgen, much more rapidly than the decline reported in castrated males during pair tests (Wallen *et al.*, 1991; Wallen, 1999). In a separate study of multiple female groups, using single male, instead of multiple male, groups found that testicular suppression took significantly longer to decrease male sexual behavior than it did in the multiple male group (Davis-DaSilva and Wallen, 1989). This effect of social context is seen most clearly for two males who were used in both of these testicular suppression studies (Davis-DaSilva and Wallen, 1989; Wallen *et al.*, 1991). In the single male social group these males continued to mate after 4 weeks of testicular suppression, but stopped mating after 1 week of testicular suppression when tested in the multimale group (Wallen *et al.*, 1991). This more rapid decline in sexual behavior when multiple males are present suggests that testicular hormones may be important for successful sexual competition between males. Thus, as with female rhesus, the opportunity for intrasexual competition influences how tightly coupled sexual behavior is to gonadal function.

However, even in a social group setting with multiple males, the extent of decline in sexual behavior following testicular suppression was influenced by social context, in this case male social rank and sexual experience. Low-ranking, or sexually inexperienced, males completely stopped mating following 1 week of testicular suppression, but the sexual behavior of high-ranking, sexually experienced males was not measurably affected by their lowered testosterone (Wallen, 1999). Figure 5 illustrates the magnitude of the correlation between male testosterone level before, during, and after GnRH antagonist testicular suppression and weekly ejaculation frequency in relation to male social rank. The two highest ranking subjects were natal males going through their first postpubertal breeding season. The other five males were older and more sexually experienced, averaging eight breeding seasons of mating.

Two important points are evident from this figure. First, testosterone and ejaculation frequency were significantly correlated only for the four lowest ranking males. Second, among the sexually experienced males, the highest ranking male essentially had no correlation between T and ejaculations, whereas the lowest ranking male's ejaculatory behavior was almost perfectly predicted by his T level. Even within the sexually inexperienced natal males, the higher ranking of



FIG. 5. Magnitude of individual correlations between weekly ejaculatory frequency and testosterone level before, during, and after GnRH antagonist-induced testicular suppression in relation to sexual experience and male social rank. Within each subgroup lower ranking males had higher correlations between testosterone and behavior, suggesting a greater dependence upon hormonal stimulation for the occurrence of sexual activity. * $P < 0.10$; ** $P < 0.05$; *** $P < 0.001$.

the two had a lower correlation between T and sexual behavior.

These data support the notion that androgen-modulated sexual motivation enables lower ranking males to successfully compete with higher ranking males. They also indicate that the sexual behavior of lower ranking males is more dependent upon the presence of testosterone than is the behavior of high-ranking males. For example, the sexual behavior of the highest ranking sexually experienced male was not detectably affected by testicular suppression. Even though his T was suppressed for almost 8 weeks, this male continued to mount and ejaculate at frequencies not significantly different from his pretreatment levels. In contrast, the lowest ranking male stopped mounting and ejaculating during the first week of testicular suppression and did not mate again until his T levels returned to normal. These findings underline the point that androgens are not required for the capacity to mate or to show an ejaculatory reflex, but dramatically influence the motivation to mate and compete for access to sexual partners. This is most likely the reason that castrated male rhesus monkeys and humans continue to engage in sexual intercourse for years after castration (Phoenix, 1978; Heim, 1981). It seems likely that

such males, while they are physically able to continue to engage in sex, would both be little motivated to do so and not compete successfully with males having normal androgen levels. Motivational systems regulated by testicular hormones are likely an adaptation to group living that provide the necessary impetus for successful sexual competition.

Little is known about the role of sexual motivation in human sexual competition. It is clear, however, that testicular androgens powerfully affect human male sexual motivation. Two findings strongly support the idea that testicular hormones primarily influence male sexual interest and not the ability to perform sexually. The first comes from a study of castrated male sex offenders in Europe (Heim, 1981). These males retrospectively reported their level of sexual intercourse and masturbation before and after castration. While castration significantly reduced both types of sexual activity, the postcastration decline in masturbation was significantly greater than the decline in sexual intercourse. It seems plausible that the greater decline in masturbation reflects its complete dependence upon the male's sexual desire, while intercourse reflects both the male and his partner's degree of sexual interest. This finding suggests that castration primar-

ily affected sexual interest and not ability to engage in sex, a view that received support in studies of erectile response in hypogonadal males. Kwan *et al.* (1983) and Bancroft and Wu (1983) investigated, in separate studies, the erectile response of hypogonadal males, who produced castrate levels of T, to sexually explicit films. Surprisingly, these men developed erections as rapidly as did control males and maintained their erections longer than did control males. Thus the ability to get an erection to sexual stimuli is not under hormonal control in humans. While it may be related to exposure to androgens during sexual differentiation it does not require concurrent circulating levels of T for its occurrence. In contrast, sexual desire strongly depends upon the concurrent presence of testicular hormones.

Bagatelle, Heiman, Rivier, and Bremner (1994) suppressed testicular function with a GnRH antagonist in normal male volunteers and assessed its effect on male sexual desire and fantasy in comparison to testicular suppression with androgen replacement. Males reported a significant decrease in sexual desire and fantasy after 2 weeks of testicular suppression, which was also accompanied by a significant decrease in masturbation. No decline was found when males received concurrent androgen therapy with the GnRH antagonist. Interestingly, the addition of an aromatase inhibitor with the androgen therapy, which markedly suppressed the production of estradiol, had no effect on male sexual motivation, suggesting that estrogenic metabolites of T are probably not the active steroids affecting sexual motivation in men (Bagatelle *et al.*, 1994). The importance of sexual competition in human society is a matter of speculation based upon little solid evidence. It is clear, however, that sexual motivation in men, like that of our nonhuman primate cousins, depends upon testicular androgens and that sexual behavior is strongly influenced by social context.

SOCIAL CONTEXT AND HORMONAL INFLUENCES ON HUMAN SEXUAL BEHAVIOR

While rhesus monkey sexual behavior is strongly affected by the specific composition of their social groups, humans create cultural contexts that affect their sexual behavior in ways beyond that possible with simple aggregations of individuals. The most obvious of these is that most human cultures restrict

the ages, places, and times where sexual activity is socially appropriate. Animals may copulate cryptically to avoid predation, but only humans have conventions that restrict sexual activity to appropriate social contexts. Significantly less is known about human than animal sexual behavior simply because animals often do not hide their sexual activity from human observation. Even when humans report their sexual activity it is clear that cultural conventions profoundly affect its occurrence, as shown in Fig. 6. This illustrates that the occurrence of sexual intercourse in 1941 couples is not randomly distributed across the days of the week, but peaks on weekends, with the highest occurrence, surprisingly, on Sunday morning (Palmer, Udry, and Morris, 1982). It is not the case, however, that human sexual behavior is completely regulated by social context.

Hormonal influences on women's sexuality are difficult to demonstrate because of the cryptic nature of human sexual behavior and the unwillingness of subjects to be sampled, behaviorally and physiologically, as frequently as would be necessary to definitively demonstrate hormonal effects. However, several studies provide evidence that hormones affect female sexual interest. The most compelling data come from surgically menopausal women, who report a marked decline in sexual desire that cannot be accounted for by acute effects of the surgery and which is reversed by hormonal therapy (Dennerstein, Wood, and Burrows, 1977; Leiblum *et al.*, 1983; Sherwin, Gelfand, and Brender, 1985; Sherwin and Gelfand, 1987).

Correlating endogenous hormonal rhythms with sexual behavior has often produced contradictory findings. While much of the disagreement stems from methodological issues, such as sampling frequency, the fact remains that it will be hard to establish consistent relationships between hormonal changes and behavior because human sexual behavior does not require hormones for its occurrence. This trait means that they can engage in sexual activity without regard to hormonal state, allowing factors other than hormonal condition to determine the occurrence of sexual behavior. Even given this constraint there is still evidence that ovarian hormones affect sexual motivation in humans. Stanislaw and Rice (1988) provided one of the most striking demonstrations of cyclical variation in sexual desire. They used a relatively simple methodology to sample sexual desire in several thousand women who used basal body temperature to indicate presumed ovulation and reported the first day that they noticed increased sexual desire. When the thousands of responses were plotted against cycle day, a

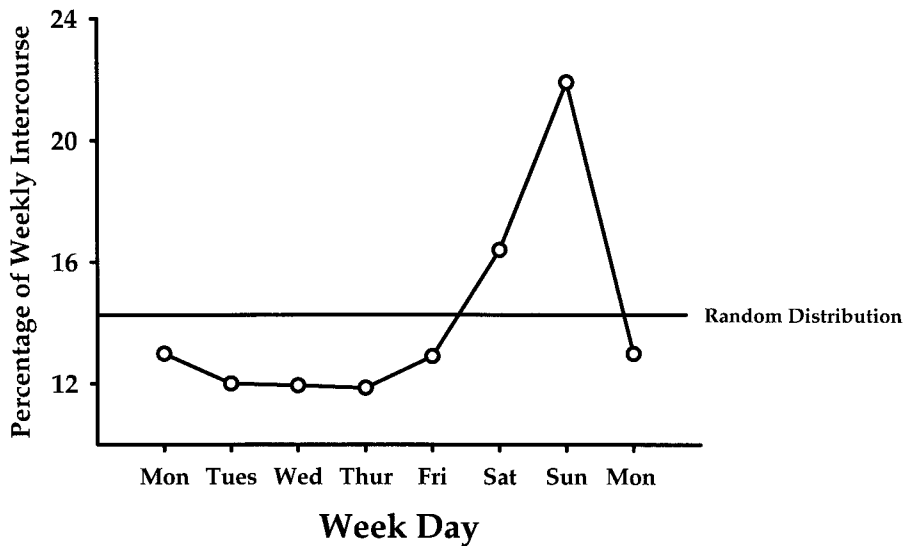


FIG. 6. The distribution of human coitus across days of the week. The horizontal line represents the daily proportion of sexual intercourse that would be expected if sexual intercourse was randomly distributed throughout the week (figure adapted using data contained in Palmer, Udry, and Morris, 1982).

significant and remarkable peak in increased sexual interest centered on the presumed day of ovulation. Similarly Bancroft, Sanders, Davidson, and Warner (1983) reported that female sexual desire markedly increased between the early follicular and midfollicular portions of the cycle, but that intercourse frequency did not show a similar cyclic variation. More recently Dennerstein *et al.* (1994) studied a sample of 168 women and found that female sexual desire increased during the follicular phase and declined markedly during the luteal phase in cycles aligned using daily urinary estrogens.

Studies of human sexual activity are complicated by the relatively constant male sexual interest that can mask the effects of cyclical changes in female sexual desire. One study that investigated both male and female sexual initiation in relation to the female's ovarian cycle illustrates the striking difference between how the female's ovarian cycle affects her and her male partner (Van Goozen *et al.*, 1998). As shown in Fig. 7, female sexual initiation was strongly coupled to her ovarian cycle phase, being highest preovulatory and lowest luteally, but the partner's sexual initiation did not vary across the female's cycle. One way around this problem is to study the sexual activity of women without the presence of men or to look at female sexual initiation instead of simply noting the occurrence of sexual intercourse.

Matteo and Rissman (1984) found that the sexual activity of lesbian women was highest when one part-

ner was at midcycle. Similarly, in heterosexual couples, Adams Gold, and Burt (1978) were the first to separate female sexual initiation from male sexual initiation and reported that female sexual initiation increased at midcycle in those women using reliable nonhormonal contraception (see Wallen, 1999, for a more complete discussion). They also presented evidence that women using less reliable contraceptives

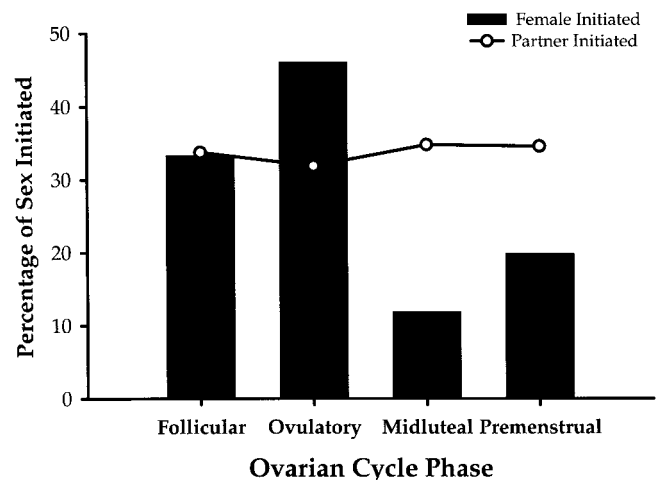


FIG. 7. The distribution of sexual activity initiated by women and their sexual partner in relation to the stage of the female's ovarian cycle. Female initiation, but not that of her partner, varied with ovarian cycle phase (figure adapted from data contained in Van Goozen *et al.*, 1998).

showed a less pronounced midcycle increase in heterosexual initiation and a much more pronounced midcycle increase in autosexual behavior. These data suggested that the women experienced cyclical sexual desire, but that the behavioral outlet for this sexual interest, heterosexual or autosexual activity, was affected by other factors, such as the perceived risk of pregnancy.

More recently, Harvey (1987) investigated the sexual behavior of women who tracked their cycle phases using basal body temperature and recorded the occurrence of sexual behavior that the women initiated or that was initiated by their partner and the women's autosexual activity. When all sources of sexual outlet were combined there was a distinct and significant periovulatory peak in sexual activity, as shown in Fig. 8A, suggesting a clear influence of the female's ovarian cycle on sexual activity. When male and female sexual initiation and female autosexual activity were considered separately a more complicated pattern emerged. Male sexual initiation was relatively constant across the partner's cycle, with a modest periovulatory increase. Throughout the cycle, female sexual initiation accounted for less than 40% of sexual initiation and unlike that of the male partner, actually decreased at midcycle. However, when female autosexual activity was considered as a percentage of total female sexual outlets, it increased markedly during the periovulatory phase. Figure 8B illustrates the variation in these two patterns of female sexual behavior, initiation and autosexual activity, in relation to ovarian cycle phase. While the periovulatory decline in female sexual initiation might be interpreted as evidence of a decrease in sexual interest at this time, the simultaneous increase in female autosexual activity argues against this. Instead, the data are more consistent with the idea that women vary their sexual practices according to their perceived risk of pregnancy (Tsui, de Silva, and Marinshaw, 1991).

Fear of pregnancy is commonly acknowledged to influence human sexual activity, but it is surprisingly little investigated and rarely considered in studies evaluating cyclical changes in human sexual behavior. Most studies typically assume that humans will engage in sexual behavior whenever they can, even though humans regulate their sexuality for any number of social and reproductive reasons. Unlike other animals, humans are, as far as we are aware, the only species that actively avoids pregnancy and recognizes pregnancy as a consequence of sexual activity. For example in a sample of 274 undergraduate women more than 96% tracked their ovarian cycles, 74% knew

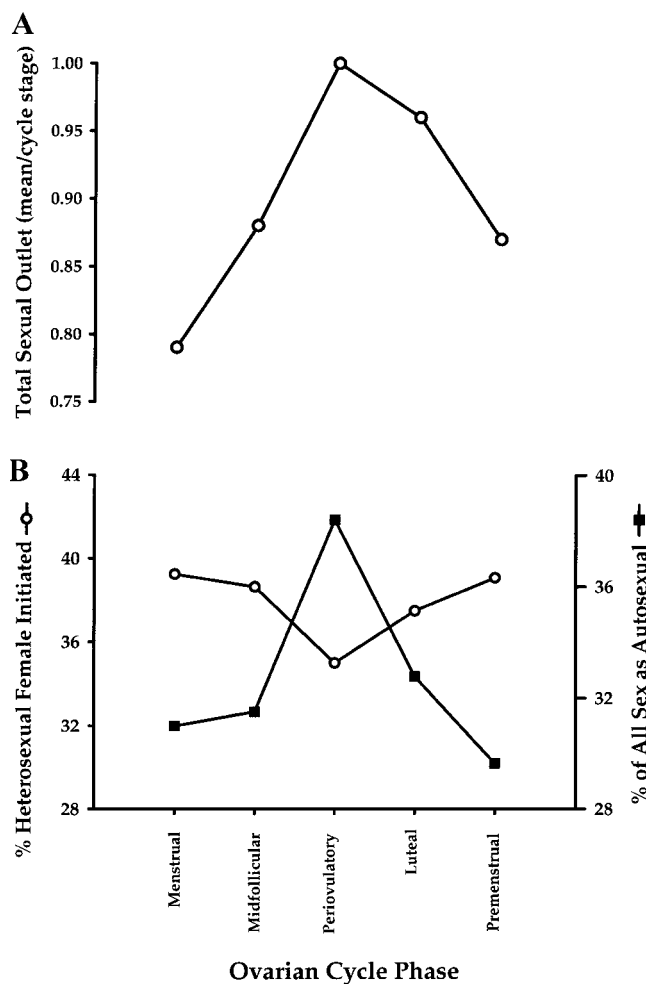


FIG. 8. (A) Total human female sexual outlet in relation to stage of the ovarian cycle. Female sexual activity was highest during the periovulatory portion of the ovarian cycle. (B) The relative distribution of human female-initiated sexual intercourse as a percentage of all heterosexual intercourse in comparison to female autosexual behavior as a percentage of all sexual activity in relation to ovarian cycle phase. Female sexual heterosexual initiation declined and autosexual activity increased during the periovulatory phase, when fertility would be highest, suggesting that the type of sexual activity engaged in is related to perceived pregnancy risk (figure adapted from data contained in Harvey, 1987).

that ovulation occurred at midcycle, and 42% believed they could detect when they ovulated (Small, 1996). Similarly Harvey (1987) reported that 87% of the 69 women in the study were aware that there is an increased probability of conception at midcycle. Thus, in humans, certainly in technological societies, the calculus of sexual behavior includes an awareness of changes in the female's cycle and consideration of the likelihood of pregnancy. This is a particularly profound, but little studied, influence on female sexuality,

since women or couples may modify their sexual activity during the time they think fertility is highest. Thus at exactly the point in the ovarian cycle where one might hope to detect increases in sexual activity related to fertility, some subjects may be actively inhibiting their sexual behavior to avoid pregnancy.

The role that hormones play in this complex interaction is probably limited to providing increased motivation coincident with fertility. This increased motivation may be insufficient to overcome other motivating factors, such as avoiding pregnancy. In this context, it is interesting that strong relationships have ever been reported between ovarian hormones and sexual behavior. Similarly, it is not surprising that the strongest relationships are in regard to sexual desire and not to sexual intercourse.

WHAT HORMONES INFLUENCE FEMALE PRIMATE SEXUAL DESIRE?

The work already summarized provides powerful evidence that ovarian hormones modulate female sexual desire and thus female sexual behavior. Which ovarian hormones are responsible for these effects? In mammalian females estradiol and progesterone typically regulate female sexual behavior (Young, 1961). Even in the female musk shrew, where androgens are the principle circulating steroid during the initiation of sexual receptivity, they are apparently aromatized to estrogens for their behavioral effects (Rissman, 1991; Veney and Rissman, 2000). In contrast among primates, human and nonhuman, there is research implicating androgens, particularly androgens of adrenal-cortical origin, in the modulation of female sexual behavior (Baum, Everitt, Herbert, and Keverne, 1977; Waxenberg, Drellich, and Sutherland, 1959; Sherwin and Gelfand, 1987). While adrenal androgens could not produce the cyclical fluctuations in female sexual initiation and desire typical of human and nonhuman primate females, the notion that ovarian androgens may be critical for female sexuality is widely held and, if true, would set primate females apart from other mammalian females. While a history of how this view developed is beyond this review, it is worthwhile to consider the evidence related to this issue to try to resolve whether a markedly different set of hormones modulate primate female sexual desire than the estrogens and progestins responsible in other mammalian females.

Adrenal Androgens and Female Sexuality

The idea that androgens affected female sex drive started with studies of synthetic androgens on female sexuality in the 1930s and 1940s. These studies suggested that large doses of testosterone (frequently inducing clitoral growth and deepening of the voice) increased libido in women (Loeser, 1940; Geist, 1941; Salmon and Geist, 1943; Foss, 1951). Such findings when combined with studies claiming that 88% of ovariectomized women had no diminution of libido (Filler and Drezner, 1944) led to the view that the ovary was unimportant to female sexuality, but that androgens were. The view that the adrenal cortex was the source of these androgens derives from a study which reported that adrenalectomy plus ovariectomy almost completely eliminated sexual activity in terminally ill breast cancer patients, whereas ovariectomy by itself had little effect on sexual intercourse (Waxenberg *et al.*, 1959). Although this study has had a marked impact on thinking about female sexuality in both humans and nonhuman primates, there are several reasons to be skeptical about its conclusions.

While the authors found little effect of ovariectomy on sexual intercourse, they reported a decrease in "sexual interest" following ovariectomy, which was not significantly different from the decrease they reported following ovariectomy and adrenalectomy (Waxenberg *et al.*, 1959). Thus, this study actually provided the first evidence that ovariectomy reduced female sexual motivation, but had minimal effects on the capacity to have sexual intercourse. Furthermore, although the authors concluded that the adrenal cortex is critical to female sexual function, their study lacked the critical experimental group of women who only received adrenalectomy that is necessary to demonstrate that the adrenal cortex is required for female sexuality when a functioning ovary is present. The design of this study could not have demonstrated a critical role for the adrenal in female sexuality. Surprisingly, since 1959, no one has attempted to demonstrate that the adrenal cortex is important to the sexuality of women, yet this notion remains commonly held in human sexuality.

Following this study in humans, the role of adrenal secretions in rhesus monkeys was investigated using pair tests. These studies suggested that adrenalectomy (or suppression of the adrenal gland with the synthetic glucocorticoid dexamethasone) diminished or abolished female sexual receptivity (Everitt and Herbert, 1969, 1971; Everitt, Herbert, and Hamer, 1972; Johnson and Phoenix, 1976). Testosterone and, in some cases,

androstenedione reinstated the behavior in adrenal suppressed animals. It is significant that an aromatizable androgen (e.g., testosterone) was required to reinstate the behavior, while the nonaromatizable androgen 5 α -dihydrotestosterone was ineffective (Wallen and Goy, 1977). Thus, it is not androgens in general which reinstate female sexual behavior, but large amounts of androgens that can be converted to estrogen (Johnson and Phoenix, 1976), a situation very comparable to that of the musk shrew, where androgens are important for female sexual receptivity, but only if they are aromatized (Rissman, 1991; Veney and Rissman, 2000). In contrast to these early studies in rhesus monkeys, adrenalectomy has no detectable effect on female sexual behavior in pairs of marmosets (Dixon, 1987) or stump-tail macaques (Baum, Slob, Jong, and Westbroek, 1978; Goldfoot, Wiegand, and Scheffler, 1978). In addition to species differences it also appears that the effects reported in rhesus monkeys reflect the use of pair tests and ovariectomized monkeys.

Adrenal suppression, via chronic dexamethasone treatment, in group-living rhesus monkey females with intact functioning ovaries had no detectable effect on female sexual behavior (Lovejoy and Wallen, 1990). This treatment reduced androgen levels by more than 75%, but, because ovarian function remained, had no effect on estradiol levels. In contrast, ovarian suppression using a GnRH agonist almost eliminated female sexual initiation and stopped sexual activity in group-living females (Wallen *et al.*, 1986). This GnRH-agonist treatment produced ovariectomized levels of estradiol, but reduced androgen by less than 50% (Wallen and Lovejoy, 1993). Thus ovarian suppression, but not adrenal suppression, reduces or eliminates female sexual activity in group-living rhesus monkeys and the effect is more related to estradiol than to androgen levels.

Correlative Studies of Androgens and Estrogens and Female Sexual Behavior

Further evidence that estrogens, not androgens, regulate female rhesus monkey sexual behavior comes from studies correlating daily changes in hormones with daily changes in behavior. In such studies, estrogens were significantly correlated with increases in female sexual initiation and progesterone with decreases in female sexual initiation, but testosterone was not significantly correlated with any pattern of female sexual behavior (Wallen *et al.*, 1984). In a different study, when female estradiol, progesterone, and

testosterone levels were used to predict when copulation would occur in female rhesus monkeys living in a social group, a model using estradiol and progesterone accounted for more variance ($R^2 = 0.49$) than estradiol, progesterone, or testosterone alone (Wilson, Gordon, and Collins, 1982). The addition of testosterone to the model did not significantly increase the variance accounted for ($R^2 = 0.52$; Wilson *et al.*, 1982). More recently, to address the capacity of estradiol to increase female sexual motivation, five ovariectomized females embedded in a large heterosexual group were studied during 6 weeks of estradiol or control treatment in a counterbalanced design (Zehr *et al.*, 1998). Observations took place during the non-breeding season, when males are sexually inactive, so that any changes in female behavior were unlikely to result from male sexual interest. Female initiation of contact with males and presentation to males were both highly correlated with estrogen level ($r_s = 0.88$ and 0.81 , respectively, $P < 0.005$; Zehr *et al.*, 1998), supporting the idea that estrogen increases female sexual interest, even when the males are not sexually active.

Together these findings support the idea that ovarian function regulates female sexual motivation and that estrogens not androgens are the critical gonadal steroids. They cannot rule out any effect of androgens, as in all studies some estrogens and androgens were present. However, they clearly demonstrate that androgens are not sufficient by themselves to increase female sexual desire and they clearly demonstrate that adrenal cortical function is unnecessary for complete female sexual activity. In addition these studies reinforce the point that the effects of hormonal manipulation on female sexual behavior in rhesus monkeys are sensitive to the social conditions under which the animals are studied.

One last point concerning possible adrenal influences on female sexuality is that cyclic fluctuations in sexual desire are incompatible with the tonic sex steroid secretion produced by the adrenal. As evidence accumulates of cyclic changes in sexual interest there is little reason to continue to hold the notion that adrenal function is critical to normal sexual functioning in women. Still, the question of whether ovarian androgens or estrogens are the primary modulators of female sexuality remains controversial because human studies have failed to find robust correlations between either group of steroids and female sexual behavior. This is partly because studies rarely measure both estrogens and androgens within the same population and partly because human studies do not

allow the frequent sampling possible in monkeys that would allow the detection of correlations with estradiol's rapidly changing levels.

One set of studies that measured both estrogens and androgens (Persky *et al.*, 1978a,b) reported no significant relationships between changes in either estrogen or testosterone and sexual behavior. However, when the average peak estradiol values for the women in each of the 11 couples in the study are related to the wife's average sexual initiation score there is a very strong correlation ($r = 0.68$, $n = 11$, $P = 0.02$). In contrast the women's testosterone levels were not significantly correlated with their sexual initiation scores ($r = 0.37$, $n = 11$, $P = 0.26$). This analysis suggests that estradiol may be having an effect on female sexual initiation that is masked in the daily variance of the cycle. In this regard it is interesting that in another study that used daily urine samples to measure estradiol levels in a sample of 16 women, intercourse frequency was significantly higher on the day preceding peak urinary estrogen levels, the presumed day of peak blood estrogen levels (Hedricks, Piccinino, Udry, and Chimbara, 1987; Hedricks, Schramm, and Udry, 1994). However, other investigators have not found a significant relationship between daily urinary estrogens and a measure of female sexual interest (Dennerstein *et al.*, 1994).

The most recent study investigating this issue (Van Goozen *et al.*, 1997) measured estrogens and androgens three times per week and reported that average androgen levels across the cycle correlated significantly with frequency of sexual intercourse, masturbation, and average sexual interest, but that ovulatory androgen levels were not significantly correlated with sexual interest even though female sexual initiation was highest at this time (Fig. 7). Furthermore, these significant correlations pertained only to a subgroup of approximately one half of the subjects who had premenstrual complaints, but not the women who experienced no premenstrual problems. Average estradiol levels were not significantly correlated with any measure of sexual behavior, but the use of average cyclic values prevented detecting periovulatory changes in estrogens and behavior. Surprisingly, even though this study had alternate-day estradiol measurements no attempt was made to correlate estradiol levels with behavioral measures, even though inspection of the behavioral and hormonal figures presented in the article suggests a tighter fit between female sexual initiation and estradiol than with testosterone (Van Goozen *et al.*, 1997). Thus, after more than 30 years of study, there is still no human study that has

correlated daily changes in ovarian hormones with daily ratings of sexual desire and looked at both estrogens and androgens. What evidence there is suggests weak, but sometime significant, correlations with testosterone, but often these reflect average levels across the cycle and not peak levels or cyclic variation in androgens. In addition there is also evidence that estrogens strongly influence sexual motivation in women, but unfortunately this possibility has been less investigated than the potential role of androgens on female sexual motivation. The same imbalance is evident in studies of hormonal replacement therapy (HRT).

Postmenopausal Hormonal Replacement Therapy in Women

Perhaps the largest body of research on the effects of androgens comes from studies of HRT for postmenopausal women experiencing decreased sexual desire. It is now well established that ovariectomy markedly reduces female sexual desire (Dennerstein *et al.*, 1977; Leiblum *et al.*, 1983; Sherwin, 1985); however, what hormones restore sexual desire is controversial. In one of the first studies of surgically menopausal women, Dennerstein *et al.* (1977) suggested that estrogen replacement therapy did not affect overall sexual behavior, although there was a specific decrease in pain from intercourse, which might reflect increased vaginal lubrication. In a later study this laboratory reported increased sexual desire and orgasm frequency in surgically menopausal women during estrogen therapy in comparison to either a placebo or a progestational control (Dennerstein, Burrows, Wood, and Hyman, 1980). More recently, Sherwin and colleagues (Sherwin, 1985, 1991; Sherwin *et al.*, 1985; Sherwin and Gelfand, 1987) have investigated the effects of estrogen therapy or combined estrogen-androgen therapy in surgically menopausal women. A double-blind, placebo-controlled study by this group reported that women receiving a combination of estrogen and androgen had higher levels of sexual desire, arousal, and fantasy than those receiving estrogen alone or placebo (Sherwin *et al.*, 1985). However the results are not completely consistent with androgen regulation of female sexual desire since the surgical control group, which had naturally low androgen levels, expressed levels of sexual desire comparable to those of the group receiving the estrogen and androgen HRT, which had substantially higher androgen levels, but comparable estrogen levels. Furthermore, during the placebo month, the control group showed higher lev-

els of desire and fantasy than any other group, despite having lower androgen levels than these other groups (Sherwin *et al.*, 1985). Because subjects had recently undergone oophorectomy, lingering effects of surgical trauma may have affected these results. A later study in long-term oophorectomized subjects controlled for this and appeared to show a marked enhancement of sexual desire in women receiving a combined estrogen-androgen therapy in comparison to either estrogen alone or no HRT (Sherwin and Gelfand, 1987). However, the results of this study are also not consistent with androgen regulation of female sexual desire.

All subjects had previously received either the estrogen-androgen HRT or estrogen HRT or no HRT treatment. Prior to the start of data collection all subjects stopped their specific hormonal therapy for 8 weeks. At baseline, after 8 weeks without HRT, all subjects reported very low levels of sexual desire and sexual fantasy (Sherwin and Gelfand, 1987), demonstrating that ovariectomy reduces female sexual motivation. HRT therapy was reinstated and sexual desire and fantasy increased markedly in the combined estrogen-androgen HRT group, but not in the estrogen HRT or no HRT groups. While this appeared to strongly support the importance of androgen in combination with estrogen for reinstating female sexual desire, this study also provided clear evidence that androgen by itself was not capable of increasing female sexual desire. At baseline, all three subject groups reported low levels of sexual desire and had uniformly low basal estradiol levels. However, the basal testosterone levels in the group that previously received the estrogen-androgen HRT were significantly higher than those of the other two groups of subjects and four to five times higher than peak mid-cycle levels in nonovariectomized women (Sherwin and Gelfand, 1987). Thus even though all three groups of women reported uniformly low levels of sexual desire, one group had circulating androgen levels well above peak physiological levels without any apparent effect on sexual desire. It was only when additional estrogen was given to these women that sexual desire increased, strongly suggesting that androgen alone does not increase female sexual desire. Further support for little or no effect of testosterone on female sexual desire comes from a recent study of 65 surgically menopausal women experiencing low sexual desire and sexual satisfaction (Shifren *et al.*, 2000). All subjects received conjugated estrogens and either a placebo or 150 or 300 μg of daily testosterone in a

counterbalanced double-blind design. None of the treatments increased an index of sexual thoughts or desire to the level reported for nonmenopausal women, but all treatments, including the nonhormonal placebo, increased sexual thoughts or desire above the baseline. There was some evidence that the 300- μg testosterone dose increased the composite score of sexual functioning above the placebo, but also produced serum levels of testosterone that were almost twice peak endogenous levels (Shifren *et al.*, 2000). Furthermore, for the 31 women in the study under 47 years old, there was a significant effect of the placebo on their sexual functioning score, but no additional effect of either testosterone treatment. Thus, although this study purports to show an effect of testosterone on female sexuality, it finds only a weak effect with nonphysiological doses and provided evidence of a larger effect of the placebo than either T treatment. Interestingly, subjects under all treatment conditions showed significantly elevated LH and FSH levels, suggesting that the chronic estrogen treatment all subjects received was not sufficient to produce negative feedback suppression of gonadotropin secretion. The possibility remains that this estrogen treatment was also insufficient to influence female sexual desire.

Recent data (Sherwin, 1991) from naturally, not surgically, menopausal women suggest that estrogen by itself increases sexual desire and arousal in postmenopausal women relative to a period of no hormonal administration. Progesterone added to estrogen under these conditions did not alter libido, although it increased scores of negative psychological symptoms. These data from naturally menopausal women are in contrast to those from studies suggesting that estrogen therapy alone (or in combination with progesterone) has little effect in improving sexual desire or behavior (Utian, 1972; Coope, 1976; Campbell and Whitehead, 1977; Furuholm, Karlgren, and Carstrom, 1984; Sherwin, 1985). However, several studies have found a positive effect of estrogen administration on female sexuality in menopausal women (Dennerstein *et al.*, 1980; Iatrakis *et al.*, 1986; Sherwin, 1991). In contrast, authors have reported that long-term androgen levels, but not estrogen levels, correlate with postmenopausal sexual interest (Bachman *et al.*, 1985; McCoy and Davidson, 1985; Bachmann and Leiblum, 1991). Dow, Hart, and Forrest (1983), however, could show no advantage of estrogen plus testosterone administration over estrogen alone, whereas Sarrel, Dobay, and Wiita (1998) found a significant increase in sexual

desire using a combined estrogen–androgen HRT in women dissatisfied with estrogen HRT. The reason for the discrepancies among these studies is unclear, but may be related to the role that sex hormone binding globulin (SHBG) plays in regulating free hormone levels. It has been suggested that SHBG serves as a biological servomechanism that regulates the relative bioavailability of estrogens and androgens through differential binding and the differential effects of these two classes of hormones on SHBG production (Burke and Anderson, 1972). Sarrel *et al.* (1998) reported that SHBG levels increased during estrogen HRT, but decreased during combined estrogen–androgen HRT. They also reported that free T increased during the combined HRT, but did not measure free estradiol, which also binds to SHBG and would be expected to increase with decreased SHBG levels. Recent work in our laboratory found that androgen administration to chronically estradiol-treated ovariectomized rhesus monkeys increased the unbound fraction of estradiol in the blood. This increase in unbound estradiol was related to increased female sexual initiation in female rhesus monkeys, who had been sexually inactive on estradiol treatment alone (Wallen and Parsons, 1998). This finding suggests that androgen may play a crucial role in regulating estrogen availability through dynamic interactions with serum binding proteins and that estradiol is likely to be the hormone most responsible for modulating female sexual desire.

The focus on androgens in human studies has meant that little attention has been paid to the dynamics of free estradiol and its relationship to the presence of androgens. In this regard it is interesting that in intact cycling females the strongest correlations between hormones and behavior are found with estradiol and in ovariectomized females the strongest effects on female sexual desire are obtained with combined estrogen and androgen treatments, not with either estrogen or androgen alone. Both estrogens and androgens vary in concert during the female ovarian cycle. It seems likely that their coordinated fluctuation is an adaptation that regulates the relative bioavailability of either or both to influence fertility and sexual behavior. The resolution of this issue requires investigations of the dynamics of both estrogens and androgens in relation to serum binding and changes in sexual desire. It does seem likely, however, that primate females are unlikely to be remarkably different from other mammalian females in relying solely on androgens for sexual motivation.

CONCLUDING REMARKS

Ovarian hormones have evolved to coordinate increased sexual desire with fertility to produce pregnancy. It seems likely that the specific patterns of sexual behavior engaged in by women reflect an interaction between their level of sexual desire, which is affected by their hormonal state, the level of their partner's sexual desire, and the women's or the couple's desire to avoid, or achieve, pregnancy. The interaction between these factors can result in no cyclical change in sexual activity, midcycle increases, midcycle decreases in sexual activity, or a shift from partnered to solitary sexual activity. Only when the full social and cultural context of sexual behavior is considered is it possible to disentangle social, cultural, and hormonal influences on sexual behavior.

Hormonally modulated systems of sexual motivation coordinate the occurrence of sexual activity with fertility, but primates have evolved the capacity to engage in sex at any time, whether or not one is experiencing increased sexual desire. This emancipation from hormonal control of the ability to engage in sex allows sexual behavior to be used in nonreproductive contexts. In addition, it allows social context, and cultural conventions in humans, to powerfully influence the occurrence of sexual behavior independently of current hormonal condition. While this makes elucidating the specific manner in which hormones affect sexual behavior difficult, it also results in a more complete integration of sexuality into primate life. While the selective pressures that have produced this flexibility remain to be discovered, the benefits to species living in long-term complex social organizations are readily apparent.

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