HORMONAL CORRELATES AND CAUSES OF SEXUAL DESIRE: A REVIEW

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ABSTRACT: This review defines sexual desire, distinguishes sexual desire from other sexual experiences (e.g. arousal, activity), discusses common operationalizations of sexual desire, and then examines empirical research on the relationship of androgens, estrogens, progesterone and prolactin to sexual desire in men and women. The findings suggest that minimum critical levels of androgens appear necessary (although not sufficient) for the experience of sexual desire.

Key words: Sexual desire Hormones Sex hormones

INTRODUCTION

Sexual desire is associated with several significant individual and interpersonal human life events. Feelings of desire or sexual attraction may prompt individuals to seek and engage in sexual activity; such feelings therefore have implications for reproduction and species survival (for additional discussion, see Buss & Schmitt, 1993; Regan & Berscheid, 1999). Sexual desire also appears intricately linked to relationship adjustment and quality. A growing body of empirical evidence suggests that relationships in which one or both partners experience low sexual desire are believed to be, and often are, characterized by dissatisfaction and poor overall adjustment (e.g., Kaplan, 1979; Leiblum & Rosen, 1988; Levine, 1987; Regan, 1998; Verhulst & Heiman, 1979; Zilbergeld & Ellison, 1980). Consequently, a number of theoretical and empirical attempts have been made to delineate and explore the correlates and presumed causal antecedents of sexual desire. Although some researchers have examined external causes located in the physical or social environment (e.g., physically attractive others, erotic or pornographic media; Kenrick, Gutierres, & Goldberg, 1989; Regan & Berscheid, 1995, 1997), the majority have focused on causes located within or under the control of the individual. A variety of intraindividual factors have been examined, including acute or chronic drug use, age, sex or gender, mood and negative emotional states, personality, and hormonal or biological processes (for reviews, see Regan, 1996; Regan & Berscheid, 1999). Of these presumed correlates of sexual desire, the latter have received the most empirical attention. While human sexual responses are less biologically determined and more volitional than any popular reference to "raging hormones" would have us believe, many men and women believe that biological and hormonal processes cause sexual desire (e.g., Regan & Berscheid, 1995). Indeed, research does suggest that both endogenous and exogenous hormones contribute at least partially to the timing and magnitude of this particular aspect of sexuality.

This paper reviews past and current literature on the experience, measurement and causes of sexual desire and on the role of endocrine factors, specifically androgens, estrogens, progesterone, and prolactin, as causes or correlates of desire.

Each of these hormones, produced variously by the adrenal glands, the pituitary gland, the ovaries in women, and the testes in men, has been targeted as a possible antecedent or necessary precursor to sexual desire. This paper considers the issues involved in defining sexual desire, distinguishes this experience from other aspects of human sexual response, discusses the ways in which sexual desire is commonly operationalized, and reviews empirical research on the relationship between the sex hormones and sexual desire as experienced by men

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and women. The discussion of each hormone includes an overview of the endocrine glands associated with its production and of the major naturally occurring forms of the hormone and the amounts typically present in the blood plasma of adult men and women. It also examines empirical research on the relationship between the hormone and sexual desire. Because androgens and prolactin appear to have a similar relationship to desire in both men and women, the sections on these hormones are organized according to effect upon sexual desire (i.e., increase, decrease, none) rather than by sex (i.e., effect upon men, effect upon women), to avoid unnecessary repetition. However, the discussions of sexual desire and estrogens and progesterone are organized according to sex, primarily because research about the impact of these sex hormones on male sexuality is relatively sparse.

Papers were included in this review if the author(s): (1) specifically mentioned *sexual desire* or *sexual interest* in the body of their paper and/or included these terms in the measures administered to their participants; (2) invoked such terms as *sexual motivation*, *sex or sexual drive*, *sexual appetite*, and *libido* but were clearly referring to sexual desire; or (3) indirectly assessed sexual desire by utilizing an operationalization that adequately reflected the construct of sexual desire (e.g., they asked about or measured such subjective, psychological sexual experiences as sexual thoughts, sexual wishes, sexual feelings, sexual cravings, sexual attraction). Given the variety of such operationalizations in the literature, the specific operationalizations of desire employed in each of the studies reviewed are provided.

Definition and Operationalization of Sexual Desire

Sexual desire is commonly defined as a subjective, psychological experience or state that can be understood broadly as an interest in sexual objects or activities, or as a wish, need, or drive to seek out sexual objects or to engage in sexual activities (e.g., Regan & Berscheid, 1995, 1996). This experience is presumed to be distinct from physiological/genital sexual arousal (i.e., a state of reflex activation that involves the sex organs and nervous system; e.g., Masters, Johnson, & Kolodny, 1982, 1994), subjective sexual arousal (i.e., the subjective awareness of physiological/genital arousal; e.g., Green & Mosher, 1985), and sexual activity (e.g., masturbation, intercourse). Sexual desire, arousal, and activity may co-occur (for discussion, see Regan & Berscheid, 1999); nonetheless, the latter do not themselves constitute adequate indicants of sexual desire. That is, the occurrence of sexual activity does not necessarily imply a desire for such activity, nor does the absence of sexual activity necessarily reflect a lack of desire.

A variety of operationalizations for sexual desire are utilized in the literature. The majority of researchers directly ask their participants about *sexual desire* or *sexual interest* (frequency, level, degree, or amount). Others employ motivationally oriented terms, including *sexual motivation*, *sex drive*, *sexual urge*, *sexual craving*, and *sexual appetite*. Still others refer to the Freudian motivational concept of *libido*, a potentially problematic practice if participants do not interpret this term to mean sexual desire.

Other researchers attempting to measure sexual desire have operationalized the concept in terms of cognitive events (e.g., *sexual wishes*, *sexual thoughts*, *sexual fantasies*, *sexual imagery*) that are not associated with any overt sexual activity. The assumption is that these phenomena represent motivational aspects of sexual experience and therefore may serve as indirect measures of sexual desire (e.g., Sherwin, 1985). The fact that both men and women who seek treatment for low sexual desire also fantasize less during sexual activity and general daydreaming than normal controls lends support to this assumption (e.g., Nutter & Condron, 1983, 1985).

A final operational category includes such psychological events as *sexual feelings* not associated with overt sexual activity and not meant to include genital sensations and *sexual attraction* or an attraction to another individual that is explicitly based on sexual feelings.

Androgens

Androgens, or masculinizing hormones, are primarily synthesized in the testes and the adrenal cortex (the

outer section of the adrenal glands) and to a lesser extent in the ovaries. The primary naturally occurring androgens are *testosterone*, *androstenedione*, and *dehydroepiandrosterone*, and, under normal circumstances, a man's bloodstream contains a much greater concentration of these hormones than does a woman's bloodstream. For example, average values for plasma testosterone concentrations in healthy adult men fall within the range of 300-1500 nanograms per 100 milliliters of plasma (ng/100 ml; Regan & Berscheid, 1999). These levels are significantly lower in premenopausal women, ranging from 14 to 176 ng/100 ml throughout the menstrual cycle (Regan, 1996). Mean plasma levels for androstenedione, however, are higher in women than in men, ranging between 112-200 ng/100 ml during the menstrual cycle compared to the lower 60 ng/100 ml typically present in male plasma (Regan, 1996; Regan & Berscheid, 1999).

Researchers interested in the relation between the androgens and sexual desire have concentrated primarily on testosterone, the most potent androgen. Testosterone is present in unbound ("free" or "bioavailable") and bound forms in both the blood (serum or plasma) and urine. The majority of this hormone (roughly 96 to 98 percent) is bound to protein, primarily to sex hormone-binding globulin (SHBG) and albumin, and therefore is not available to enter target cells and exert biological effects. The remaining proportion, called "free" or "unbound" or "bioavailable" testosterone, constitutes the bioactive moiety of testosterone; that is, the fraction available to target tissues (Itoh et al., 1991; Khan-Dawood, Choe, & Dawood, 1984; Rosenfield, 1990). Free testosterone, then, is the hormonal component most useful for assessing androgenic influences on or correlates of sexual response. However, because the portion of testosterone bound to albumin can dissociate to free, active testosterone (Dunn, Nisula, & Rodbard, 1981; Umstot, Baxter, & Andersen, 1985), it is often difficult for researchers to obtain reliable measurements of free testosterone. Consequently, results may be somewhat method-dependent (for additional discussion, see Petak, Baskin, Bergman, Dickey, & Nankin, 1998; Winters, Kelley, & Goodpaster, 1998).

Measurement issues aside, a growing body of evidence indicates that sexual desire is to some extent androgen-dependent in both men and women (for additional discussion, see Davis, 1998; Sherwin, 1994). For example, Persky et al. (1982) demonstrated that both testosterone and androstenedione were significantly and negatively correlated with their female participants' "sexual avoidance" scores, defined by the researchers as the null of sexual desire. Udry, Billy, Morris, Groff, & Raj (1985) determined that levels of free testosterone predicted the frequency of sexual thoughts experienced by adolescent boys better than six other hormones, pubertal development, and age. Halpern, Udry, Campbell, Suchindran, & Mason (1994) found a similar positive correlation between free testosterone levels and frequency of sexual thoughts in adolescent boys, and Alexander and Sherwin (1993) demonstrated that plasma levels of free testosterone were positively associated with their female participants' self-reported sexual desire and sexual thoughts.

The proposed relationship between endogenous androgens and sexual functioning has also been examined by studying women who have undergone surgical procedures such as oophorectomy (removal of the ovaries) and adrenalectomy (removal of the adrenal glands) and have thus undergone a sudden decrease in circulating levels of plasma testosterone and estrogen. In one of the first attempts to examine this relationship, Waxenberg, Drellich, and Sutherland (1959) interviewed 29 women diagnosed with metastatic breast cancer who had undergone total bilateral oophorectomy and adrenalectomy. Of the 17 who reported some consciousness of sexual desire prior to adrenalectomy, 10 (58.8%) reported a total loss of desire and 4 (23.5%) reported a noticeable decrease in desire after the procedure. In addition, interviews with the seven women who had undergone oophorectomy 16 to 60 months prior to adrenalectomy allowed the researchers to compare the effects of the loss of a major source of estrogens with the effects of the loss of a major source of androgens. Results indicated that removal of the ovaries in and of themselves decreased but did not eliminate the participants' experience of sexual desire; sexual desire only underwent a significant reduction subsequent to removal of the adrenal glands. Although this pioneering study can be

criticized for its lack of an experimental control group, its extremely limited sample size, and its use of a physically traumatized participant population, the results strongly suggested that adrenal androgens do influence what Waxenberg et al. (1959) term the "erotic component" of sexuality.

Treatment with synthetic steroids that suppress the synthesis of testosterone and interfere with the activity of adrenal androgens has been reliably associated with diminished sexual interest and desire in at least 3 groups of individuals: Sex offenders, prostate cancer patients, and women suffering from various androgen-dependent hair and skin conditions. Antiandrogenic substances such as cyproterone acetate (CPA) and medroxy-progesterone acetate (MPA) were originally developed for use as oral contraceptives and to treat various gynaecologic disorders (Briggs & Briggs, 1972; Cooper, 1986), but they have been most widely used and recognized as a treatment for sex offenders and other individuals unable to control socially or personally unacceptable sexual urges and dysfunctional sexual desires (e.g., Berlin & Meinecke, 1981; Briggs & Briggs, 1971; Cooper, 1995; Ferracuti & Bartilotti, 1975; Kiersch, 1990; Laschet & Laschet, 1969; Maletzky, 1991; Money, 1970; Myers, 1991; Neumann & Kalmus, 1991; Young, 1987). Men treated with CPA, MPA, or other antiandrogens (e.g., flutamide) have reported a reduction in their frequency of sexual thoughts, fantasies, and "urges" (e.g., Bancroft, Tennent, Loucas, & Cass, 1974; Berlin & Meinecke, 1981; Cooper, Ismail, Phanjoo, & Love, 1972; Cooper, Sandhu, Losztyn, & Cernovsky, 1992; Hucker, Langevin, & Bain, 1988; Kravitz et al., 1995; Rousseau, Couture, Dupont, Labrie, & Couture, 1990; Young, 1987). Marked decreases in sexual desire also have been noted in prostate cancer patients who received antiandrogenic treatment in combination with medical or surgical castration as a means of depriving cancerous cells of the stimulation provided by the adrenal sex steroids (Labrie et al., 1986; Rousseau, Dupont, Labrie, & Couture, 1988).

While androgen antagonists are generally viewed as a viable and reasonably successful form of treatment for such androgen-dependent hair and skin problems as acne, alopecia, hirsutism, and seborrhea, some researchers have noted a loss of "libido" as one of the various sexual side effects (e.g., Appelt & Strauss, 1986; Cittadini & Barreca, 1977; Cremoncini, Vignati, & Libroia, 1977; Hammerstein, Meckies, Leo-Rosberg, Moltz, & Zielske, 1975). Since the majority of these studies did not provide a more specific definition of "libido," and it is not clear whether the results point to an effect of decreased androgen production on subjective sexual experiences (e.g., sexual desire, feelings, fantasies, and thoughts), or whether the antiandrogens affected more physiologically-based responses (e.g., genital arousal, orgasm). Given the dampening effect of antiandrogens on sexual interest and desire in prostate cancer patients and sex offenders, it seems reasonable to assume that they had a similar effect in populations treated for skin conditions.

Not only does a decrease in the level of circulating androgens, brought about by surgical procedures or by antiandrogenic steroids, reliably result in a diminution of sexual desire in both men and women, an *increase* in androgen level can yield an associated increase in sexual interest or desire in some cases. The administration of exogenous androgens (usually testosterone) to men and women complaining of low sexual interest has been found to result in an increase in the self-reported strength of sexual desire (Rabkin, Rabkin, & Wagner, 1995) and in the frequency of sexual thoughts (O'Carroll & Bancroft, 1984) and desire for intercourse (Greenblatt, Mortara, & Torpin, 1942; Kennedy, 1973; Salmon & Geist, 1943). Androgen replacement therapy also has been used successfully to increase the self-reported frequency of sexual thoughts and feelings (Gooren, 1987; Kwan, Greenleaf, Mann, Crapo, & Davidson, 1983; O'Carroll, Shapiro, & Bancroft, 1985; Skakkebaek, Bancroft, Davidson, & Warner, 1981) and sexual desire (Arver et al., 1996; Kennedy, 1973; Kwan et al., 1983; Salmimies, Kockott, Pirke, Vogt, & Schill, 1982) of men with hypogonadism or eugonadism (conditions caused by various disorders of the endocrine system that result in abnormally low levels of testosterone. If given before puberty, androgen treatment can reverse the failure to develop normal adult secondary sex characteristics (Wade & Cirese, 1991).

Results similar to those described for men have also been obtained in women. Kaplan and Owett (1993)

described the spontaneous return of "libido" and "sexual feelings" following testosterone replacement therapy in a sample of 7 women with androgen deficiency syndrome (i.e., a demonstrable androgen deficiency caused by chemotherapy with cytotoxic agents and/or bilateral salpingo-oophorectomies). Combined subcutaneous implants of testosterone and the estrogenic hormone estradiol also have been associated with significant increases in women's sexual interest, sexual desire, and "libido", as well as in the number of sexual thoughts and fantasies (e.g., Brincat et al., 1984; Burger et al., 1984; Cardozo et al., 1984; Sherwin, 1985; but see Dow & Gallagher, 1989; Slob et al., 1993). Although the design of the latter studies does not permit discrimination between the effects of testosterone and estradiol, it is interesting to note that the "libido" of Burger et al.'s (1984) participants was neither noticeably affected nor improved by an earlier treatment with oral estrogens. In addition, an appropriately controlled investigation conducted by Sherwin, Gelfand, and Brender (1985) clearly suggests that it is the exogenous testosterone rather than the estradiol that contributes to enhanced sexual desire. These investigators gave injections of an androgenic, estrogenic, androgenic-estrogenic combined, or placebo preparation to premenopausal women who required hysterectomy and oophorectomy for reasons other than malignant disease. The study began with an initial, three-month treatment phase in which each woman was randomly assigned to receive one of the four preparations, continued through a one-month placebo phase, and concluded with a second treatment phase in which each woman received one of the treatments to which she had not previously been exposed. Hormonal assay and analysis of daily ratings made by the participants indicated that women who received either the androgenic or androgenic-estrogenic combined preparation not only developed significantly higher levels of plasma testosterone but experienced higher levels of sexual desire and reported a greater number of sexual fantasies during both of the treatment phases than did those women who received the estrogenic drug or the placebo. In addition, withdrawal of treatment during the intervening placebo month was associated with simultaneous decreases in plasma testosterone levels, sexual desire scores, and frequency of sexual fantasies in both androgen-treated groups. This well-controlled study, along with the other results reviewed above, provides support for the hypothesis that sexual desire in women is to some extent androgen-dependent.

While testosterone and other androgens appear to influence desire, how necessary are they for desire to occur and how much is needed? Researchers have explored the possibility that only some baseline amount of androgenic substance need be present in the hormonal environment for desire to occur. For example, Sherwin (1988) (see also Bancroft, 1988; Campbell & Udry, 1994) has proposed that sexual desire will be noticeably affected only when the level of hormone has dropped below some unspecified critical threshold; at or above this threshold, increasing levels of hormone will have no further influence on desire. This notion would help explain why the serum testosterone levels of the physically healthy men in a study conducted by Brown, Monti, and Corriveau (1978) failed to significantly correlate with the daily frequency of sexual thoughts and sexual interest—the majority of healthy men have more testosterone than is required for the experience of sexual desire. The same may be true for women. Bancroft, Sanders, Davidson, and Warner (1983) discovered that the testosterone levels of the women in their study were highly correlated with such behavioural sexual events as masturbation, but were negatively or insignificantly correlated with such subjective measures as sexual feelings and pleasant sexual thoughts. Similarly, the mean adrenal levels of the older, postmenopausal women in the Persky et al. (1982) study were significantly lower than those of the premenopausal women, although both groups of women reported the same levels of sexual desire.

These above results suggest that the presence of some specified level of androgens may be necessary for the experience of sexual desire. However, at least two studies of physically healthy women diagnosed with hypoactive sexual desire disorder (HSD) or inhibited sexual desire (ISD) suggest that androgens alone are not sufficient for sexual desire. A radioimmunoassay conducted by Stuart, Hammond, & Pett (1987) of blood samples collected from ISD and non-ISD women revealed no significant differences with regard to mean testosterone levels,

although members of the non-ISD group were more dissatisfied with their current mean monthly frequency of intercourse and desired a greater mean frequency than the ISD women. (It should be pointed out, however, that these results are themselves somewhat ambiguous insofar as the desired frequency of intercourse may or may not reflect the frequency of sexual desire; that is, "I want to have sex 10 times a month" is not necessarily the same thing as "10 times a month I experience a desire to have sex".) Similarly, Schreiner-Engel, Schiavi, White, and Ghizzani (1989) found no significant differences between 17 women who met DSM-III-R criteria for HSD and 13 healthy, sexually functional women in such parameters as: mean endocrine values sampled within each of three consecutive menstrual cycle phases; menstrual cycle variation of testosterone, estradiol, progesterone, and prolactin; and in follicular or luteal phase values of bioavailable testosterone. Significant differences were obtained, however, in levels of subjectively experienced sexual desire; specifically, the non-HSD group expressed a greater frequency of interest in and desire for sex, and experienced a greater number of sexual thoughts and fantasies. Apparently, more than circulating levels of testosterone influences whether an individual will experience sexual desire.

Although Carney, Bancroft, and Mathews (1978) found that sexually dysfunctional women who received testosterone in conjunction with counselling reported a greater increase in the number of sexual thoughts experienced on a weekly basis than did women who completed a program consisting of counselling and an anxiety-reducing drug (diazepam), the failure of subsequent researchers to replicate these results (e.g., Mathews, Whitehead, & Kellet, 1983) suggests that testosterone alone (at least in the dosage used by Mathews et al., 1983) is insufficient to produce increases in sexual desire. The differences observed in the Mathews et al. study between women who received testosterone and those who received diazepam may have been due to a decrease in functioning, and hence in sexual desire, caused by the diazepam rather than to an increase in sexual desire in women who received testosterone.

In summary, it appears that a certain baseline level of androgen (in particular, free or bioavailable testosterone) is necessary, although not necessarily sufficient, for the experience of sexual desire in both men and women.

Estrogens

The feminizing hormones known as estrogens are largely secreted by the ovaries, with lesser amounts manufactured in the testes, adrenal cortex, and peripheral tissues (e.g., fat, muscle, kidney, liver, hypothalamus). The placenta is a major source of estrogens during pregnancy (Baird, 1976). The most potent of the naturally occurring estrogenic hormones is *estradiol*, which is derived from metabolism of testosterone and is present in SHBG-bound, albumin-bound, and free forms (the latter is the only portion that exerts biological effects; Mounib et al., 1988). Although weaker and less active, the estrogenic hormones *estrone* and *estriol* also are considered to have important effects upon human sexual function (Fotherby, 1984; Mazenod, Pugeat, & Forest, 1988; Naik & Pennington, 1981). In premenopausal women, plasma levels of estradiol and estrone generally greatly exceed concentrations found in male plasma (Regan, 1996; Regan & Berscheid, 1999). Specifically, throughout the menstrual cycle estradiol levels range from 30 to 570 picograms per milliliter of plasma (pg/ml); in men, plasma concentrations of estradiol range from 10-30 pg/ml. Estrone levels tend to range from 30-220 pg/ml in women, and from 10-70 pg/ml in men.

The role of such estrogens as estradiol and estrone in male sexual interest and desire has yet to be clearly delineated, although there is some evidence that heightened total estrogen levels may result in decreased sexual desire. For example, the administration of exogenous estrogenic preparations appears to be somewhat effective in reducing the sexual interest and fantasies of sex offenders and men who engage in deviant sexual behaviour or who suffer from uncontrollable sexual urges (Bancroft et al., 1974; Cooper, 1986; Field & Williams, 1970; Golla & Hodge, 1949). This treatment, however, has been associated with a potpourri of deleterious side effects, including nausea, vomiting, feminization, and even breast cancer (e.g., Cooper, 1986; Field, 1973; Field & Williams, 1970; Golla & Hodge, 1949; Symmers, 1968; Tennent, Bancroft, & Cass, 1974),

that in themselves may have a negative impact upon the desire to seek out and engage in sexual activity.

Although male sexual desire is presumed to be adversely affected by higher levels of estrogens, some researchers have argued that the presence of estrogens (particularly estradiol) is necessary for normal sexual desire in women (e.g., Benedek & Rubenstein, 1939a, b). However, the majority of evidence suggests that these hormones have little direct influence on sexual desire in women (e.g., Campbell, 1976; Campbell & Whitehead, 1977; Coope, 1976; Furuhjelm, Karlgren, & Carlstrom, 1984; Kane, Lipton, & Ewing, 1969; Leiblum, Bachmann, Kemmann, Colburn, & Schwartzman, 1983; Schreiner-Engel, Schiavi, Smith, & White, 1981; Sherwin, 1985; Studd et al., 1977; Waxenberg, Finkbeiner, Drellich, & Sutherland, 1960; also see Kaplan, 1992). Schreiner-Engel et al. (1989) found no significant differences in the menstrual cycle variation of estradiol between 17 women who met DSM-III-R criteria for hypoactive sexual desire disorder and 13 healthy, sexually functional women even though the two groups differed significantly in terms of levels of subjectively experienced sexual desire. More recently, Dennerstein et al. (1994) found no significant correlation between self-reported sexual desire and level of urinary estrogen in a sample of women assessed daily over the course of one menstrual cycle. In addition, research on the sexual experience of perimenopausal and menopausal women indicates that while sexual desire may decrease after the ovaries cease to function, this decline is not necessarily estrogen-dependent (e.g., Bancroft, 1988). Similarly, the sexual desire of premenopausal women who undergo oophorectomy does not appear to be critically altered by the loss of a major source of estrogens (e.g., Filler & Drezner, 1944; Waxenberg et al., 1959).

The administration of exogenous estrogenic compounds to women suffering from various gynaecologic disorders or menopausal symptoms also does not usually affect sexual desire (e.g., Burger et al., 1984; Furuhjelm et al., 1984; Nathorst-Boos, von Schoultz, & Carlstrom, 1993; Salmon & Geist, 1943; Sherwin et al., 1985; but see Dennerstein & Burrows, 1982; Dennerstein, Burrows, Wood, & Hyman, 1980). Salmon and Geist (1943) treated 30 women who experienced diminished desire associated with menopause, oophorectomy, or gynaecological disturbances (e.g., amenorrhoea) with either an estrogen, an androgen, or an estrogen and an androgen simultaneously. Not one of the 11 women who received the estrogen-alone treatment reported a resurgence of her desire for intercourse, although 8 who had previously noted painful intercourse caused by vaginitis and a subsequent disinterest in that activity did experience relief from both the vaginal symptom and coital discomfort. When an androgen was substituted for the estrogenic preparation, however, all but one of this subsample noticed an appreciable increase in sexual desire. Consistent with these results, the surgically menopausal women in Sherwin et al.'s (1985) study who received an estrogenic preparation (as opposed to an androgenic, androgenic-estrogenic combined, or placebo preparation) reported the lowest levels of sexual desire and the fewest sexual fantasies, even though their plasma estrogen levels during both treatment phases were as high as those of the androgen-estrogen combined group.

In summary, it remains possible that estrogenic hormones indirectly affect female sexual desire by preventing and relieving vaginal symptoms (e.g., dryness, lack of elasticity) that often result in painful intercourse and that may contribute to diminished interest in sexual activity. However, it seems more likely that these hormones are not directly causally related to sexual desire in men or women.

Progesterone

Progesterone is primarily produced by the ovaries (and, during pregnancy, the placenta), with lesser amounts manufactured in the adrenal cortex and testes. In premenopausal healthy women, progesterone concentrations range from mean levels of 31 to 1550 ng/100 ml during the course of the menstrual cycle (Regan, 1996). These levels are higher than those seen in normal, healthy men, for whom plasma concentrations range from between 19 and 30 ng/100 ml (Regan & Berscheid, 1999). Only a small proportion of progesterone is present in the free, bioavailable form.

Much of the research in this area has focused on the effect of progesterone on the sexual desire of

women; as a result, very little is known about the influence of this hormone upon male sexuality. However, some evidence indicates that progesterone may have a dampening effect on male sexual desire. For example, in an uncontrolled study Heller, Laidlaw, Harvey, & Nelson (1958) noted decreased "libido" in 4 males receiving intramuscular progesterone, and progesterone has also been used to treat hypersexuality or reduce excessive sexual desire and urges in men (Money, 1970).

Some researchers speculate that exogenous progesterone may have a similar inhibitory impact upon sexual interest in women (e.g., Bancroft, 1984, 1988; Benedek & Rubenstein, 1939a, b; Greenblatt, McCall, & Torpin, 1941; McCauley & Ehrhardt, 1976). Indeed, the use of oral contraceptives that elevate progesterone throughout the cycle (for example, a combined rather than sequential pill) has been linked with decreased sexual interest and desire (e.g., Huffer, Levin, & Aronson, 1970; Kane et al., 1969; Warner & Bancroft, 1988; but see McCullough, 1974). In addition, subfascially implanted progesterone pellets used to treat a variety of gynaecological disorders have been associated with a marked reduction in sexual desire (e.g., Greenblatt et al., 1942).

Other research, however, suggests that progesterone has no demonstrable effect on female sexual interest (e.g., Abplanalp, Rose, Donnelly, & Livingstone-Vaughan, 1979; Schreiner-Engel et al., 1989). Schreiner-Engel et al. (1989) found no significant differences in the menstrual cycle variation of progesterone between groups of sexually dysfunctional and sexually functional women, although those in the functional group expressed a greater frequency of interest in and desire for sex, and experienced a greater number of sexual thoughts and fantasies.

To further complicate matters, exogenously administered progesterone has been noted to *increase* desire in women under certain circumstances. Bakke (1965) reported that 11 out of a sample of 27 hysterectomized, menopausal women who were administered an estrogenic preparation, a combined estrogen and progestin preparation, and a placebo in a random sequence noticed an increase in sexual interest while on the combined pill that was substantially greater than anything experienced while taking the estrogen-alone or placebo pills.

The effect of progesterone upon human sexual desire, then, is not clear; it may adversely affect male sexual desire, and it may or may not increase or decrease female sexual desire.

Prolactin

Prolactin is produced by the pituitary gland. Mean levels of this hormone tend to range from approximately 9 to 18 ng/ml in women and 5 to 14 ng/ml in men, although normal serum levels for healthy adults of both sexes are generally defined as equal to or less than 30 ng/ml (Regan, 1996). In women, the lactational period following pregnancy and delivery is characterized by elevated levels of prolactin and often suppression of ovarian activity and resulting lowered levels of progesterone and estrogen (Regan & Berscheid, 1999). Prolactin levels also tend to increase slightly at ovulation, and prolactin levels in both men and women appear subject to a diurnal rhythm in which they rise after the onset of sleep and peak in the early morning (Pennington, Naik, & Bevan, 1981).

Most research involving prolactin has focused on behavioural or physiological aspects of sexual function (e.g., intercourse, erection, and ejaculation in men, and intercourse and orgasm in women; for reviews see Drago, 1984; Muller, Musch, & Wolf, 1979; Sadowsky, Antonovsky, Sobel, & Maoz, 1993). A number of researchers, however, have reported an association between elevated prolactin levels and decreased sexual desire (e.g., Lundberg & Hulter, 1991; Walsh & Pullan, 1997). For example, men and women with hyperprolactinemia, a condition characterized by prolactin levels greater than 30-35 ng/ml, frequently report a decrease in sexual interest that subsequently improves when their prolactin levels are reduced by treatment with, for example, bromocriptine (a dopamine agonist that lowers levels of plasma prolactin; e.g., Bancroft, 1984; Bancroft, O'Carroll, McNeilly, & Shaw, 1984; Buckman & Kellner, 1985; Dornan & Malsbury, 1989; Muller et al., 1979; Riley, 1984; Schwartz, Bauman, & Masters, 1982). Chronic renal failure frequently is

associated with both hyperprolactinemia and decreased sexual desire, and men and women effectively treated for the hormonal abnormality tend to report a restoration of their lost interest (e.g., Weizman et al., 1983). Similarly, although many studies involving lactating women have focused on the relation between sexual interest and such physiological changes as altered breast sensitivity and size rather than on hormonal changes (e.g., Hames, 1980), some researchers report that lactating women, like many individuals with elevated prolactin levels, also experience less sexual desire than when not nursing. For example, Kayner and Zagar (1983) found that the majority (62.6%) of their sample of 121 presently or recently lactating women reported experiencing less or no sexual desire while nursing as compared to prepregnancy levels.

This pattern, however, is far from universal. Some men and women with hyperprolactinemia do not experience a loss of sexual interest, while others who do lose interest do not have this alleviated by treatment that successfully reduces prolactin levels (see, for example, Franks, Jacobs, Martin, & Nabarro, 1978; Koppelman, Parry, Hamilton, Alagna, & Loriaux, 1987). For that matter, a normal prolactin level does not insure the presence of sexual desire; hormonal assays have revealed normal prolactin levels in women who report low frequencies of interest in and desire for sex, experience a decreased number of sexual thoughts and fantasies, and express strong dissatisfaction with their perceived low monthly frequency of intercourse (e.g., Alder, Cook, Davidson, West, & Bancroft, 1986; Schreiner-Engel et al., 1989; Stuart et al., 1987). In addition, some reports suggest that the increased levels of prolactin associated with lactation may not have a noticeable effect on sexual desire. A majority (75%) of the 32 women in Kenny's (1973) study, for example, retrospectively reported no effect of the lactational period on sexual desire, and 78% of those who had weaned their children prior to assessment failed to recollect any change in desire after weaning (when prolactin levels had presumably returned to prelactation levels).

What effect, then, does prolactin have on sexual desire? We can glean no clear answer from the available studies, although the majority seem to indicate that prolactin itself may have little direct impact upon desire. It is possible, however, that prolactin may influence desire indirectly, via an effect upon mood or androgen production. Some researchers have hypothesized and provided evidence that prolactin secretion may provoke an increase or decrease in androgen levels (e.g., Eskin, Aspinall, & Segrave-Daly, 1985; Rubin, Gouin, Lubin, Poland, & Pirke, 1976; Zwirska-Korczala, Ostrowska, Zych, & Buntner, 1991). Low plasma testosterone levels have been noted in men with prolactin-secreting pituitary tumours (e.g., Faglia et al., 1977; Friesen, Tolis, Shiu, & Hwang, 1973), and hyperprolactinemia is frequently accompanied by low (in men) or high (in women) levels of plasma testosterone and other androgenic abnormalities (see Carter et al., 1978; Franks et al., 1978; Glickman, Rosenfield, Bergenstal, & Helke, 1982; Goodman, Molitch, Post, & Jackson, 1980; Legros, Chiodera, & Servalis, 1980; Schwartz et al., 1982). Insofar as androgens influence sexual interest, it is possible that the lowered desire seen in some hyperprolactinemic individuals may be due to the effect of elevated prolactin levels on androgen secretion.

Prolactin levels also may affect sexual desire indirectly by influencing mood. Mild depression, anxiety, and hostility have been associated with high levels of prolactin (e.g., Buckman & Kellner, 1985; Fava, Fava, Kellner, Serafini, & Mastrogiacomo, 1981; Kellner, Buckman, Fava, & Pathak, 1984; Koppelman et al., 1987; but see Waterman et al., 1994), and hyperprolactinemic individuals with mood disturbances frequently return to normal mood during treatment with bromocriptine (Buckman & Kellner, 1985; Koppelman et al., 1987). Evidence now suggests that mood (in particular, depressed mood) affects aspects of sexuality including desire and interest (for a review, see Regan & Berscheid, 1999); thus, it is possible that any decreases in sexual desire seen in hyperprolactinemic men and women are the result of mood alterations influenced by abnormally high prolactin secretion.

In summary, the role of prolactin in sexual desire remains unclear, despite numerous studies and substantial interest on the part of the scientific community.

CONCLUSION

There is strong empirical support for the notion that sexual desire in men and women requires certain baseline amounts of the androgens (in particular, free or non-SHBG-bound testosterone). While it is important to recognize endocrine influences on the experience of sexual desire, the precise nature of these effects is unclear. Future research might profitably explore the association between hormonally-mediated life events (e.g., the menstrual cycle, pregnancy, the menopause) and sexual desire, but it will also be important to emphasize the interaction of hormones and other subjective, psychological and social factors in the mediation of sexual desire.

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