
Chapter 16

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Sexual Dysfunction

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Sexual problems are broadly defined as the inability to participate in a sexual relationship as one wishes. In order for the problem to be diagnosed as a sexual dysfunction in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)* (American Psychiatric Association, 2000), the individual needs to report distress and should identify the problem as persistent and recurrent. While people may complain of a variety of sexual concerns, the *DSM-IV-TR* recognizes only four major categories of sexual dysfunction: Sexual Desire, Sexual Arousal, Orgasm, and Sexual Pain Disorders. This chapter provides an overview of the definition, prevalence, etiology, assessment, and treatment of each of the sexual disorders described by the *DSM-IV-TR* that fall within these categories.

Sexual Desire Disorders

HYPOACTIVE SEXUAL DESIRE DISORDER (HSDD)

Definition, Diagnosis, and Prevalence

The *DSM-IV-TR* refers to clinically low levels of sexual desire as Hypoactive Sexual Desire Disorder (HSDD). In order to meet the diagnostic criteria for HSDD the person must experience a persistent or recurrent deficiency or absence of sexual fantasies and desire for sexual activity that causes marked distress or interpersonal difficulty. Whether a person is distressed by his or her level of desire is necessarily impacted by whether his or her partner has similar sexual needs. For example, a couple in which both partners prefer sexual activity only once a month or less would, by most standards, be exhibiting levels of desire below normal. However, because they are well matched in their sexual needs, it is unlikely they would be distressed by their low levels and, therefore, would not meet the *DSM-IV-TR* diagnostic criteria.

The *DSM-IV-TR* criteria for HSDD is based on early models of sexual response outlined by Masters and Johnson (1966) and Kaplan (1979) in which desire is assumed to precede arousal and orgasm in a linear, sequential manner. Clinical experience indicates, however, that oftentimes arousal precedes desire in women. For example, a woman may not necessarily feel a desire to engage in sexual activity but, if approached, she may be receptive to sexual activity and, once engaged, she may then experience a desire for further sexual activity. A consensus panel of 19 experts in female sexual dysfunction selected from five countries was recently assembled to readdress the classification of women's sexual problems (Basson et al., 2003). The panel suggested that a lack of responsive desire (i.e., unwilling or uninterested in engaging in sexual activity when approached) be added to the *DSM-IV-TR* diagnostic criteria. Under this new classification, women who may not have sexual thoughts, fantasies, or seek out sexual interactions but are open to engaging in sexual activity when approached by their partner would not necessarily have HSDD.

In a random probability sample of 1,410 U.S. men and 1,749 U.S. women, Laumann, Gagnon, Michael, and Michaels (1994) reported 32% of women between the ages of 18 and 29 experienced a lack of sexual interest compared to 14% of men in the same age group. Though these estimates for women are somewhat higher than estimated levels of clinically diagnosed HSDD, they are consistent with the notion that desire problems are the number one sexual complaint reported by women. Findings from the Laumann et al. study also revealed a number of interesting gender differences in sexual drive mechanisms. For example, women did not show a change in rates of inhibited desire according to age, whereas men were significantly more likely to report lack of sexual interest as they aged—particularly after age 50. Women did not differ in rates of inhibited desire based on marital status; whereas, married men were significantly less likely to report inhibited desire compared to divorced or never married men. Women—but not men—who had less than a high school level of education reported significantly higher rates of inhibited desire compared to women with more education. Possibly, more educated women are more open to improving sexual communication and sexual knowledge that, in turn, may allow them to better fulfill their sexual needs. African American women reported significantly higher rates of inhibited desire compared to Caucasian or Hispanic women, whereas (among men) there were no ethnic differences in sexual desire (Laumann, Paik, & Rosen, 1999).

Same-sex couples show different patterns of sexual desire compared to heterosexual couples. For example, men in relationships with men are more likely to report low sexual desire when they view their sexual orientation more negatively. The lesbian "death-bed," referring to a complete lack of sexual desire from both partners in lesbian relationships, has recently received much attention from the literature. Women-focused studies have revealed that although lesbian couples may not express interest in more traditional sexual activities including vaginal penetration, they do maintain an active sexual life rich in foreplay and other forms of sexual stimulation (Nichols, 2004).

Factors Associated with HSDD

Cases of low desire in men are often related to medical conditions or pharmacological treatments that affect hormone levels, particularly testosterone. Men receiving testosterone replacement therapy because of a deficient secretion of gonadal hormones show a significant drop in sexual interest when treatment is stopped, and a return of sexual interest when hormone treatment is reinstated. This indicates that very low testosterone

levels may impair sexual desire in men. However, once testosterone levels reach a certain threshold, additional testosterone does not further enhance sexual desire. In other words, testosterone administration to men with normal testosterone levels will not increase sexual desire even if they are experiencing low sexual desire. In adolescent males, higher testosterone levels are associated with increased frequency of sexual fantasies and sexual activity but this relationship does not hold true in adult men. Possibly, during and around puberty internal factors including hormones trigger sexual appetite, while in adulthood external cues such as relationship factors play a more key role in facilitating desire. Some evidence suggests that estrogen and progesterone administration reduces sexual desire in men with excessive or inappropriate desire, although few studies have been published on this topic (Meston & Frohlich, 2000).

In pre-menopausal women, androgens are secreted from both the adrenal glands and the ovaries. Unusually low testosterone levels that result from removal of the adrenal glands (adrenalectomy), removal of the ovaries (oophorectomy), or as a consequence of menopause, impair sexual desire in women. In the case of surgery-related declines in desire, it is often difficult to discriminate between the effects of reduced hormones on sexual desire and the negative psychological factors surrounding the circumstances that led to the surgery (e.g., cancer, polycystic ovaries). There has been recent interest in whether decreases in testosterone resulting from estrogenic oral contraceptive use negatively impacts desire mechanisms in women. Indeed, a number of studies have reported complaints of impaired desire among women on oral contraceptives, and it is well known that oral contraceptives produce substantial increases in sex hormone-binding globulin, which can lower testosterone levels.

Evidence for the role of testosterone in women's sexual desire is also provided by studies showing that testosterone effectively restores sexual desire in women with abnormally low testosterone levels. The enhancing effects of testosterone could be via direct hormonal mechanisms or indirectly by positively impacting mood and overall well-being (Traish & Kim, 2006). As is the case with men, administering testosterone to women with normal testosterone levels does not enhance sexual desire in women and—in the case of women—may lead to a number of undesirable side effects (e.g., acne, facial hair).

It is well known that many psychoactive medications affect sexual drive. Selective serotonin reuptake inhibitors (SSRIs)—used most commonly for treating depression—increase serotonin levels and produce a variety of sexual side effects in both men and women, including decreased desire. Sexual dysfunction secondary to SSRI use is believed to result, in part, from activation of the serotonin₂ receptor. Newer generations of antidepressants that act as antagonists (blockers) at the serotonin₂ receptor (e.g., nefazodone) are associated with fewer sexual side effects. Drugs that facilitate dopamine activity—such as the antiparkinsonian medication levodopa—tend to increase sexual desire in men but the role of dopamine activity in female sexual desire is not known.

HSDD has also been linked with a number of psychosocial factors in both men and women (Kaplan, 1979). Daily hassles such as worrying about children, paying the bills, and high stress jobs are offenders for suppressing sexual desire—as are a multitude of relationship or partner-related issues. In regard to the latter, couples reporting sexual difficulties—compared to nonclinical control couples—have been characterized as having less overall satisfaction within their relationships, an increased number of disagreements, more communication and conflict resolution problems, and more sexual

communication problems including discomfort discussing sexual activities. They also tend to display less playfulness and spontaneity within their relationships, less closeness, intimacy, and feelings of mutual love, and more aversive feelings and thoughts within their sexual interactions. Warmth, caring, and affection within the relationship are undoubtedly linked to feelings of sexual desire.

In a recent study, McCall and Meston (2006) reported four distinct factors that describe triggers or cues for sexual desire in women. These include, emotional bonding cues (e.g., feeling a sense of love with your partner, feeling a sense of commitment from your partner), erotic/explicit cues (e.g., watching an erotic movie, asking for or anticipating sexual activity), visual/proximity cues (e.g., seeing/talking with someone famous, seeing a well toned body), and romantic/implicit cues (e.g., having a romantic dinner with your partner, laughing with a romantic partner). Not surprisingly, women diagnosed with HSDD scored significantly lower than sexually healthy women on each of these domains.

Psychological conditions most commonly associated with a lack of sexual desire include Social Phobia, Obsessive-Compulsive Disorder, Panic Disorder, and Mood Disorders—depression in particular. It is feasible that sexuality becomes of secondary importance when an individual is experiencing substantial distress in other areas of his or her life. With regards to depression, it is feasible that rumination of negative events, a common cognitive aspect of depression, may contribute to the decrease in desire noted in depressed persons by causing an exclusive focus on aspects of sexuality that are unpleasant. Also, it is well known that people with depression are prone to interpret negative events as caused by stable, global causes (Hankin, Fraley, & Abela, 2005) and this cognitive style could certainly negatively affect one's perception of sexuality.

A history of unwanted sexual experiences can also negatively affect sexual desire. Many, but not all, women with a history of childhood sexual abuse fear sexual intimacy, are likely to avoid sexual interactions with a partner, and are less receptive to sexual approaches from their partners (Rellini, 2006). A high proportion of women with a history of childhood sexual abuse also engage in risky sexual behaviors—such as engaging in sex with strangers while intoxicated (Bensley, Eenwyk, & Simmons, 2000). It is unknown whether this behavior is a reflection of high levels of sexual desire, a lack of ability to maintain or enforce physical boundaries, a compulsive act, or some combination of the three.

Assessment and Treatment of HSDD

Diagnosing HSDD is difficult because of the subjective nature of what constitutes sexual desire. Clinicians and researchers have often used frequency of sexual thoughts and fantasies as a measure of desire. Research suggests that this may accurately reflect desire mechanisms in men but not women. Engaging in sexual fantasy seems to be characteristic of women in new relationships, but research strongly suggests that the majority of sexually healthy women in longer-term relationships do not frequently engage in sexual fantasies. Measuring sexual desire according to frequency of sexual interactions is also problematic since this is dependent on partner availability. Also, people engage in sexual activities for a multitude of reasons other than desire (e.g., attempts to please a partner), and often there is a great discrepancy between the amount of sexual desire and the frequency of sexual activities. Comparing a client's sexual desire to the norms derived from the population does not provide an accurate assessment because it ignores

important relational variables and also the diagnostic criteria of distress. Diagnosis of HSDD needs to be carefully considered within the context of the dyadic relationship and must take into consideration factors known to affect sexual functioning—such as the person's age, religion, culture, the length of the relationship, the partner's sexual function, and the context of the person's life.

Assessment of HSDD should comprise a complete sexual, medical, and psychosocial history, which can be obtained through standardized interviews and validated self-administered questionnaires. The clinician should explore the onset of the sexual problem keeping in mind dates of surgeries, medication changes, and diagnoses of medical conditions. Laboratory testing may be warranted, especially for men given the close relationship between androgens and sexual desire. A complete psychosocial history should include: situational problems, relationship history, sexual problems of the partner, mood, sexual satisfaction, and psychological disorders.

Testosterone treatment is effective for restoring desire in men and women with abnormally low levels of testosterone. Psychological treatments for HSDD include education about factors that affect sexual desire, couples exercises (e.g., scheduling times for physical and emotional intimacy), communication training (e.g., opening up about sexual issues and needs), cognitive restructuring of dysfunctional beliefs (e.g., a good sexual experience does not always end with an orgasm), sexual fantasy training (e.g., training people to develop and explore mental imagery), and *sensate focus*. Sensate focus, introduced by Masters and Johnson in the 1970s, is a behavioral technique in which couples learn to focus on the pleasurable sensations that are brought about by touching, while decreasing attention on goal directed sex (e.g., orgasm).

For persons in satisfying relationships, treatment may include identifying potential distracting, negative thoughts and helping them let go of these thoughts during sexual activity. Leiblum and Wiegel (2002) described four such types of distracting thoughts in women: myths and misconceptions (e.g., women are not supposed to enjoy sex), negative emotions, performance anxiety, and body image concerns (e.g., focusing on unattractive aspects of one's body). Behavioral techniques designed to help men and women explore their sexual likes and dislikes—alone or with their partners—can be used to help them associate sexual behaviors with positive affect and experiences. For individuals who are distracted by feelings of shame or embarrassment about their bodies, cognitive restructuring might involve helping them to identify their fears (e.g., a fear of rejection) and dysfunctional beliefs (e.g., "My partner thinks my body isn't sexy") and then test the accuracy of these beliefs through a series of strategically designed behavioral experiments. The experiments aim at reducing avoidance behavior and provide corrective experiences to counteract dysfunctional beliefs. For example, a woman who keeps her clothing on during sex because she feels that her partner would reject her if he saw her naked would be encouraged to incrementally remove pieces of clothing and test the reaction of her partner.

SEXUAL AVERSION

Sexual aversion is conceptualized as a phobic reaction to sexual contact and, in many ways, is more similar to an Anxiety Disorder than to a Sexual Disorder. In the *DSM-IV-TR*, it is defined as the recurrent or persistent extreme avoidance of (or aversion to) all or nearly all genital sexual contact with a sexual partner. It can be so severe that an individual may avoid any type of physical contact including holding hands

for fear that such contact may lead to sexual interaction. There is a paucity of information on the prevalence of Aversion Disorder, but it is generally not thought to be a rare disorder.

Little is known about the etiology of Sexual Aversion Disorder other than people with sexual aversion report high levels of anxiety in anticipation of potential sexual contact. The aversion is not always towards intercourse, but may be towards specific sexual elements such as semen, which, over time, may or may not become more generalized. Clinical reports have found that many women reporting sexual aversion symptoms are survivors of child sexual abuse (Van Berlo & Ensink, 2000).

Sexual Aversion Disorder is commonly treated with anxiety-reduction techniques such as systematic desensitization, which involves creating a hierarchy of sexual activities that provoke increasing levels of anxiety and then exposing the person to the anxiety-producing stimuli while he or she engages in relaxation exercises. After several sessions of pairing the fear-arousing stimuli with a state of relaxation, the person is usually able to imagine the scenario without becoming intensely anxious and can then proceed to the next scenario on the hierarchy. Once the person is able to imagine all of the scenarios on the hierarchy without experiencing substantial distress, the same technique is applied to experiencing the scenarios in real life either alone or with a partner.

Sexual Arousal Disorders

ERECTILE DYSFUNCTION (ED)

Definition, Diagnosis, and Prevalence

ED is defined in the *DSM-IV-TR* as the inability to reach or maintain adequate erection of the penis to engage in intercourse. Patients are diagnosed with ED when their erectile difficulties are exclusively psychogenic in nature or are caused by a combination of psychological and medical factors. Men of all ages occasionally have difficulty obtaining or maintaining an erection but true Erectile Disorder is more common after age 50. Laumann et al. (1999) reported approximately 7% of men aged 18–29 years have erectile problems compared to 18% of men aged 50–59 years. Level of education and ethnicity are not associated with erectile difficulties, but married men are less likely to report erectile problems compared to never married or divorced men (Laumann et al., 1999). It has been estimated that approximately 60 to 80% of ED cases are of organic etiology. However, anxiety, self-confidence, and relationship factors can contribute to the maintenance or exacerbation of the condition even when the etiology is organic.

Factors Associated with Erection and Erectile Dysfunction

Erections are caused by increased blood pressure in the corpora cavernosa (the sinusoid of the erectile tissue) via increased blood inflow and decreased blood outflow. The increment of blood inflow is regulated by the relaxation of the smooth muscles surrounding the arterioles—a phenomenon that allows the arteriole to dilate. Smooth muscle relaxation has been attributed to an increase in parasympathetic activity that causes a release of the neurotransmitters acetylcholine, vasoactive intestinal polypeptide, and nitric oxide. Nitric oxide causes a greater amount of cyclic guanosine monophosphate

(cGMP) to be available in the smooth muscles and this causes the smooth muscles to relax. Normally, cGMP is broken down by enzymes known as phosphodiesterases (PDEs). However, this may be circumvented by inhibiting the activity of these enzymes. Viagra (sildenafil) and other drugs used to treat ED inhibit PDE type 5. In doing so, these drugs enhance the concentration of cGMP, allowing for greater smooth muscle relaxation and therefore improved erection. Detumescence (i.e., loss of erection) occurs with the release of catecholamines during orgasm and ejaculation.

Surgery, metabolic disorders such as diabetes mellitus, alcoholism, hypothyroidism, infectious diseases such as HIV and other viral infections, and pelvic pathologies such as systemic lupus, and vascular problems associated with atherosclerosis and hypertension, are all potential causes of ED. Drugs that decrease dopamine or reduce testosterone production are also implicated in ED. These include antihypertensive medications, antipsychotic drugs, anxiolytics, antiandrogens, anticholesterol agents, and drugs used to regulate heart rate. Antiparkinsonian medications increase dopamine and facilitate erection.

The major psychological contributors to ED as identified by Barlow's (1986) model of sexual dysfunction are anxiety, negative expectations, and spectatoring. Men who are anxious about not being able to have an erection tend to focus on themselves and how they are performing more than on what gives them pleasure. This *spectatoring* increases anxiety that (physiologically) inhibits the relaxation of the smooth muscles necessary for erection and (psychologically) leads to a negative mood state and a focus on negative expectancies. Since the result is impaired erectile response, the man's fears of not being able to perform are confirmed and they are likely to repeat the process in subsequent sexual situations. By contrast, men with normal erectile responding approach sexual situations with positive expectancies and a focus on erotic cues. Consequently, they become aroused and are able to obtain and sustain an erection, which creates a positive feedback loop for future sexual encounters.

Assessment and Treatment of Erectile Dysfunction

The assessment of ED includes identifying the situation(s) surrounding the onset of ED and the potential beliefs that were formed at that time. Beliefs may be specific to the relationship (i.e., a feeling of inadequacy with one specific partner) or generalize to all situations. In cases where ED is the result of a vascular problem, laboratory assessments that measure genital blood inflow and outflow during sexual stimulation may be helpful. Blood outflow can be measured by injecting agents such as papaverine into the penile corpora cavernosa. The substance relaxes the smooth muscles at the base of the penis, which—in presence of normal blood outflow—produces penile erection even without sexual stimulation. When the drug fails to provide the expected erection, it is considered evidence for impairment in vascular mechanisms. Measurement of nocturnal penile erections—which are expected to increase during the REM sleep cycle in sexually healthy men—is another commonly used technique for assessing potential vascular causes of ED. Assays of free and bioavailable serum testosterone are used to rule out abnormal hormone levels.

Treatments for ED include vacuum devices and constriction rings, intracavernosal injections, intraurethral pharmacotherapy, topical pharmacotherapy, oral pharmacotherapy, and penile implants. Vacuum constriction devices are the most safe and least expensive ED treatment. These consist of a tube that is placed over the penis, and a vacuum pump that draws blood into the penile arteries. A constriction ring is placed at

the base of the penis to prevent blood outflow so that the erection is maintained until completion of the sexual act. Intracavernosal injections refer to medications such as papaverine, phentolamine, and prostaglandin E_1 that are injected into the corpus cavernosum of the penis to induce erection. These all act to dilate penile capillaries, allowing blood to flow into the penis. Although intracavernosal injections are effective in approximately 70 to 90% of patients, a large percentage of users discontinue treatment due to the inconvenience, cost, and/or invasiveness of treatment.

The most popular and effective pharmacological treatments for ED are phosphodiesterase type 5 (PDE₅) inhibitors such as sildenafil (Viagra), vardenafil (Levitra), and tadalafil (Cialis). In fact, since Viagra was introduced to the market in 1998, many of these other rather cumbersome and involved treatments have become less popular. Viagra has been estimated to successfully treat two-thirds of men with ED, is well tolerated by a variety of patients, and is an effective treatment for both organic and psychogenic ED. However, despite these drugs' success with facilitating erection, one study reported between 40 and 80% of men with ED who began treatment with Viagra stopped taking the medication (Leiblum, 2002). Some men discontinue Viagra use because, although it enhances their ability to have an erection, this is not always enough to restore overall sexual desire and satisfaction—which may be more closely intertwined with psychological and relational factors. In a recent study, Melnik and Abdo (2005) compared the effects of six months treatment with Viagra to psychotherapy or Viagra plus psychotherapy, and psychotherapy alone outperformed Viagra in terms of decreasing erectile problems. The psychotherapy aimed at developing realistic and positive expectations for the sexual relationship, and also encouraged patients to explore the emotional components linked to ED.

Penile implants are generally considered a last resort treatment technique when tissue damage or deterioration is severe or when other treatments have failed. This may be the case in men with severe diabetes mellitus or who have had radical prostatectomy. Implants can be hydraulic, semirigid, or soft silicone, and consist of two or three cylinders placed in the space normally occupied by the spongy tissue in the penis. The patient's ability to ejaculate after the implant surgery remains in tact; however, the implant does not restore sensitivity or sexual drive that may have been present prior to the onset of ED. Implant surgeries usually result in decreased penis size, which may dissuade some men from undergoing surgery.

FEMALE SEXUAL AROUSAL DISORDER (FSAD)

Definition, Diagnosis, and Prevalence

The *DSM-IV-TR* defines Female Sexual Arousal Disorder (FSAD) exclusively in physiological terms as a persistent or recurrent inability to attain or maintain an adequate lubrication, swelling response until completion of sexual activity. It has been suggested by a committee of experts in the field of women's sexuality that three subtypes of FSAD would more accurately reflect women's experiences with sexual arousal problems (Basson et al., 2003). These are: Subjective Sexual Arousal Disorder, which refers to the absence of feelings of sexual arousal (sexual excitement and sexual pleasure) but vaginal lubrication or other signs of physical response still occur; Genital Sexual Arousal Disorder, which refers to absent or impaired genital sexual arousal (e.g., minimal vaginal lubrication from any type of sexual stimulation and reduced sexual sensations from

caressing genitalia) but psychological sexual excitement still occurs; and Combined Genital and Subjective Arousal Disorder. Most women who complain of arousal problems would meet criteria for the combined category. Though no prevalence data are available on the FSAD subtypes, the estimated lifetime prevalence of problems with general FSAD is 20% (Laumann et al., 1999).

Factors Associated with Women's Sexual Arousal and FSAD

Estrogen is critical for the maintenance of vaginal tissue function and structure, and estrogen deficiency has been linked with various vaginal problems including reduced or delayed lubrication, reduced vaginal blood flow, and increased likelihood of pain during sex. The majority of research on the effects of estrogen on sexual function has been conducted in postmenopausal women and women who have undergone surgery to remove their ovaries (oophorectomy). In post-menopausal women, the reduction in estrogen levels that occurs when the menstrual cycle ends is associated with increased pH levels in the vagina, a reduction or delayed onset of lubrication in response to sexual stimulation, and structural changes to the vagina and vulva such as thinning and reduction of elasticity of the vaginal wall, changes to the vaginal epithelium, and loss of collagen in the vulva (Bachmann, Ebert, & Burd, 1999). All of these changes can lead to arousal difficulties due to a reduction in tissue sensitivity and vaginal lubrication.

As noted earlier, in premenopausal women androgens secreted from the adrenal glands and the ovaries have been linked to desire mechanisms in women. Some speculate that androgens are also involved in female sexual arousal. One study found a positive correlation between testosterone (a form of androgen) and genital arousal in healthy, premenopausal women when levels of testosterone and arousal were compared across the menstrual cycle (Schreiner-Engel, Schiavi, Smith, & White, 1981). A more recent study found that administering testosterone to premenopausal women increased their genital arousal (Tuiten et al., 2002). Nitric oxide—the neurotransmitter involved in male erection—is produced in clitoral tissue and may also be important for women's sexual arousal.

Sympathetic and parasympathetic nervous system arousal (SNS and PNS) both play a role in genital arousal in women but the relationship between the two systems is not well understood. Norepinephrine (NE) is the primary neurotransmitter involved in SNS communication, and when measured after exposure to a sexually arousing film, blood levels of NE are higher than prefilm levels (Exton et al., 2000). Women with spinal cord injuries between areas T10 and T12 in the spinal cord show a lack of lubrication during psychological sexual arousal (Berard, 1989). This is the area of the spinal cord where sympathetic nerves project to the genital region. A number of laboratory studies have also provided evidence for the role of SNS involvement in women's sexual arousal. For example, Hoon, Wincze, and Hoon (1977) demonstrated that when women were shown an anxiety-evoking film prior to an erotic film, they experienced higher levels of vaginal blood volume (a measure of genital engorgement) than when they were shown a neutral (i.e., travel) film prior to an erotic film. Anxiety-evoking films are likely to increase SNS activation. Recent research has shown a curvilinear relationship between acute anxiety and vaginal engorgement, with the optimal arousal response occurring at moderate levels of anxiety (Bradford & Meston, 2006). Meston and colleagues have completed several studies on the effects of exercise (Meston & Gorzalka, 1996) and ephedrine (Meston & Heiman,

1998) on sexual arousal, two manipulation techniques designed to increase SNS activity. These studies also support the notion that there is an optimal level of SNS arousal that is necessary for adequate genital arousal in women. Mechanisms that interfere with normal SNS activity—such as stress—can negatively impact a woman's ability to become aroused.

Given the high coexistence of sexual desire and arousal problems in women, it is not surprising that the myriad of factors affecting women's sexual desire noted earlier also affect women's sexual arousal. According to the Dual-Control Model proposed by Bancroft and colleagues (Bancroft & Janssen, 2000), sexual arousal is the combination of both excitatory and inhibitory forces. Five main themes have been described as potential inhibitors or enhancers of sexual arousal for women ages 18 to 84 years: feelings about one's body, negative consequences of sexual activity (e.g., bad reputation, pregnancy), feeling desired and accepted by a sexual partner, feeling used by a sexual partner, and negative mood (Graham, Sanders, Milhausen, & McBride, 2004).

Assessment and Treatment of FSAD

The assessment of FSAD is similar to that of HSDD in women and should include a comprehensive sexual, medical, and psychosocial history. Levels of physiological sexual arousal can be assessed indirectly using a vaginal photoplethysmograph to assess vaginal blood engorgement, sonograms (pictures of internal organs derived by sound waves bouncing off organs and other tissues) and fMRI (imaging techniques that track changes in blood concentration in inner organs) to assess blood engorgement in the genitals. These techniques are more commonly used for research purposes than as diagnostic tools.

Many of the psychological treatments described earlier to treat HSDD are used to treat psychological feelings of impaired sexual arousal. Physiological aspects of FSAD are most commonly treated with topical lubricants that help mask impairments in vaginal lubrication. They do not, however, enhance genital/clitoral blood flow or genital sensations that are often decreased with FSAD, and they do not directly impact psychological sexual arousal.

Currently, there are no Food and Drug Administration (FDA) approved pharmacological treatments for FSAD. However, since the enormous success of using PDE5 inhibitors (e.g., sildenafil, levitra, cialis) for treating Male Arousal Disorder (ED), a number of pharmaceutical companies have examined whether these and similar vasodilator drugs may also be effective for treating FSAD. Evidence from limited placebo-controlled studies indicates Viagra increases genital engorgement in healthy, premenopausal women (Laan et al., 2002) and in postmenopausal women with severe levels of genital arousal concerns (Basson & Brotto, 2003). Despite reports of increased physiological sexual arousal, studies in general have not found that these drugs positively impact a woman's psychological experience of sexual arousal. This suggests that—for women—psychological factors such as relationship satisfaction, mood state, and sexual scenarios may play a more important role in assessing feelings of sexual desire and arousal than do physiological genital cues. The EROS clitoral therapy device is an FDA-approved treatment for women's sexual concerns. This small hand held device increases vasocongestion in the clitoral and labial region via a suction mechanism and has been reported to increase vaginal lubrication and sensation (Billups et al., 2001).

Orgasm

MALE ORGASMIC DISORDER

Delayed or Inhibited Ejaculation

Delayed or inhibited ejaculation following normal sexual arousal and adequate sexual stimulation is a rare condition, affecting up to only 3% of the male population (Laumann et al., 1994). Impairments in orgasm and ejaculation have often been noted as a side effect of SSRI antidepressant use and has led some researchers to speculate that serotonin may play a role in the etiology of this disorder. In most cases, however, delayed or inhibited ejaculation tends to be psychological rather than physiological, and performance anxiety seems to play a major role. Also, rigid views about sexuality—such as the belief that orgasm is the only way to experience sexual satisfaction—can direct the focus of attention during sexual activity to reaching an orgasm and this may distract a man from experiencing sexual pleasure, which can cause impaired ejaculation and orgasm. Lack of communication about sexual likes and dislikes between partners, and feeling uncomfortable with one's partner are typical relational factors that may prevent a man from enjoying sexual interactions with his partner and impair ejaculatory ability. Treatment for delayed or inhibited ejaculation generally includes helping the couple to break their focus on orgasm and refocus their attention to sexual pleasure and intimacy.

Premature Ejaculation

Definition, Diagnosis, and Prevalence. The *DSM-IV-TR* defines premature ejaculation (PE) as ejaculation that occurs with limited stimulation before, or shortly after, penetration and sooner than the man desires. An important criterion for this condition is the feeling that the man does not have control over ejaculation and this causes him distress. The time from penetration to ejaculation (ejaculation latency) varies greatly between men, with 10 minutes being the average for men with no sexual problems. An individual with PE tends to ejaculate within the first minute of intercourse, with the majority of men reporting an average of 15 seconds or 15 thrusts of intercourse before ejaculation. At times, a man may report distress because he is unable to prevent ejaculation for 20 or 30 minutes. In this instance, the diagnosis of PE is not warranted even if the individual reports high levels of distress.

PE is the most commonly reported Sexual Disorder in men, with approximately 30% of men in the United States reporting PE in the previous year (Laumann et al., 1994). Unlike ED, this condition has been estimated to affect younger men more than older men. As many as 40% of men under 40 years of age and only 10% of men over age 70 have been estimated to experience PE (Corona et al., 2004). The cause of PE is usually assumed to be psychological rather than physiological, although both medical and psychotherapy techniques have been developed to treat this problem.

Factors Associated with Ejaculation and PE. During the first stage of ejaculation (sperm emission), sperm is emitted from the epididymus into the vas deferens. This process is controlled by the contraction of smooth muscles, which is generated by the sympathetic branch of the autonomic nervous system. After sperm emission, the individual has the subjective experience that ejaculation is inevitable—known as the “point of inevitable ejaculation” or, more commonly, “the point of no return.” The striate

muscles surrounding the spongy tissue, the cavernous tissue, and in the pelvic floor, contract rhythmically causing ejaculation to occur. Usually, the subjective experience of orgasm is associated with the contractions of the striate muscles and—in most men—emission, ejaculation, and orgasm are interconnected. For a small portion of men, however, these phenomena are independent. For example, some men train themselves to have the subjective experience of orgasm without ejaculation and some men with PE experience emission without ejaculation.

The precise cause of PE is not known, but it can arise from a deficiency in any of the afferent or efferent circuits involved in the ejaculatory process. For what concerns the sensory (afferent) circuits, researchers have postulated that men with PE have a lower sensitivity threshold such that less stimulation is needed to attain ejaculation. This explanation cannot account for all cases of PE, however, as studies show that PE exists in men with both high and low sensitivity thresholds. It has also been proposed that men with PE may respond with a higher level of arousal to sexual stimuli (hyperarousability). Again, this explanation cannot account for all cases of PE. One psychophysiological study (Rowland & Slob, 1997) that measured penile rigidity in the laboratory showed that men with PE had a weaker genital response to visual stimuli compared to men with no sexual dysfunction, but had a comparable genital response to men with no sexual dysfunction during tactile plus visual stimuli.

Anxiety has most frequently been hypothesized to be the primary cause and maintaining factor for PE. Anxiety increases sympathetic nervous system activity, which is involved in semen emission, and, thus, high levels of anxiety could feasibly accelerate ejaculation. Laboratory studies have generally not shown significant differences in levels of anxiety reported by men with and without PE. One psychological variable that has shown significant differences between men with and without PE is perceived control over ejaculation. During exposure to visual and tactile stimuli, men with and without PE showed comparable degrees of genital sexual arousal (measured in penile circumference), but men with PE reported significantly less control over their ejaculation. A greater understanding of the meaning men attribute to ejaculatory control may provide important insight into the psychological factors involved in this disorder.

Assessment and Treatment of PE. A thorough assessment of PE includes measuring three factors: length of time from penetration to ejaculation (ejaculation latency), subjective feelings of control over ejaculation, and personal and relational distress caused by the condition. Usually these dimensions of PE are assessed with retrospective self-reports provided by the patient. Sometimes the patient is asked to use a chronometer to measure the time from insertion to ejaculation or to have their partner provide an estimate of the man's ejaculatory latency in order to help increase measurement reliability.

The most commonly used psychotherapy techniques for increasing ejaculatory latency are the squeeze technique developed by Masters and Johnson (1970) and the pause technique (Kaplan, 1989). The squeeze technique consists of engaging in sexual stimulation alone or with a partner for as long as possible before ejaculation. Before reaching the point of inevitable ejaculation the man is instructed to stop the activity and apply tactile pressure to the penile glands to decrease the urge to ejaculate but not to the point that he completely loses his erection. When the urge has subsided, the man resumes masturbation or intercourse stopping as many times as needed in order to delay ejaculation. The pause technique is similar to the squeeze technique with the exception that no pressure is applied to the penis. At times, clinicians may suggest using a PDE₅

inhibitor (e.g., Viagra) along with these techniques so that the man can practice delaying ejaculation without worrying about maintaining an erection.

Medical treatments include the use of topical anesthetics to diminish sensitivity used in combination with condoms (to prevent the partner's genitals from being anesthetized). SSRIs such as sertraline, fluoxetine, and paroxetine have been used because of their known side effects of delaying or inhibiting orgasm. In men with PE, there is some evidence these drugs increase ejaculation latency and sexual pleasure and satisfaction.

FEMALE ORGASMIC DISORDER (FOD)

Definition, Diagnosis, and Prevalence

Female Orgasmic Disorder (FOD) is diagnosed when the woman experiences persistent or recurrent delay in—or absence of—orgasm following a normal sexual excitement phase. In order to meet *DSM-IV-TR* diagnostic criteria, the woman's orgasmic capacity must be less than what would be reasonable for her age, sexual experience, and adequacy of sexual stimulation she receives (APA, 2000). Clinical consensus is that women who can achieve orgasm through masturbation or through manual stimulation with a partner, but not from intercourse alone, would not meet diagnostic criteria for FOD.

Orgasm difficulties are the second most frequently reported sexual problems for women in the United States, with between 22 to 28% of women ages 18 to 59 years reporting they are unable to attain orgasm (Laumann et al., 1994). Young women (18 to 24 years) show rates of orgasm lower than older women for both orgasm with a partner and orgasm during masturbation (Laumann et al., 1994). This is likely due to age differences in sexual experience.

Factors Associated with Women's Orgasm and FOD

In most cases, FOD is thought to stem from psychological causes and there are no specific physiological factors linked to orgasmic dysfunction in women. Impairments in endocrine, nervous system, or brain mechanisms involved in female orgasm, however, may cause orgasmic dysfunction in women. Studies examining blood plasma levels of neuromodulators before, during, and after orgasm suggest that epinephrine and norepinephrine levels peak during orgasm in normally functioning women (Exton et al., 2000). Among orgasmic women, oxytocin levels are positively correlated with subjective intensity of orgasm and prolactin levels are elevated for up to 60 minutes following orgasm (Meston & Frohlich, 2000). Studies in humans suggest that the paraventricular nucleus of the hypothalamus—an area of the brain that produces oxytocin—is involved in the orgasmic response (McKenna, 1999). Impairments in any of these systems could feasibly lead to FOD.

Medical conditions that affect women's orgasmic ability include: damage to the sacral/pelvic nerves, multiple sclerosis, Parkinson's disease, epilepsy, hysterectomy complications, vulvodynia, diabetes mellitus, hypothalamus-pituitary disorders, and sickle cell anemia. A number of psychotherapeutic drugs have also been noted to affect the ability of women to attain orgasm. The SSRIs frequently affect orgasmic functioning, leading to delayed orgasm or a complete inability to reach orgasm. There is variability, however, in that some antidepressants have been associated with impaired orgasm more often than others. This seems to be related to which specific serotonin receptor subtype

is being activated. As noted earlier, drugs that inhibit serotonin activity at the serotonin₂ receptor (e.g., nefazodone, cyproheptadine) cause fewer sexual side effects in women (Meston, Hull, Levin, & Sipski, 2004).

The psychological factors associated with FOD include sexual guilt, anxiety related to sex, childhood loss or separation from the father, and relationship issues (Meston et al., 2004). Sexual guilt can affect orgasmic abilities by increasing anxiety and discomfort during sex, and also by distracting a woman from what gives her pleasure. Women who strictly abide to the values of western religions sometimes view sexual pleasure as a sin. Sins are later connected with a sense of shame and guilt, which could produce negative affect and cause distracting thoughts during sexual activities. Indeed, a reduction in sexual guilt has been associated with improvements in orgasmic abilities (Shotly et al., 1984). Women who initiate and are more active participants during sexual activities report more frequent orgasms, most likely because being active allows women to assume positions that can provide a greater sense of sexual pleasure. More frequent masturbation and sexual activities are associated with more frequent orgasms. It is likely that women who engage in more sexual activities have a greater understanding of what gives them sexual pleasure and this can help them reach orgasm more easily. Culturally, women who live in societies that value female orgasm tend to have more orgasms than women living in societies that discourage the concept of sexual pleasure for women (Meston et al. (2004). Examples of societies that foster sexual pleasure for women and expect them to enjoy intercourse include the Mundugumor and the Manguaia. Manguaian women are taught to have orgasms, hopefully two or three to each one of her male partner's, and to try to attain mutual orgasm. Manguaian males who are not able to give their partners multiple orgasms are not held in high esteem. At the opposite end of the spectrum are societies that assume women will have no pleasure from coitus and that the female orgasm does not exist. The Arapesh are such a society. In fact, they do not even have a word in their language for the female orgasm. It is feasible that women in societies that promote women's sexual pleasure are more likely to experiment and, therefore, learn about what facilitates their ability to have an orgasm. It may also be that in societies where sexual pleasure is discouraged it may be shameful to admit to having an orgasm.

Assessment and Treatment of FOD

Assessment of FOD involves a comprehensive sexual, medical, and psychosocial history similar to that used for assessing HSDD and FSAD. It is important for the clinician to determine whether the woman is unable to attain orgasm in all situations or just with a certain partner or during certain intercourse positions or sexual techniques because this information may determine the type of therapy she receives. In general, sex therapy for FOD focuses on promoting healthy changes in attitudes and sexually relevant thoughts, decreasing anxiety, and increasing orgasmic ability and satisfaction. Sensate focus and systematic desensitization (described earlier) are used to treat FOD when anxiety seems to play a role. Sex education and communication skills training are often included as adjuncts to treatment. Kegel exercises (Kegel, 1952), which involve tightening and relaxing the pubococcygeous muscle, are also sometimes included as part of a treatment regime. Feasibly, they could help facilitate orgasm by increasing blood flow to the genitals, or by helping the woman become more aware of and comfortable with her genitals.

To date, the most efficacious treatment for FOD is directed masturbation (DM). The first step of DM involves having the woman visually examine her nude body with the help of a mirror and diagrams of female genital anatomy. She is then instructed to explore her genitals using touch with an emphasis on locating sensitive areas that produce feelings of pleasure. Once pleasure-producing areas are located, the woman is instructed to concentrate on manual stimulation of these areas and to increase the intensity and duration until "something happens." The use of topical lubricants, vibrators, and erotic videotapes are often incorporated into the exercises. Next, once the woman is able to attain orgasm alone, her partner is usually included in the sessions in order to desensitize her to displaying arousal and orgasm in his presence, and to educate the partner on how to provide her with effective stimulation. DM has been shown to effectively treat FOD, with some studies reporting a 100% success rate (Meston et al., 2004). Given masturbation can be performed alone, any anxiety that may be associated with partner evaluation is necessarily eliminated.

For women who have orgasm difficulties resulting from hysterectomy and oophorectomy, combined estrogen and testosterone therapy has been shown to enhance orgasmic ability (Shifren et al., 2000). A number of psychotherapeutic drugs have been used to try to eliminate orgasm problems that are secondary to antidepressant drug treatments. Results from placebo-controlled studies, to date, suggest none of the drugs enhance orgasmic ability better than placebo.

Sexual Pain Disorders

DEFINITION, DIAGNOSIS, AND PREVALENCE

Sexual pain can affect men as well as women—albeit much less frequently—and very little is known about sexual pain in men. In this chapter we discuss sexual pain only as it pertains to women. The *DSM-IV-TR* Sexual Pain Disorders include Dyspareunia and Vaginismus. Dyspareunia is defined as persistent and recurrent genital pain during sexual activity. The pain may also occur in situations other than sexual encounters, such as gynecological examinations. Dyspareunia is usually described by women as a sharp, dull, burning, or shooting pain, and can be either localized or generalized. Most often, the pain is considered a superficial pain in that it is associated with the vulva or entrance to the vagina. It may, however, also be experienced as a deeper pain in the abdomen or internal organs. Laumann et al. (1999) reported that approximately 16% of American women reported persistent or recurrent sexual pain in the past year. Sexual pain was noted to be three times more likely among women in the 18–29 age range than those in the 50–59 age range. Poor health, lower education, low family income, high stress, more frequent emotional problems, and the presence of urinary tract symptoms are more common among women with sexual pain.

Vaginismus is defined by the *DSM-IV-TR* as a repeated and persistent involuntary spasm of the vaginal muscles that interferes with intercourse. Although it is clear that contraction of the pelvic floor musculature can prevent vaginal penetration in women, recent empirical work has demonstrated that vaginal spasms can also occur in women who do not report difficulty with vaginal penetration, and not all women who have difficulty with vaginal penetration experience vaginal spasms (Reissing, Binik, Khalife, Cohen, & Amsel, 2004). Also of note is the fact that, although Vaginismus is classified as a

Pain Disorder and appears to be associated with genital pain in most cases (Ter Kuile, Van Lankveld, Vlieland, Willekes, & Weigenborg, 2005), the presence of pain is not required to make a diagnosis of Vaginismus. Epidemiological studies generally exclude questions about Vaginismus; thus, the prevalence of the disorder is not well established. However, it has been estimated to be between 1% and 6% (Lewis et al., 2004).

FACTORS ASSOCIATED WITH DYSPAREUNIA AND VAGINISMUS

Dyspareunia may result from a variety of medical conditions and anatomical complications that should be ruled out by medical examination. Superficial pain may be a symptom of dermatological disorders affecting the external genitalia, vaginal atrophy, anatomical variations, urinary tract infections, injury, and other diseases and infections of the vulva. The majority of women who experience superficial sexual pain show a reliable symptom pattern that includes sensitivity to touch and pressure of the vulvar vestibule, a region bounded by the inner labia minora, the frenulum of the clitoris, and the lower portion of the vaginal opening. Touch or pressure to these areas evokes a sharp, burning pain (Pukall, Payne, Kao, Khalife, & Binik, 2005). This disorder is known as Vulvar Vestibulitis Syndrome, and it is considered separate from other pain syndromes of the vulva. The etiology of Vulvar Vestibulitis Syndrome is uncertain, but women with the disorder often have a history of yeast infections and may have had significant hormonal events in adolescence, including early onset of menstruation and early use of oral contraceptives (Pukall et al., 2005).

Deep sexual pain may result from uterine fibroids, endometriosis, urinary disease, and ovarian disease, among other conditions. Sensitization of the neurons in the spinal cord and in parts of the brain has been postulated as one of the most likely causes of Dyspareunia. According to this theory, intense stimulation of peripheral tissue that occurs because of a trauma or because repetitive abrasive stimulation can sensitize the neurons that bring the pain information to the brain. Consequently, the sensitized neurons require less stimulation to be activated, or they may even be activated without the presence of stimulation. Thus, the individual may feel pain after only a slight touch or even in the presence of no touch. Low pain threshold has been identified as a potential correlate of women with Dyspareunia (Pukal, Reissing, Binik, Khalife, & Abbott, 2000). The low pain thresholds in women with Dyspareunia is not specific to the genital area but, rather, it includes the overall body which supports some theorists' views that Dyspareunia should be classified as a Pain Disorder rather than a Sexual Disorder (Binik, Meana, Berkley, & Kalippe, 1999). Indeed, sexual pain shares many etiological similarities with chronic low back pain and other chronic pain syndromes.

The fear of pain and anxiety has been postulated as both a psychological symptom and cause of Dyspareunia. Empirical studies have indeed found a strong association between the presence of sexual pain and anxiety, although the degree of anxiety is not correlated with the intensity of pain, and not all women who experience sexual anxiety experience sexual pain. Women with Dyspareunia tend to fear sexual interactions and show more phobic anxiety than do women with no sexual pain. Payne, Binik, Amsel, and Khalife (2005) found that women with Vulvar Vestibulitis Syndrome reported hypervigilance for sexual pain, and displayed an attentional bias toward pain-related stimuli on an emotional Stroop task when compared to matched control women without Vulvar Vestibulitis Syndrome.

Depression has also been frequently associated with Dyspareunia; however, longitudinal studies have failed to find a direct relationship between depression and sexual pain. It is likely that women who are more depressed are more likely to report pain in general and sexual pain specifically, but there is no evidence at this point that depression *causes* sexual pain or vice versa. Negative cognitions such as: "My partner will leave me," "I am a failure as a woman," and "I must be tearing inside," are commonly reported by women with sexual pain. From a relational point of view, women with dyspareunia report more pain when their relational distress increases, an indication that sexual pain may be partially associated with negative feelings between partners.

The precise factors associated with Vaginismus are unknown. Experts have proposed that Vaginismus may be a physiological response to an intense pain. That is, the hypothesized vaginal spasm in Vaginismus could be an automatic reaction of the body to protect itself from an expected pain. Indeed, the comorbidity between Vaginismus and Dyspareunia is relatively high (Basson & Riley, 1994). Sexual trauma has been linked to Vaginismus in the empirical literature, but the data to support this association are inconsistent.

ASSESSMENT AND TREATMENT OF DYSPAREUNIA AND VAGINISMUS

The assessment of Dyspareunia should include an accurate description of the location, intensity, quality, duration, and time course of the pain, the degree of interference it has with sexuality, a summary of what elicits the pain (both sexual and nonsexual behaviors), and the meaning attributed to the pain. Assessment usually requires a gynecological examination to help identify the specific area(s) of the pain. Vaginismus is diagnosed if (a) the woman reports she has never been able to have intercourse after at least 10 attempts, and (b) she has showed active avoidance. Active avoidance is either less than one attempt at intercourse every two months, the inability to have a complete pelvic exam or the inability to use tampons.

Treatments for Dyspareunia include cognitive-behavioral therapy, electromyographic feedback, and vestibulectomy. Topical anesthetics and other medications are also sometimes used to alleviate genital pain, but well controlled studies examining their long-term effectiveness are currently lacking. Cognitive-behavioral therapy generally includes educating the woman about sexual pain, the effect it has on sexual desire, arousal, and orgasm, and the factors that maintain the pain. Often cognitive restructuring exercises are used to help the women identify faulty cognitions (e.g., "If I have sex my vagina may tear apart") and to replace them with more accurate beliefs (e.g., "My vagina is made of stretchable muscles that stretch out during intercourse"). Bergeron et al. (2001) found that eight sessions of group cognitive behavior therapy for Vulvar Vestibulitis Syndrome significantly reduced genital pain from pre- to post-treatment, with 39% of women endorsing great improvement or complete pain relief at the six-month follow-up interval.

Electromyographic biofeedback consists of providing information to the woman about the tension in her pelvic floor muscles with the goal of helping her to maintain a relaxed pelvic musculature state during sexual activity. This technique was developed by Glazer, Rodke, Swencionis, Hertz, and Young (1995), who observed a relationship between Vulvar Vestibulitis Syndrome and abnormal responding of the pelvic floor musculature. Evidence suggests that the pelvic floor training approach significantly reduces Vulvar Vestibulitis Syndrome pain and may occasionally eliminate it altogether.

(e.g., Bergeron et al., 2001). Vestibulectomy, an outpatient procedure that involves removal of vulvar vestibular tissue, has also been shown to significantly reduce or completely alleviate genital pain among the majority of recipients (Goldstein & Goldstein, 2006).

Treatment for Vaginismus includes many of the same elements as those employed for Dyspareunia. In addition, relaxation exercises and vaginal dilation exercises, which involve having the women insert progressively larger dilation cones into her vagina, are used to teach the woman to pair thoughts of vaginal penetration with positive affect and muscular relaxation.

Summary and Future Directions

In summary, this chapter provided an overview of the four major categories of sexual concerns in men and women as outlined by the *DSM-IV-TR*. It is apparent that biological, psychological, and social factors all play a prominent role in the etiology of sexual dysfunctions in men and women and must be carefully considered both in assessment and treatment. To date, efficacious treatments exist for Orgasm Disorders in men (Premature Ejaculation Disorder) and women (Female Orgasmic Disorder), for Arousal Disorders in men (Erectile Disorder) and—to a more limited extent—for Pain Disorders (Vaginismus, Dyspareunia) in women. Sexual Desire Disorders remain the most challenging of sexual disorders to treat perhaps due to the subjective nature of what constitutes desire, and the vastly individual nature of what leads a person to desire sexual activity.

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