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The Psychobiology of Sexual and Gender Identity Disorders

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INTRODUCTION

Interest in human sexual function has increased in the past decade, in large part as a result of increased recognition of the sexual side effects of various medications, the high incidence of sexual dysfunction among men and women, and the highly publicized success of some treatments for sexual dysfunction (e.g., Viagra for erectile dysfunction). This paper will describe the present knowledge of the endocrine, neurotransmitter, and central and peripheral nervous system mechanisms governing sexual function and dysfunction. The primary focus will be the underlying physiological processes although it should be noted at the outset that it would be misleading to assume that sexual dysfunction is best conceptualized in this manner.

Psychological problems, such as depression or anxiety, and relationship issues, such as marital discord or stress, can have a profound effect on sexual functioning. Although such cognitive and emotional factors are often integral to a sexual problem, these aspects will be reviewed only briefly here.

SEXUAL DESIRE DISORDERS

Hypoactive Sexual Desire Disorder

Sexual desire is commonly defined as the broad interest in sexual objects or experiences. One of the difficulties in diagnosing inhibited desire is determining exactly what constitutes low desire. Sexual desire cannot be measured exclusively by frequency of sexual activity—a person may desire sexual activity a great deal more or less often than their actual level of activity. It is problematic to measure sexual desire based on a discrepancy between partners; a man who desires sexual activity once a day may be frustrated by a partner who desires sexual activity twice a week, yet both partners have a level of sexual desire that falls within the normal range. Because there is no objective physiological criterion for desire, it is generally inferred by self-reported frequency of sexual thoughts, fantasies, dreams, wishes, and interest in initiating and/or engaging in sexual experiences. However, it is also problematic to diagnose hypoactive sexual desire based on a simple comparison with typical levels of desire. A couple who both prefer sexual activity only once a month would be exhibiting levels of desire below normal, yet it is unlikely that they would be unsatisfied with their degree of activity (LoPiccolo and Friedman, 1988). In order to meet the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV), criteria for hypoactive sexual desire disorder, the person must not only experience a persistent or recurrent deficiency or absence of sexual fantasies and desire for sexual activity, but the situation must create marked distress or interpersonal difficulty—indeed, it should be noted at the outset that in order to be diagnosed with any of the sexual disorders a person must be experiencing significant distress or interpersonal difficulty (American Psychiatric Association, 1994).

Hypoactive sexual desire disorder is much more common in women than in men. Thirty-two percent of women between the ages of 18 and 29 years old reported a lack of sexual interest compared to 14% of men in the same age group. Women did not demonstrate a change in rates of inhibited desire according to age while men were significantly more likely to report lack of sexual interest as they aged, particularly after age 50 years old. 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 who had less than a high school level of education reported significantly higher rates of inhibited desire compared to women with more education. Perhaps more educated women are more open to improving sexual communication and sexual knowledge. Exploring what is sexually pleasurable, and communicating sexual needs are techniques used for enhancing sexual desire. Unlike women, men showed no significant differences in desire according to education. African-American women reported significantly higher rates of inhibited desire compared to Caucasian or Hispanic women whereas men demonstrated no ethnic differences in sexual desire (Laumann et al., 1999).

Physiological Factors

Cases of low desire in men are often related to medical conditions or treatments that affect hormone levels. Hypogonadal men (i.e., men with deficient secretion of gonadal hormones) receiving testosterone replacement therapy demonstrated a significant drop in sexual interest following removal of the hormone treatment, and a return in sexual interest when the hormone treatment was resumed. This indicates that very low testosterone levels may impair sexual desire in men. Once testosterone levels reach a certain threshold, additional testosterone does not affect sexual desire—thus, testosterone administration to a male with normal testosterone levels will not increase 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. Perhaps during and around puberty internal factors (e.g., hormones) trigger sexual appetite while in adulthood external cues (e.g., relationship factors) play more of a central role. Some evidence suggests that oestrogen and progesterone administration reduces sexual desire in men with excessive or inappropriate desire,

although few studies have been published on this topic (Meston and Frohlich, 2000).

Unusually low testosterone levels that result from removal of the adrenal glands (adrenalectomy), removal of the ovaries (oophorectomy), or as a consequence of menopause, may impair sexual desire in women. Testosterone is effective in restoring sexual desire in these women with abnormally low testosterone levels. It should be noted that most women with hypoactive sexual desire disorder do not have abnormally low testosterone levels and administering exogenous testosterone to women with normal testosterone levels does not enhance sexual desire and can lead to a number of adverse side effects (e.g., acne, facial hair). Oestrogen levels do not significantly affect sexual desire in women and evidence is mixed regarding the affects of progesterone administration on sexual desire in women. Some evidence suggests that increases in prolactin levels that occur with breast-feeding may diminish sexual desire in women but myriad other psychological factors (e.g., post-partum depression) could account for these changes (Meston and Frohlich, 2000).

A variety of psychoactive medications affect sexual drive. Selective serotonin reuptake inhibitors (SSRIs, used most commonly for treating depression), which acutely increase the amount of serotonin in the synapse, produce a variety of sexual side effects including diminished libido in both men and women. Sexual dysfunction secondary to SSRI use is believed to result from the activation of the serotonin$_2$ receptor, although it is unclear whether activation of this specific receptor type is responsible for SSRI-induced loss of sexual drive *per se*.

Drugs that facilitate dopamine activity, such as the antiparkinsonian medication levodopa, tend to increase sexual desire in men. Dopamine activity also increases sexual drive in animals; selective dopamine agonists and apomorphine increased mounting behaviour in male rats (for review, see Meston and Frohlich, 2000). The role of dopamine activity in female sexual desire is not known although one report described a case of a middle-aged woman who exhibited increased sexual behaviour while receiving antiparkinsonian medication, and a return to normal sexual behaviour when the dosage was decreased (Uitti *et al.*, 1989). Long-term opioid (e.g., heroin) use diminishes libido in men and women, perhaps due to the testosterone and luteinizing hormone reducing properties of these drugs (Meston and Frohlich, 2000).

Although it is generally believed that sexual drive is controlled by the central nervous system, the specific brain regions and mechanisms controlling drive are not well understood. In male rats, lesion to the medial preoptic area impairs copulatory behaviour by disrupting the animal's ability to identify potential partners. Some evidence suggests that the medial amygdala plays an important role in sexual motivation in males. Large temporal lobe lesions have been reported to produce hypersexuality, although it is believed that damage to inhibitory neurons in the pyriform cortex may be responsible for these results (McKenna, 1999). It is feasible that activity in one or more of these regions may be abnormal in cases of hypoactive sexual desire.

Psychological Factors

Daily hassles (e.g., worrying about childcare, bills, etc.) and high stress jobs are probably the worst offenders for suppressing sexual desire, as are a multitude of relationship or partner-related issues. If the couple is experiencing conflict, one or both partners may experience a drop in desire. One member of a couple may experience a drop in desire because he or she no longer feels attracted to his or her partner. This typically occurs when the partner undergoes a dramatic change in appearance (e.g., significant weight gain). A person may experience a drop in sexual desire if their partner is unwilling to experiment sexually or lacks sexual skill,

making the sexual experience frustrating, unpleasant, or in other ways unappealing. Differences in the desired amount of emotional intimacy or time spent together can impair sexual desire; one person may feel frustrated by the lack of closeness while the other may feel suffocated. Some people fear intimacy, perhaps as a result of being hurt in the past, and thus may have little desire for intimacy in the form of sexual activity. If one partner has more power in the relationship, the other partner may feel bullied or harassed, and may experience a drop in desire (LoPiccolo and Friedman, 1988). Religious concerns and certain psychological difficulties, such as depression or obsessive–compulsive disorder, may also be associated with inhibited desire.

Treatment

Treatment for inhibited sexual desire may be difficult because the person with low desire may be seeking treatment at the urging of their partner, and thus they may not be internally motivated for therapy. Provided physiological causes have been ruled out (e.g., low testosterone levels) it is essential that treatment begin by structuring the impaired desire as a couple problem, rather than as an individual problem (Pridal and LoPiccolo, 2000). Cognitive therapy is often used to restructure thoughts and beliefs about sexuality that may be inhibiting desire (e.g., good women should not desire sex) to reflect ideas more conducive to sexual enjoyment, and to address negative underlying relationship issues. Behavioural interventions are often used to treat physical expressions of affection and intimacy and to increase sexual communication. In some cases, couples have ceased not only sexual activity, but all physical affection — the partner with low desire may have stopped showing any affection for fear that it be interpreted as interest in sexual activity. In such cases, the couple is encouraged to begin expressing affection through non-sexual means such as holding hands, cuddling, hugging, and brief kissing, and then to gradually reintegrate sexual intercourse into their relationship.

Sexual Aversion Disorder

In the DSM-IV, sexual aversion disorder is defined as the recurrent or persistent extreme avoidance of or aversion to all, or nearly all, genital sexual contact with a sexual partner. The association between sexual aversion disorder and anxiety has led some to argue that sexual aversion disorder may be best conceptualized as a type of anxiety disorder, like a snake phobia or a fear of heights. Sexual aversions in some people may be related to a history of sexual trauma or unwanted sexual activity. Men may also develop sexual aversion as a result of fear of erectile failure and/or a desire to avoid unpleasant sensations associated with anxiety (Gold and Gold, 1993).

Sexual aversion disorders may be treated by addressing the underlying anxiety. Anxiolytic medications may be used to reduce the anxiety, sometimes in conjunction with sex therapy. If the sexual aversion appears to result from a history of sexual abuse, counselling directed at coming to terms with this history may be most effective. For men, treatment may involve sex education regarding realistic expectations about performance and female response (Gold and Gold, 1993).

SEXUAL AROUSAL DISORDERS

Closely connected with sexual desire, sexual arousal is defined in both psychological (e.g., 'feeling sexually excited') and physiological terms (e.g., genital blood flow). Physiological sexual arousal in males involves signals from central (brain and spinal cord) and peripheral nervous systems, and on a complex interplay between

neurotransmitters, vasoactive agents and endocrine factors. Within the penis is a central artery and veins that exit and drain the erectile bodies. The muscles that line the sinusoidal spaces and the central artery are contracted during the non-aroused state. Erection begins with muscle relaxation that is controlled by autonomic nerves and by the release of nitric oxide (described below). Smooth muscle relaxation reduces vascular resistance and the erectile bodies fill with blood. Once the erectile bodies become engorged with blood, the veins are compressed under the penis's tough fibroelastic covering and blood is trapped in the penis. Normally, detumescence (i.e., loss of erection) occurs with the release of catecholamines during orgasm and ejaculation.

Physiological sexual arousal in women begins with vasocongestion of the vagina, vulva, clitoris, uterus, and possibly the urethra, and can occur within only a few seconds of sexual stimulation. Vaginal lubrication occurs when the blood vessels of the vaginal epithelium (vaginal wall) become engorged with blood, causing fluid to pass between the cells of the vaginal epithelium and emerge on the vaginal wall as sweat-like droplets. These droplets can quickly build up to form a lubricating film that facilitates penetration of the penis. Nitric oxide has also been implicated in female sexual arousal (see below).

Female Sexual Arousal Disorder

According to the DSM-IV, a woman may have female sexual arousal disorder if she experiences repeated and persistent difficulty attaining or maintaining, until sexual activity is completed, a sufficient lubrication-swelling response of sexual excitement. As with many sexual disorders, female sexual arousal disorder can be subdivided into lifelong versus acquired types, generalized versus situational types, and due to psychological versus organic, or combined factors (American Psychiatric Association, 1994). Women of all ages may experience difficulty lubricating, although it tends to be more of a problem in later life, typically after menopause. Approximately 20% of women aged 18–49 years old reported problems lubricating compared to 27% of women aged 50–59 years old. Difficulty lubricating is not associated with marital status or level of education (Laumann *et al.*, 1999).

Physiological Factors

Oestrogen levels can have a profound effect on sexual arousal. This is most apparent through the menopause transition. Oestrogen levels decline during menopause, which results in atrophy of the vaginal epithelium (vaginal walls), and a decline in blood flow into the capillaries of the vaginal wall. A loss of oestrogen following menopause can also indirectly impair sexual arousal by impairing mood. Oestrogen replacement therapy can help remedy some of the vasomotor symptoms and vaginal atrophy in postmenopausal women (Sherwin, 1991). Several researchers have examined whether hormonal fluctuations across the menstrual cycle affect sexual arousal but failed to find any consistent relationship (Meuwissen and Over, 1992).

As noted earlier, some studies have shown that activation of the sympathetic nervous system can facilitate female sexual arousal. When sexually functional women and women with low sexual desire engaged in vigorous exercise (stationary cycling, which stimulates the sympathetic nervous system), they demonstrated significantly higher levels of physiological sexual arousal to an erotic film than without exercise (Meston and Gorzalka, 1995a, 1996). Physiological sexual arousal was measured using a vaginal photoplethysmograph, a tampon-like device that the woman inserts into her vagina, which measures blood flow in the vaginal capillaries. It is important to note that in these studies exercise alone did not facilitate sexual arousal—it was only when the women viewed an erotic film that vaginal blood flow was increased. This suggests that exercise somehow prepares the women's body for sexual arousal so that when she enters a situation she views as sexually appealing, her level of physiological sexual responding is intensified. Meston and Gorzalka (1995b) found a facilitatory effect of exercise at 15 and 30 minutes following exercise. Whether the effect remains past 30 minutes has not been examined. When the sympathetic nervous system is activated using a medication to increase sympathetic nervous system activity, such as ephedrine, physiological sexual arousal to an erotic film is also facilitated (Meston and Heiman, 1998). The reverse has also been shown; medications such as clonidine that block sympathetic nervous system activity impair sexual arousal (Meston *et al.*, 1997).

Animal studies and studies examining the physiology and side effects of medications suggest that neurotransmitters in the central nervous system and neuropeptides in the periphery of the body impact sexual arousal. Nitric oxide activity in the penile tissue triggers a cascade of events leading to increased blood flow into the penile capillaries. The medication Viagra, which is an orally administered treatment for erectile dysfunction, acts by initiating these events (the mechanism of action of Viagra will be described in more detail below under the erectile dysfunction section). Recent studies show that nitric oxide is also produced in clitoral tissue.

Convergent evidence suggests that serotonin activity in the periphery of the body may be involved in female sexual function and dysfunction. Serotonin is a powerful vasoactive substance that, depending on the site and tissue type, may produce vasodilation or vasoconstriction. Normal vaginal lubrication is dependent upon adequate vasocongestion of the genital tissue and it is feasible that serotonin may be involved in this process. If so, abnormalities in serotonin mechanisms may impair vaginal vasocongestion (e.g., with SSRI use). Serotonin is also active in several other peripheral mechanisms that are likely to affect sexual functioning such as nonvascular smooth muscle contraction, endocrine functions, and the spinal cord and peripheral nerves (Frohlich and Meston, 2000).

Mild abnormalities in cutaneous sensation, the sense of touch, have been associated with difficulty becoming lubricated. Cutaneous sensation was measured using Von Frey monofilaments, hair-like fibres that when pressed against the skin reliably apply a specific amount of force—the amount of force applied depends upon the diameter and length of the hair. College aged women with sexual arousal disorder required a higher degree of stimulation (more force) to their skin before they perceived the stimulation as compared to women with normal sexual functioning. This suggests that women with sexual arousal disorder may have mild abnormalities in peripheral nervous system functioning (Frohlich and Meston, 1999).

Very little research has been published regarding brain areas involved in female sexual arousal although several studies have implicated the paraventricular nucleus of the hypothalamus. Transneural viral labelling indicates that the clitoris and uterus are connected to the paraventricular nucleus and the findings from one study in rats suggests that the paraventricular nucleus is active during copulation. During sexual arousal, oxytocin produced in the paraventricular nucleus is secreted into the blood stream by the posterior pituitary (McKenna, 1999). It is feasible that paraventricular nucleus activity is abnormal in women with sexual arousal disorder.

Psychological Factors

In addition to affecting sexual desire, factors such as performance demand, anxiety, and expectancies can also affect physiological sexual arousal (i.e., vaginal vasocongestion and lubrication). Laan *et al.* (1993) found that sexually functional women became more sexually aroused after being asked to become as sexually aroused

as possible, versus being told their level of sexual arousal was not important to the study. This suggests that for women without sexual difficulties, performance demand can facilitate sexual arousal. Expectancies may interact with autonomic arousal to influence sexual arousal as well. Palace (1995) found that sexually dysfunctional women who were falsely informed that they displayed strong physiological sexual arousal to an erotic film, displayed greater physiological and self-reported sexual arousal to a subsequent erotic film. Moreover, this response was greater if the erotic stimulus was preceded by an anxiety/fear provoking film versus a neutral film. This suggests that sympathetic nervous system arousal paired with the expectation of sexual arousal may be effective in increasing physiological sexual arousal in women.

Because of the close link between sexual desire and sexual arousal in women, many of the same psychological factors that impair sexual desire also inhibit vaginal lubrication. Briefly, these include individual, relationship, and cultural factors. An estimated 49% of women who were sexually abused as children report impaired sexual arousal. Inadequate or inappropriate sexual stimulation may interfere with sexual arousal, as would negative emotions such as fear of rejection, anger, or relationship conflict (Morokoff, 1993).

Male Erectile Disorder

According to the DSM-IV, male erectile disorder is characterized by a persistent or recurrent inability to attain or maintain an adequate erection until completion of the sexual act. Erectile dysfunction may result when a medical problem or condition, such as diabetes mellitus, surgical injury, aging, or pharmaceutical intervention, affects vascular blood flow to the penis or neural innervations to and from the penis. Patients are diagnosed with erectile dysfunction when their erectile difficulties are exclusively psychogenic in nature, or are caused by a combination of psychological and medical factors (American Psychiatric Association, 1994).

Men of all ages occasionally have difficulty obtaining or maintaining an erection, but true erectile disorder is more common after age 50 years. Approximately 7% of men aged 18–29 years have erectile troubles 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).

Physiological Factors

Testosterone does not affect erectile functioning unless it falls below a critical level. Hypogonadal men, or men with unusually low testosterone levels, often experience erectile problems that are successfully treated with testosterone replacement therapy. Testosterone administration does not improve erectile response in men with normal testosterone levels. Prolactin levels also affect erectile functioning although the process is complex; men with abnormally high prolactin levels and men with abnormally low prolactin levels may experience erectile dysfunction (Besser and Thorner, 1975; Deutsch and Sherman, 1979). In normally functioning men, prolactin and oxytocin levels increase significantly during sexual arousal (Meston and Frohlich, 2000).

Acetylcholine, vasoactive intestinal peptide, and nitric oxide have also been implicated in penile tumescence (i.e., erection). Activation of cholinergic receptors produces relaxation of the penile smooth muscles, allowing blood flow into the penis, thus producing an erection (Saenz de Tejada *et al.*, 1988). Sexual stimulation leads to the production of nitric oxide in penile tissue. Nitric oxide stimulates guanylate cyclase release, which triggers the conversion of guanosine triphosphate to cGMP. cGMP activity relaxes the smooth muscles of the penile tissue allowing vasocongestion and erection (Burnett, 1995). The pharmaceutical company, Pfizer, capitalized on this process by developing the medication Viagra, which is designed to treat erectile dysfunction. Viagra potentiates the activity of cGMP by inhibiting phosphodiesterase type 5, the endogenous substance responsible for cGMP deactivation. This increases and prolongs cGMP activity, which increases and prolongs vasocongestion, and enables erection. Interestingly, Viagra was discovered by accident when researchers for Pfizer noticed that men taking an experimental drug for heart disease, which worked on nitric oxide systems, had erections as a side effect.

A variety of psychoactive medications produce erectile dysfunction. Antiparkinsonian medications are dopamine agonists and are reported to facilitate erection (Bowers *et al.*, 1971) while antipsychotic medications are dopamine antagonists and facilitate erection at low doses and impair erection at high doses (Aizenberg *et al.*, 1995; Marder and Meibach, 1994). Cocaine is a dopamine agonist and high doses can disrupt erectile capacity (Miller and Gold, 1988), perhaps due to its vasoconstrictive properties. Opioid abuse (e.g., heroin) can lead to erectile dysfunction.

Erection is dependent upon spinal reflexes and is controlled by descending inhibitory and excitatory input from the brainstem. This is most apparent when studying the effects of spinal cord injury. Transection of the spinal cord often facilitates erectile response (depending upon the region of the spinal cord injured)—animal and human studies suggest that following spinal cord injury, less stimulation is required to obtain erection and erection occurs more frequently (McKenna, 1999).

Several brain regions have been implicated in male erection. Animal studies indicate that oxytocin released from the paraventricular nucleus of the hypothalamus can produce erection as can electrical stimulation of the paraventricular nucleus of the hypothalamus and hippocampus. Electroencephalographic studies indicate that the right temporal lobe is activated when right-handed men are presented with visual sexual stimulation. Perhaps the most definitive study to date used positron emission tomography (PET scan) to examine brain activity in healthy men presented with visual sexual stimulation. The areas of the brain activated included visual, sensory, and neuroendocrine and autonomic areas. Specifically, visually presented information produced bilateral activation of the inferior temporal cortex, a visual association area, activation of the right inferior frontal cortex and right insula, paralimbic areas that relate motivational states with highly processed sensory information, and activation of the left anterior cingulated cortex, a paralimbic area that controls neuroendocrine and autonomic functions (McKenna, 1999).

Psychological Factors

It is common for men to have occasional episodes of erectile failure without it developing into a full blown erectile disorder. Barlow (1986) argued that men with erectile dysfunction respond differently in sexual situations compared to normally functioning males. When placed in a sexual situation, men with erectile dysfunction focus on non-sexual cues such as fears about inadequate performance, and worries about inability to control performance. These thoughts lead to increased anxiety, increased focus on non-erotic cues and fears of erectile failure. This process inhibits erection and thereby confirms the men's fears. Since the men's fears were confirmed, they are likely to repeat the process in subsequent sexual situations and a negative feedback loop develops. In contrast, when placed in a sexual situation, men with normal erectile responding focus on erotic cues and subsequently become aroused and are able to obtain and sustain an erection. They experience the sexual situation as pleasurable and look forward to future sexual situations creating a positive feedback loop.

Treatment

A variety of tools can be used to assess whether the erectile dysfunction is of psychological or physiological origin. If the male exhibits nocturnal erections, or obtains an erection when a vasoactive substance is injected into the corpora, the erectile problem is likely to be psychological in nature. Treatments for erectile dysfunction include vacuum devices and constriction rings, intracavernosal injections, intraurethral pharmacotherapy, topical pharmacotherapy, oral pharmacotherapy, and penile implants. The vacuum device consists 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 venous outflow so that the erection is maintained until completion of the sexual act. Several medications are available that can be injected into the corpus cavernosum of the penis to induce erection including papaverine, phentolamine, and prostaglandin E_1. These all act to dilate penile capillaries, allowing blood to flow into the penis. Although intracavernosal injections are fairly effective (between 70–90%), between 50% and 80% of patients discontinue treatment, citing problems such as inconvenience, cost, and invasiveness of treatment. An intraurethral-administered medication, MUSE (Medicated Urethral System for Erection — active ingredient prostaglandin E_1), has recently been introduced. This medication is administered in suppository form and is absorbed through the urethral mucosa. It is most effective when used in conjunction with a constriction ring. Side effects of the medication include urogenital pain and urethral bleeding. Topical medications, such as Minoxidil, are also available, although these types of treatments are not commonly used, in part because their effects on vaginal mucosa are not well understood. Viagra, an orally administered treatment, was introduced to the market in 1998, and since then, many of these rather cumbersome and involved treatments have become less popular. Viagra is well tolerated by a variety of patients and is an effective treatment for both organic and psychogenic impotence (Montorsi et al., 1999). Because sildenafil produces vasodilation and a minor drop in blood pressure, it may be contraindicated for patients diagnosed with or receiving treatment for cardiovascular disease. Data are not presently available regarding sildenafil-use in patients with certain cardiac conditions such as unstable angina, stroke, or recent myocardial infarction (heart attack) and/or arrhythmias. Nonetheless, sexual activity increases the likelihood of ischaemia or infarction and thus patients with cardiac risk factors are often referred for an exercise stress test prior to receiving sildenafil. Sildenafil can safely be administered in conjunction with some cardiovascular medications, such as most antihypertensives, but if it is taken in conjunction with organic nitrates, it can produce a major and life-threatening drop in blood pressure (Klonger, 2000).

Penile implants are considered a last resort and are used when tissue damage or deterioration is severe, and when other treatments have failed. Penile implants typically consist of a cylinder (implanted in the penis) that can be mechanically inflated and deflated (Rowland and Burnett, 2000).

Studies are currently underway to determine whether Viagra and other drugs that act as vasodilators on genital tissue will be effective for treating Female Sexual Arousal Disorder. The first Federal Drug Administration (FDA) approved treatment for female sexual arousal disorder was recently introduced to the market. The treatment is a hand-held battery-operated device, called the EROS-CTD, which contains a soft plastic cup and a suction device. When the cup is placed over the clitoral tissue and the device is activated it draws blood into the genital tissue.

ORGASM DISORDERS

The normal ejaculatory response typically occurs following sensory stimulation to the penis. The stimulation initiates a nerve signal that travels along the pudendal nerve (i.e., genital sensory nerve) and synapses at the sacral level of the spinal cord. The spinal cord input stimulates an autonomic and a somatic response. The autonomic response involves adrenergic neurons (within the sympathetic nervous system), which stimulate contraction of the smooth muscles of the vas deferens, seminal vesicles, and prostate. This leads to closure of the bladder neck and movement of seminal fluid into the urethral duct. In both men and women, orgasm is characterized by a peak in sexual pleasure that is accompanied by rhythmic contractions of the genital and reproductive organs, cardiovascular and respiratory changes, and a release of sexual tension. In men, during the emission stage of orgasm, seminal fluid is propelled into the bulbar urethra via the release of norepinephrine that acts on alpha-adrenergic receptors, the smooth muscles of the vas deferens, prostate, and seminal vessels. During the ejaculatory phase, which is mediated by a sacral spinal reflex, semen is released through the urethra via contractions of muscles that surround the bulbar urethra. The extent to which central neurophysiologic events are related to the intensity or experience of orgasm is not known. While orgasm is generally the result of both genital and psychological stimulation, evidence suggests central stimulation alone may trigger orgasm.

Female Orgasmic Disorder

Female orgasmic disorder 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 diagnostic criteria, the woman's orgasmic capacity is less than would be reasonable for her age, sexual experience, and adequacy of sexual stimulation she receives (American Psychiatric Association, 1994). Between 22–28% of women ages 18 to 59 years report that they are unable to attain orgasm. Married women and women who have some college education are significantly less likely to report being unable to attain orgasm as compared to never married and divorced women, and women who have not attended college (Laumann et al., 1999).

Physiological Factors

Among normally orgasmic women oxytocin levels were positively correlated with subjective intensity of orgasm, and prolactin levels were elevated for up to 60 minutes following orgasm (Meston and Frohlich, 2000). It is feasible that oxytocin and prolactin regulation is abnormal in women with orgasmic disorders, although a study examining oxytocin and prolactin levels in orgasmic disordered women has not yet been published.

The SSRIs frequently affect orgasmic functioning, leading to delayed orgasm or anorgasmia. The antidepressant, nefazodone, produces fewer sexual side effects in women (Feiger et al., 1996), possibly because it increases serotonin activity in general while simultaneously inhibiting serotonin activity at the serotonin$_2$ receptor — the receptor implicated in SSRI-induced sexual dysfunction (Eison et al., 1990). Cyproheptadine is also a serotonin$_2$ receptor antagonist and has been an effective antidote to SSRI-induced orgasmic dysfunction (although it is not an ideal antidote as it can disrupt the effectiveness of the antidepressant medication). Drugs that affect dopamine mechanisms, such as antipsychotics (which inhibit dopamine) or cocaine (which facilitate dopamine), delay or inhibit orgasm in women. Female heroin addicts also report delayed or inhibited orgasm. Taken together, these findings suggest that serotonin, dopamine, and opioid mechanisms may be functioning abnormally in orgasmic disordered 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., 1999; Wiedeking et al., 1979). This process

may be impaired in anorgasmic women. One study showed that when vigorous exercise (which stimulates the sympathetic nervous system) is followed by an erotic stimulus, sexual arousal is facilitated in normally functioning women, but inhibited in anorgasmic women (Meston and Gorzalka, 1996).

Brain and spinal cord mechanisms are integral to the orgasm response. The neuronal pathway connecting the genitals to the brain was identified via transneuronal labelling. Virus injected into the clitoris revealed that sexual afferent neurons synapse on neurons in the lumbosacral spinal cord and connect to the nucleus paragigantocellularis in the brainstem. Lesions to the nucleus paragigantocellularis and spinal cord transection can suppress tonic inhibition of the orgasm-like response, suggesting that this pathway exerts inhibitory control over orgasm. Neurons in this region stain positively for serotonin suggesting that it may be implicated in SSRI-induced anorgasmia in women. Studies in humans suggest that the paraventricular nucleus of the hypothalamus is also involved in the orgasmic response. The paraventricular nucleus produces oxytocin that is released from the posterior pituitary during arousal and orgasm (McKenna, 1999).

The neurological disease, multiple sclerosis, often results in orgasmic disorders. In multiple sclerosis patients, orgasmic problems are related to neurological problems such as changes in genital sensation, muscle weakness in the pelvis (Lundberg and Hulter, 1996), and brain abnormalities (in the pyramidal and brain-stem regions) (Barak *et al.*, 1996). It is feasible that similar anatomical structures are involved in orgasmic disorders, albeit non-disease related.

Psychological Factors

The degree to which a woman is distracted during sexual situations may affect orgasm ability. Orgasmic women tend to focus on their own arousal and their partners' arousal throughout the sexual situation, while anorgasmic women tend to focus on trying to attain orgasm, focus on non-sexual thoughts, and are more easily distracted by non-sexual thoughts. Anorgasmic women are also more likely to report discomfort with sex compared to orgasmic women; they are more likely to be uncomfortable discussing direct clitoral stimulation, and are more likely to have guilt about sex, to endorse sex myths, and to report negative attitudes about masturbation. Anorgasmic women are less likely to be aware of their physiological sexual arousal as compared to consistently orgasmic women (Stock, 1993).

Treatment

Treatment for anorgasmia involves education about female anatomy and physiology, self-exploration, and directed masturbation. The woman is encouraged to explore her body and identify regions and types of stimulation that produce sexual arousal, to experiment with different fantasies, and to use various tools (such as a vibrator) that may enhance arousal. Once she has successfully learned to attain orgasm during masturbation, she is encouraged to teach her partner which parts of her body and which types of stimulation are likely to bring her to orgasm. Directed masturbation is a highly effective treatment for anorgasmia, with outcome studies showing up to a 90% success rate (Heiman and Meston, 1998).

Male Orgasmic Disorder

DSM-IV criteria for male orgasmic disorder include recurrent or persistent difficulty obtaining orgasm, or inability to obtain orgasm, even after sufficient sexual stimulation. The clinician takes a variety of factors into account before making the diagnosis, such as age and amount of stimulation. In most cases of inhibited male orgasm the patient is able to attain orgasm but only through manual or oral stimulation, and when orgasm occurs through intercourse it is only possible after prolonged manual or oral stimulation (American Psychiatric Association, 1994).

For the patient and his partner (male or female), sexual activity is experienced as 'hard work' (Dekker, 1993). Very few studies of male orgasmic disorder have been published, likely due to the fact that it is a rare disorder. The prevalence in the general population is estimated at 1.5 in 1000, and among those presenting for sex therapy, 0–13 in 100 (Dekker, 1993).

What is presently known about the mechanisms underlying orgasm is drawn from animal studies and from studies examining side effects of recreational and pharmaceutical drugs. One of the more common side effects of SSRI medications is delayed or inhibited orgasm, suggesting that serotonin activity may play a role in normal orgasm functioning. Animal studies indicate that serotonin$_{1A}$ receptor activation facilitates orgasm (Bitran and Hull, 1987) — it is feasible that men with inhibited orgasm have abnormal serotonin$_{1A}$ receptor activity. Dopamine may also be implicated in orgasm functioning. Dopamine agonists, such as apomorphine and cocaine, have been reported to inhibit orgasm, although apomorphine has also been reported to facilitate orgasm. The fact that dopamine can both inhibit and facilitate orgasm may be a function of dose; in rats, a low dose of a dopamine agonist increased ejaculation latency while a high dose decreased ejaculation latency (Clark *et al.*, 1983). In normally functioning men, blood plasma levels of norepinephrine significantly increased at orgasm (Kruger *et al.*, 1998) and oxytocin levels were significantly positively correlated with subjective orgasm intensity (Carmichael *et al.*, 1994). Some evidence suggests that inhibited orgasm may result from spinal reflex abnormalities. Studies examining men with spinal cord injuries and normally functioning men suggest that the dorsal penile nerve and the perineal nerve are involved in the ejaculatory response. Stimulation to these nerves in normally functioning men resulted in contraction of the bulbocavernosus muscle, the muscle involved in ejaculation (Yang and Bradley, 1999).

The brain and spinal cord are regions implicated in orgasm. The neuronal pathway from the genitals to the brain was transneuronally labelled by injecting a virus into the penile tissue. Sexual afferents enter the lumbosacral region of the spinal cord and via interneurons and neurons connect to the nucleus paragigantocellularis in the brainstem. Lesions to the nucleus paragigantocellularis impair normal tonic inhibition of the orgasmic response. Neurons in this region stain positively for serotonin suggesting this region may be implicated in SSRI-induced anorgasmia in men. During arousal and orgasm in men, oxytocin produced in paraventricular nucleus of the hypothalamus is released from the posterior pituitary (McKenna, 1999). Single photon emission computed tomography was used to examine brain physiology during orgasm in eight healthy right-handed men. During orgasm, blood flow significantly increased in the right prefrontal cortex and decreased in all other cortical regions (Tiihonen *et al.*, 1994). Although these processes have been implicated in normal orgasmic functioning in men, no evidence is currently available on men with orgasmic disorders. Nonetheless, it is feasible that these regions and mechanisms function abnormally in men with orgasmic disorders.

Premature Ejaculation

When seminal fluid enters the urethral duct, it triggers a somatic response — known as ejaculatory inevitability. The bulbocavernosus and ischiocavernosus muscles contract and semen is ejaculated through the urethral opening. This event is typically associated with the subjective pleasure of orgasm (Rowland and Burnett, 2000). When ejaculation occurs with minimal sexual stimulation before, on, or shortly after penetration, it is referred to as premature ejaculation. The clinician determines whether a diagnosis should be

made after taking a variety of factors likely to affect ejaculation latency into account, such as age and novelty of partner. A diagnosis is only made if it is a recurrent or persistent problem (American Psychiatric Association, 1994).

Premature ejaculation is a fairly common problem. Approximately 30% of men ages 18 to 59 report that they orgasm too early. Marital status and ethnicity are not significantly associated with premature ejaculation, but men who have attended college or graduated from college have lower rates of premature ejaculation than men with less education (Laumann *et al.*, 1999).

Physiological Factors

As described above, several medications inhibit or delay orgasm suggesting that neurotransmitter activity affected by these medications may be involved in normal ejaculation and, possibly, premature ejaculation. Dopamine agonists such as apomorphine and cocaine have been reported to affect orgasm and SSRIs often delay ejaculation suggesting that abnormalities in serotonin and/or dopamine regulation may underlie premature ejaculation. Animal studies suggest that stimulation of the serotonin$_{1A}$ receptor decreases ejaculation latency (Ahlenius and Larsson, 1997). Usually ejaculations occur after several intromissions but in some animals the effects of serotonin$_{1A}$ stimulation are so pronounced that ejaculation occurs at the first intromission (Ahlenius *et al.*, 1981). It is feasible that the serotonin$_{1A}$ receptor is hypersensitive in men with premature ejaculation.

Kaplan (1974) proposed that men with premature ejaculation are more sensitive to erotic stimuli and thus become aroused and orgasm more quickly. They may also be less adept at perceiving the sensations leading to ejaculatory inevitability (i.e., the point when semen is in the base of the urethra and ejaculation cannot be stopped). Several studies have tested this hypothesis with mixed results. Spiess *et al.* (1984) found that men with and without premature ejaculation did not differ in how quickly they became aroused, the length of time it took for them to obtain their maximum erection, or their erectile response to an erotic film. Colpi *et al.* (1986) found that men with premature ejaculation had a more sensitive ejaculation reflex and Fanciullacci *et al.* (1988) found that men with premature ejaculation had larger areas of the somatosensory cortex devoted to the genital region compared to normal controls. Taken together, these studies suggest that some cases of premature ejaculation may be organic in nature.

Psychological Factors

Traditionally, anxiety has been implicated in the aetiology of premature ejaculation yet empirical studies have found conflicting evidence regarding its role. Strassberg *et al.* (1990) compared self-reported thoughts during sexual stimulation in men with and without premature ejaculation and found no differences in self-reported anxiety. Cooper and Magnus (1984) conducted a double-blind, placebo controlled, crossover study where men with premature ejaculation were randomly assigned to receive an anxiolytic medication versus placebo. Although the anxiolytic medication had the expected impact on anxiety (it reduced it), it did not affect ejaculation latency. Cooper *et al.* (1993) compared men with primary premature ejaculation (i.e., life-long premature ejaculation problem) to men with secondary premature ejaculation (i.e., developed the problem after a period of normal ejaculation latencies) and found that men with secondary premature ejaculation were significantly more likely to report anxiety during intercourse and scored significantly higher on a general measure of anxiety. This suggests that anxiety may play a role in secondary premature ejaculation but not primary premature ejaculation.

Treatment

Treatments for premature ejaculation include psychological as well as pharmacological interventions. The most common psychological treatment, the pause-and-squeeze technique, was introduced by Semens (1956) and popularized by Masters and Johnson (1970). This technique is fairly straightforward; the man is stimulated to a point close to orgasm, the stimulation is interrupted (pause) and firm pressure is placed under the glans of the penis (squeeze). The procedure is repeated several types (typically twice) before ejaculation is permitted. These behavioural strategies are often combined with other strategies aimed at increasing control over ejaculation such as increasing the range of sexual activities (i.e., other than intercourse) and increasing the awareness of physical sensations associated with approaching ejaculation (so that stimulation can be ceased prior to ejaculatory inevitability). Patients may also be encouraged to use sexual imagery and thoughts to slightly decrease arousal levels and thus help control ejaculation (e.g., mentally listing the players in a favourite sports team).

More recently, pharmaceutical agents that have the side-effect of delaying ejaculation have been used to treat premature ejaculation. These include antidepressants such as the SSRIs and tricyclic antidepressants, and anti-anxiety medications such as the benzodiazepines. These types of medications are often effective in delaying ejaculation, although the effectiveness can wear off after several weeks and some men do not experience any delay in ejaculation (often the men least likely to respond are also those who ejaculate the most quickly). In some people these medications can have unpleasant side effects such as gastrointestinal disturbance and headache. Topical creams that dull sensation, such as lidocaine, may also effectively delay ejaculation, although they are not appropriate for men who ejaculate prior to insertion, and the creams can be irritating to vaginal tissue (Metz and Pryor, 2000).

PAIN DISORDERS

Dyspareunia

The DSM-IV defines dyspareunia as recurrent or persistent genital pain associated with sexual intercourse. The diagnosis of dyspareunia is not given if the pain decreases or is eliminated by adequate vaginal lubrication.

Although dyspareunia is currently classified as a psychiatric disorder, experts contend that it may be better classified as a pain syndrome that results in sexual dysfunction rather than a sexual dysfunction (that involves pain). In a recent review, Binik *et al.* (2000) suggested that describing genital pain along several dimensions including location, quality, elicitors, course, intensity, and meaning could be useful in identifying the cause of the pain and directing the type and course of treatment. Some women report that the pain is localized, generalized, or wandering while some women are not able to identify the location of the pain. The pain may have a 'sharp', 'burning', 'dull', or 'shooting' quality that may reflect the type of pathology. The pain may be specific to intercourse, or may follow other types of stimulation (e.g., oral sex). It may begin before, during, or after stimulation, and may be mild, moderate, severe, or excruciating. Women may attribute meaning to the pain—they may believe it is related to a medical condition or a psychological source. Meana *et al.* (1999) found that women who attributed their pain to a psychological source rated the pain as more severe in intensity.

Dyspareunia may be caused by anatomical, pathological, iatrogenic, or psychological factors. A rigid hymen would be an anatomical factor that could result in genital pain during intercourse. Infections in the genitals could produce genital pain during intercourse, as could endometriosis and non-malignant and malignant

tumours. Surgical procedures (e.g., episiotomy) could also result in dyspareunia. Following menopause, atrophy of the vulva and vaginal tissue can increase the likelihood of dyspareunia. No one disease is associated with dyspareunia and a disease or disorder can be quite extensive without causing sexual pain. A variety of psychological factors may also lead to dyspareunia. For example, it may develop as a result of attitudes and values passed down from parents that lead to fear and anxiety in sexual situations, traumatic events where sexual or non-sexual contact with the genitals was experienced as painful, or emotional or relational factors, such as depression or discord between partners (Meana and Binik, 1994).

Recent evidence suggests that some forms of dyspareunia may be associated with abnormalities in pain sensation. The sense of touch and pain was measured in women with vulvar vestibulitis and control women (vulvar vestibulitis is a condition characterized by severe pain upon attempted intercourse or vestibular touch — the vestibule refers to the area of tissue below the clitoris, between the labia minora, and the vaginal opening). Touch and pain thresholds were obtained by applying small amounts of force to the skin; touch threshold was defined as the minimum amount of force needed for the women to consciously detect the stimulation, and pain thresholds were defined as the minimum amount of force that was experienced as painful. The women with vulvar vestibulitis were more sensitive to light touch and pain than the control women suggesting greater tactile and pain acuity (Pukall et al., 2000). Women with vulvar vestibulitis also had more densely packed sensory nerves in the vestibule, which may account for their increased sensitivity (Westrom and Willen, 1998).

Treatment

Regardless of the cause of dyspareunia, the symptoms are most effectively treated with cognitive-behavioural therapy. Even when the pain is a direct result of a medical condition, the pain often continues after medical intervention (Schover et al., 1982). Psychological treatment typically involves one or more of the following techniques: vaginal exercises, vaginal dilation, systematic desensitization, and couples therapy (education regarding communication and sexuality). The goal is for the woman and her partner to learn, through education and direct experience, that sexual contact and intercourse do not necessarily produce pain. Vaginal exercises involve the voluntary contraction of the vaginal muscles, allowing the women to gain familiarity and greater control over her muscle contractions. Vaginal dilation involves inserting increasingly larger dilators into the vagina until the woman is able to insert one that is a similar size to her partner's penis, without experiencing pain or anxiety. Vaginal dilation is one form of systematic desensitization but systematic desensitization can also be performed by fantasizing about pain producing activities. The woman is first asked to list activities in order from least painful or anxiety provoking to most painful and anxiety provoking. She is then instructed to fantasize about the least painful activity until she is able to picture it without discomfort. Once she is able to do this, she moves to the next item on the list, until she is able to fantasize about the most painful and anxiety provoking item on the list without feeling discomfort.

Vaginismus

The DSM-IV defines vaginismus as repeated and persistent involuntary spasm of the vaginal muscles that interferes with intercourse. For many women, this difficulty is not specific to intercourse; they are often unable to insert even tampons into their vaginas and fear and avoid gynaecological exams. The condition is not necessarily a generalized sexual problem; many women with vaginismus are able to enjoy sexual stimulation and orgasm that does not involve penetration of the vagina. The prevalence of vaginismus is not known. Laumann et al. (1994) interviewed a random sample of 1749 women and found that 10–15% of women reported sexual pain, either dyspareunia or vaginismus. Approximately 12–17% of women seeking sexual therapy present with symptoms of vaginismus (Spector and Carey, 1990).

Although the DSM-IV indicates that vaginismus involves spasm of the musculature of the outer third of the vagina, this description is based almost exclusively on self-report rather than physical examination. One study found no difference in vaginal muscular activity (measured via EMG) between women with vaginismus and control women (van der Velde and Everaerd, 1996). No empirical studies have explored what specifically occurs to prevent penetration. It is not clear whether muscle contraction prevents penetration or makes penetration difficult or painful, or whether penetration is not attempted due to anticipatory pain. The DSM-IV does not include pain as a characteristic of vaginismus, yet some experts in the field argue that the pain, or the anticipation of pain, may be central to the disorder (Reissing et al., 1999).

Vaginismus has traditionally been thought to result primarily from psychological factors. A review of the family histories of women with vaginismus reveals similar backgrounds. Often women with vaginismus were raised by parents with oppressive or authoritarian attitudes (Tugrul and Kabakci, 1997) and had parents who were engaged in frequent conflict (Silverstein, 1989).

Many women with vaginismus report having fathers who were domineering or threatening, alcoholic, seductive, or overprotective, and mothers who disliked sex or viewed sex as an obligation. Approximately 40% of women with vaginismus report a history of sexual trauma (Silverstein, 1989).

Medical conditions that could lead to vaginismus include: vaginal surgery, prolapse of the uterus, endometriosis, vaginal tumours, vaginal lesions, vaginal atrophy, congenital abnormalities, sexually transmitted diseases, abnormalities of the hymen, and pelvic congestion. In such cases, the condition may produce genital pain that develops over time into vaginismus. Medical conditions are associated with vaginismus in 23–32% of cases (Reissing et al., 1999).

Treatment

Vaginismus is treated with cognitive-behavioural therapy targetted at eliminating the erroneous beliefs and the vaginal spasms. Therapy involves identifying faulty beliefs (e.g., 'my vagina is too small to accommodate his penis') and educating the woman and her partner regarding normal sexual anatomy and physiology (e.g., in the aroused and non-aroused state, the vagina is capable of accommodating even a large penis). Vaginal spasms are treated with vaginal muscle exercises and progressive vaginal dilation. The woman and her partner insert dilators into her vagina, starting with very small sized dilators, progressively increasing the size until she is able to insert a dilator that is as large as an erect penis, and finally, attempting intercourse. Few well-controlled treatment outcome studies have been conducted making it difficult to evaluate the effectiveness of therapy, but estimates suggest that 60–100% of vaginismus cases are successfully treated with this type of intervention (Reissling et al., 1999).

PARAPHILIAS

According to the DSM-IV, in order to be diagnosed with a paraphilia, one must demonstrate the following features.

- "Recurrent, intense sexually arousing fantasies, sexual urges, or behaviours generally involving 1) nonhuman objects, 2) the suffering or humiliation of oneself or one's partner, or 3) children

or other non-consenting persons, that occur over a period of at least 6 months."

- The behaviour, sexual urges, or fantasies cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

The DSM-IV lists eight types of paraphilic disorders but in practice, individuals displaying one paraphilia very often also exhibit other paraphilic behaviours. Incarcerated paedophiles often report, for example, that they have also engaged in other paraphilic behaviours (e.g., exhibitionism, voyeurism) and that deviant sexual behaviours other than pedophilia are their primary interest. The presence of paraphilic behaviour may represent an underlying sexual impulsivity disorder that is characterized by sexual compulsivity and hypersexuality, and in some cases, aggression (Kafka, 1997).

Fetishism

According to the DSM-IV, fetishism involves "recurrent, intense sexually arousing fantasies, sexual urges, or behaviours involving the use of nonliving objects" as sexual stimuli (American Psychiatric Association, 1994). Most fetishists are male and nearly one in four are homosexual. Common fetish items include shoes and lingerie and common materials include rubber and leather. Fetishists become aroused by stealing the object, viewing the object, or masturbating with the object. Most fetishists are aroused by a number of different objects. The aetiology of fetishism is not known. Two reported cases of fetishism have been associated with abnormalities in the temporal lobe. In one case the patient had temporal lobe epilepsy and in the other the fetish behaviour was linked to the development of a temporal lobe tumour (Wise, 1985). Some evidence suggests that fetishism may be a learned behaviour that results when a normal sexual stimulus is paired with the fetish item. Seven heterosexual males free from any prior fetish were repeatedly shown erotic stimuli paired with a slide of a black knee-length women's boot. When the slide of the boot was later shown alone, five of the seven men demonstrated penile erection, indicating that a boot fetish had been conditioned. The conditioned fetish was shown to generalize to other types of shoes in three of the men. That is, the men also became aroused when shown a slide of a high-heeled black boot and a low-heeled black shoe. They did not become aroused to a slide of a short brown boot, a brown string sandal, or a golden sandal, suggesting that the fetish only generalized to similar types of shoes (Rachman and Hodgson, 1968). A similar study was conducted in women to determine whether women could also be conditioned to become sexually aroused to a stimulus. Subjects were randomly assigned to repeatedly view an erotic film paired with a light stimulus versus an erotic film alone. No significant differences were found in physiological sexual arousal between the experimental and control groups when a light stimulus was later presented alone (Letourneau and O'Donohue, 1997). Meston and Rachman (1994) tried to condition sexual arousal to the sound of a male's voice. Even after repeated pairings of erotic video clips and the male's voice, later presentation of the male's voice alone did not produce sexual arousal. This suggests that sexual arousal is not readily classically conditioned in women and may explain why, like other paraphilias, fetishism occurs almost exclusively in men.

Transvestic Fetishism

Transvestic fetishism is diagnosed in heterosexual males who experience "recurrent, intense sexually arousing fantasies, sexual urges, or behaviours involving cross-dressing" (American Psychiatric Association, 1994). A distinction is drawn between transvestism (cross-dressing) and transvestic fetishism. A variety of people cross-dress but the behaviour is not considered a fetish unless the cross-dressing is associated with sexual feelings. For example,

transsexuals, or people who feel that their external sex does not match their internal gender identity, may cross-dress in order to feel more congruent with their gender identity but do not find the cross-dressing sexually arousing. Similarly, homosexual males may cross-dress (e.g., drag-queens), but the cross-dressing is not considered to be a fetish unless it is sexually arousing.

Very few studies have been published regarding transvestic fetishism and those that have often grouped transvestic fetishists with transvestites who experienced little to no sexual arousal from cross-dressing. Doctor and Prince (1997) surveyed 1032 male transvestites between 1990 and 1992. They found that 40% of respondents found cross-dressing 'often' or 'nearly always' sexually exciting but only 9% described themselves as a "fetishist [who] favoured women's clothing". While keeping in mind that it is unclear what percentage of subjects would meet DSM-IV criteria for transvestic fetishism, the following characteristics were reported. Respondents ranged in age from 20 to 80 years of age, lived throughout the United States, and reported a range of religious affiliations (24% were Catholic, 38% were Protestant, 3% were Jewish, 10% were agnostic, and 25% were with other religious affiliations). The majority of respondents were well educated (65% had at least a BA), in committed relationships, and had children. Of those currently married, 83% reported that their wives were aware of their transvestic tendencies at present, but only 28% accepted the behaviour. The vast majority reported a heterosexual orientation (87%) although 29% reported having had homosexual experiences. The majority of respondents began cross-dressing before age 10 (66%) or between age 10 and 20 (29%), had been raised by both parents (76%), and reported that their father "provided a good masculine image" (76%).

A few cases have been reported of men with transvestic fetishism who had fathers or brothers who also cross-dressed. Since so few cases of familial co-occurrence have been reported in the literature, and because the occurrence of transvestic fetishism in the general population is not known, it is not clear whether family environment and/or genetics contributes to the likelihood of developing a cross-dressing fetish. Transvestic fetishism is associated with learning disabilities, and a few cases of transvestic fetishism have been associated with temporal lobe abnormalities (Zucker and Blanchard, 1997).

A number of studies have been published examining psychosocial causes of transvestic fetishism but most have serious methodological flaws that limit drawing confident conclusions. Some such studies suggest that adolescents with transvestic fetishism tendencies may have a history of separation from and hostility towards their mothers. The cross-dressing may serve as a means to make a connection with females, even if that connection often involves some expressions of anger and hostility (Zucker and Blanchard, 1997).

Pedophilia

Pedophilia is defined as intense and repeated sexually arousing fantasies, urges, or behaviours involving sexual activity with children, typically less than 14 years old (American Psychiatric Association, 1994). Since few paedophiles are likely to openly admit their preference, it is difficult to estimate the prevalence of pedophilia in the general population. Furthermore, individuals who feel sexual attraction to children may resist the temptation due to societal pressures, yet may nonetheless experience sexual fantasies involving children. Recent evidence suggests that pedophilia may be associated with homosexuality, mental retardation, and high maternal age. Homosexuality in the general population is estimated at 2% while homosexuality in paedophiles is estimated at up to 40%. When sexual orientation, intellectual functioning, and maternal age were measured in 991 male sex offenders, high maternal age and low intellectual functioning were significantly

associated with homosexual pedophilia. The association between low intelligence and pedophilia suggests that pedophilia may reflect a developmental disorder. The association between high maternal age and pedophilia is unclear, although it may reflect differences in birth order as homosexuality is associated with being later born (discussed below under gender identity disorder) (Blanchard *et al.*, 1999).

Some researchers have speculated that a childhood history of sexual abuse contributes to an adult preference for sexual activity with children. In a large sample of men who were child sex offenders, Freund *et al.* (1990) found that heterosexual and homosexual paedophiles were significantly more likely to report childhood sexual abuse by a male abuser (versus female abuser) as compared to controls. Freund and Kuban (1994) classified child sex offenders according to whether they demonstrated phallometric (increased penile volume) preference to photographs of nude children versus adults. They found that child sex offenders who demonstrated preference for children were significantly more likely to have a childhood history of sexual abuse. It should be noted that although reports indicate approximately 49% of paedophiles have a history of childhood sexual abuse, very few people with a history of childhood sexual abuse become paedophiles (Freund and Kuban, 1994).

Paedophiles may have difficulty with gender differentiation. Freund *et al.* (1991) showed slides of nude male and female children and adults to paedophiles and controls, and measured penile volume changes. The paedophiles demonstrated less differentiation between stimuli containing males versus females as compared to non-paedophiles. Although this pattern of undifferentiated arousal has also been noted in a case study of a 20-year-old woman with multiple paraphilias (Cooper *et al.*, 1990), few cases of female pedophilia have been reported in the literature.

Paedophiles may differ from non-paedophiles on several physiological dimensions as well. Baseline plasma cortisol, prolactin, and body temperature were significantly higher in paedophiles than controls. When both groups were administered a serotonin agonist, mCPP, versus placebo, plasma cortisol levels were more elevated and remained elevated longer for paedophiles compared to controls. The paedophiles reported experiencing side effects (e.g., dizzy, restless) of mCPP administration while the controls did not. Consistent with these findings, some researchers have speculated that pedophilia may be associated with disturbances in serotonin-related aggression and impulsivity (Maes *et al.*, 2001). It has also been suggested that pedophilia may be a subtype of obsessive–compulsive disorder; a problem that is marked by repetitive, irrepressible behaviour associated with serotonin dysregulation (Balyk, 1997).

Sexual Masochism and Sexual Sadism
The DSM-IV defines sexual masochism as "recurrent, intense sexually arousing fantasies, sexual urges, or behaviours involving the act (real, not simulated) of being humiliated, beaten, bound, or otherwise made to suffer" (American Psychiatric Association, 1994). In 1886, Krafft-Ebing coined the term, masochist, after Leopold von Sacher-Masoch, who wrote novels depicting men being humiliated and bound by females. Sexual sadism is characterized by "recurrent, intense sexually arousing fantasies, sexual urges, or behaviours involving acts (real, not simulated) in which the psychological or physical suffering (including humiliation) of the victim is sexually exciting to the person" (American Psychiatric Association, 1994). The term, sadism, was derived from writings of the Marquis de Sade, an 18th century author who wrote stories depicting sexual torture and brutality. A distinction is drawn between minor versus major sexually sadistic acts. Minor sexually sadistic acts would include, for example, humiliation and bondage of a willing sexual masochist while major sexually sadistic acts would involve acts such as sexual torture and rape of an unwilling participant. The key distinction here is whether the victim was consenting or not.

The practice of sadomasochism (referred to as S&M), or the consensual participation between sexual sadist and sexual masochist, involves carrying out predetermined sexual scenarios. These scenarios commonly involve several themes: flagellation (usually on the buttocks), bondage, 'water sports' (urophilia — attraction to urine, coprophilia — attraction to feces, and mysophilia — attraction to filth), and penis and nipple torture (Arndt, 1991). Sadomasochists interviewed in New York and San Francisco between 1976 and 1983 reported S&M activities that included elements of dominance and submission, role-playing (e.g., master and slave), consensuality (i.e., both participants were willing), and were of sexual context (i.e., the role-playing was sexual) (Weinberg *et al.*, 1984). Commonly reported S&M role-play themes include: "severe boss and the naughty secretary", "the queen and many slaves", "the male barber and his customer", and "arrest scenes and military training" (Sandnabba *et al.*, 1999). Although the sexual sadist appears to be in control, often the degree of domination and humiliation is agreed upon earlier, and it is the sexual masochist who indicates with a predetermined cue when he/she has reached his/her limit (Arndt, 1991).

Female sexual masochists and sadists are outnumbered by male sexual masochists and sadists and in many cases, the females are prostitutes who specialize in sadomasochism. One study found that approximately a quarter of female sexual sadists are prostitutes (Breslow *et al.*, 1985). Approximately 80% of sadomasochists reported that they were regularly engaging in sadomasochistic activities by age 30 years (Sandnabba *et al.*, 1999). Spengler (1977) obtained questionnaire data from 245 male sadomasochists recruited through S&M magazine advertisements and via S&M clubs. The majority of respondents reported that they met partners through sadomasochism advertisements, clubs, or bars. The sample contained 30% heterosexual sadomasochists, 31% bisexual sadomasochists, and 38% homosexual sadomasochists. The respondents came from all ages, socioeconomic backgrounds and levels of education. In most cases, the families knew little if anything about the respondents' S&M activities; 41% of married respondents (*n* = 109) reported that their wives knew nothing about the sadomasochistic activity.

When queried whether they thought the sadomasochistic behaviour was acceptable, 70% indicated acceptance of the behaviour, 85% reported that they "want to do it again", "it was fun" (84%), and "sexually satisfying" (79%). Although many of the respondents reported that they enjoyed non-sadomasochistic sexual activity, they reported being more likely to orgasm with sadomasochistic activity (79%) than without (45%). About a third of respondents reported fetishisms (e.g., boots and leather).

Very few studies have been conducted examining sexual sadists who target unwilling victims. Seto and Kuban (1996) examined penile volume changes in seven sadistic rapists compared to 14 non-sadistic rapists and 20 controls. The subjects were presented audiotapes depicting five different scenarios: (1) nonviolent, non-sexual interaction with a female; (2) consensual sexual activity with a female; (3) non-sexual violence against a female; (4) rape; and (5) violent rape. Compared to controls, the sadistic rapists and non-sadistic rapists were equally aroused by the different types of sexual contact — they were less likely to differentiate between consensual sexual activity, rape, and violent rape.

A subset of sexual sadists may have abnormal endocrine activity although hormone levels typically do not differ between sexual sadists and controls. In a review of individual cases, one sexual sadist had unusually high levels of luteinizing hormone (stimulates progesterone secretion) and follicle-stimulating hormone (stimulates estradiol in women and sperm development in men), another had low testosterone levels and another Klinefelter's syndrome (XXY chromosomes rather than the typical XY male pattern). Gross examination of brain functioning revealed no differences between sexual sadists and controls, but more careful examination

revealed a subtle but significant difference in the right temporal lobe. Forty-one percent of the sexual sadists had a slightly dilated right temporal horn, compared to 13% of controls. One sexual sadist had a slow growing tumour in the left frontal-temporal lobe, likely present since childhood. Another had enlargement of the ventricles, a condition typically associated with schizophrenia and suggestive of overall brain atrophy. In short, temporal lobe abnormalities may be implicated in sexual sadism, but more information is needed before any strong conclusions can be made (Langevin *et al.*, 1988).

Serial killing, which is often reported in the media and dramatized in movies, may reflect comorbid sexual sadism and antisocial personality disorder. Geberth and Turco (1997) examined records of 387 serial murderers within the United States and found that 248 had sexually assaulted their victims. These included famous cases of serial killing, such as Theodore (Ted) Bundy and the Green River Killer. Of these, they determined that 68 met DSM-IV criteria for both sexual sadism and antisocial personality disorder (in other cases, sufficient data were not available to make a determination). These 68 individuals displayed a pattern of behaviour characterized by childhood aggressiveness and antisocial behaviour, and a pattern of killing involving sexual violence, humiliation, domination and control. Examination of their records suggests that these 68 individuals engaged in sexual violence and killing because they derived pleasure from it.

Courtship Disorders: Voyeurism, Exhibitionism, and Frotteurism

Voyeurism, exhibitionism, and frotteurism may be different behavioural expressions of a single underlying courtship disorder. The overt behaviours differ, but can also be conceptualized as different stages on a continuum — different degrees of proximity to the victim. Voyeurism involves viewing the victim from a distance, exhibitionism involves approaching the victim, and frotteurism involves physically touching the victim. The preference for rape over consensual sexual activity (termed the preferential rape pattern) may represent the fourth phase in the courtship disorders (Freund *et al.*, 1983). A common aetiological factor has not been identified although evidence indicates that the courtship disorders are associated with a preference for eliciting an alarmed reaction from an unfamiliar target rather than any lack of interest in intercourse (Freund and Watson, 1990). A high degree of comorbidity exists between these disorders and even when no overt comorbid behaviour is present, some evidence suggests that presence of one disorder predisposes to another such disorder (Freund *et al.*, 1983).

Voyeurism

The DSM-IV defines voyeurism as "recurrent, intense sexually arousing fantasies, sexual urges, or behaviours involving the act of observing an unsuspecting person who is naked, in the process of disrobing or engaging in sexual activity" (American Psychiatric Association, 1994). Most men, if given the opportunity to view a woman disrobing, would not avert their eyes. A man who engages in an opportunistic 'peep' is not a voyeur, the peeping must be recurrent and the urges to do so intense. Voyeurs tend to be the youngest child in the family. Compared to other sex offenders and controls, voyeurs have fewer sisters, have a good relationship with both parents, but have parents who do not have a good marital relationship. Voyeurs are often underdeveloped socially and sexually. They tend to engage in sexual activity later than other groups, and are less likely to marry than controls and other sex offenders (Smith, 1976). The more sexually experienced a voyeur, the more frequently he is likely to engage in peeping behaviour (Langevin *et al.*, 1985). Some evidence suggests that voyeurs may be predisposed to other paraphilias as well (e.g., sadomasochism, zoophilia) (Langevin *et al.*, 1985).

Although voyeurism is rare in women, some evidence suggests that women have similar 'peeping' urges as men. Friday (1975) interviewed women from all ages (teen to retirement) and walks of life and found that women expressed fantasies about peeping and, in some cases, engaged in actual peeping behaviour.

Learning theorists have suggested that voyeurism develops when the subject is provided a voyeuristic opportunity, and then subsequently masturbates while fantasizing about the experience. Some evidence supports this hypothesis; 50% of voyeurs reported that prior to the onset of their peeping behaviour they believed that normal sexual relations were not likely to be an option for them, and so they fantasized about scenarios they believed to be more obtainable, such as peeping. In addition, 75% of voyeurs reported that the sexual scenario they envision while masturbating reflected their first peeping experience (Smith, 1976).

Exhibitionism

Exhibitionism is defined as "the exposure of one's genitals to an unsuspecting stranger" (American Psychiatric Association, 1994) and involves some form of sexual gratification. Exhibitionism occurs almost exclusively in men. Very few cases of female exhibitionists have been reported in the literature, but the characteristics of these women differed from typical male exhibitionists. Male exhibitionists tend to be timid and unassertive men who have underdeveloped social skills and who are uncomfortable with angry or hostile feelings. Some studies suggest that exhibitionists were more likely to have been raised in a sexually puritanical background. The few female exhibitionists described in the literature, and studies examining female strippers, would suggest that the majority of female exhibitionists gain no pleasure from exposing their genitals but do so either to gain money or attention (Blair and Lanyon, 1981).

Behavioural theory proposes that exhibitionism develops as a result of a learned behaviour that is subsequently reinforced. This theory has been applied successfully to the treatment of exhibitionism (i.e., a learned behaviour can be replaced with a more socially acceptable behaviour) but it is not clear whether this reflects the actually aetiology of exhibitionism. Attempts to identify a physiological cause of exhibitionism have thus far been unsuccessful.

Frotteurism

Frotteurism involves "intense sexually arousing fantasies, sexual urges, or behaviours involving touching and rubbing against a non-consenting person" (American Psychiatric Association, 1994). The majority of published articles on this disorder group frotteurism with other paraphilic disorders or report cases of men with multiple paraphilias, including frotteurism. Abel *et al.* (1987) examined 62 males diagnosed with frotteurism, as well as other paraphilic disorders, and found that, at the time of the interview, they had committed an average of 849 frottage acts. Rooth (1973) interviewed 561 nonincarcerated men with paraphilias and found that of those exhibiting frotteurism, 79% had other paraphilias, with an average of 4.8 paraphilias each.

It is unclear whether true frotteurism in women exists, perhaps in part because of the decreased likelihood that male victims would view the behaviour as unwelcome or threatening. A handful of case reports of sexual molestation of men by women have been reported in the literature. The molestation typically occurred subsequent to erectile failure or inhibited desire (Sarrel and Masters, 1982). Although these cases do not represent female frotteurism, they suggest that it is feasible that rare cases of female frotteurism may exist, but are rarely reported.

Treatment of Paraphilias

In the mid 1900s, some European countries used castration as a means of treating exhibitionism, pedophilia, and other forms of sexual crimes. In West Germany, psychosurgery, which involved removing the nucleus ventromedialis of the hypothalamus, was used as a treatment for male sex offenders. Published reports of these practices rarely provided sufficient information to determine whether this intervention was successful in eliminating the inappropriate sexual behaviour. Of course there are serious consequences to performing such extreme and permanent techniques.

Cognitive–behavioural therapies, such as aversion therapy, are often used to treat paraphilias. The arousing stimulus is paired with an aversive stimulus such as a shock or noxious odour until the paraphilic behaviour no longer produces sexual arousal. A review of the handful of studies and case reports published suggests that aversion therapy alone is effective in reducing arousal, but that relapse rates are high (Kilmann *et al.*, 1982). More recently, other forms of cognitive–behavioural therapy such as covert sensitization or orgasmic reconditioning, are being used. Orgasmic reconditioning involves fantasizing about the paraphilic behaviour while masturbating, and at the moment just before orgasm, switching the fantasy to a more acceptable stimulus, such as one's partner. The belief is that orgasm, being an intensely pleasurable sensation, will serve to reinforce the more accepted sexual fantasy. Few well-controlled treatment outcome studies have been published, however, making it difficult to determine whether these types of interventions are effective. Covert sensitization involves fantasizing about the paraphilic behaviour followed by imagining a noxious scenario, such as vomiting, or an undesirable consequence such as being discovered by one's family. It is not yet clear how successful these techniques are in eliminating the behaviour although a few reports indicate that they can be highly successful for some patients.

Pharmacological interventions include hormonal supplements or psychotropic medications. Hormonal treatments are designed to inhibit deviant sexual behaviour by reducing sexual drive and sexual arousal. They include the following: (1) oestrogen; (2) medroxyprogesterone acetate (MPA), which lowers plasma testosterone and reduces gonadotropin secretion; (3) luteinizing hormone-releasing hormone agonists (LHRH agonists), which produce the pharmacological equivalent of castration by significantly inhibiting gonadotropin secretion; and (4) antiandrogens such as cyproterone acetate (CPA), which blocks testosterone uptake and metabolism. Treatment outcome studies suggest that these treatments are effective in reducing deviant sexual behaviour provided that the treatment regimen is maintained, although more well-controlled treatment outcome studies are needed before the true effectiveness of these treatments can be determined. Psychotropic medications that affect the serotonin systems have recently been used to treat paraphilias. Clinical studies suggest that SSRIs such as Prozac are effective in reducing paraphilic arousal and may be effective in reorienting arousal to more socially acceptable scenarios. The effectiveness of SSRIs in reducing paraphilic fantasies and behaviours suggests that these disorders may have an obsessive–compulsive component, as SSRIs are often used to treat obsessive–compulsive disorders. As with hormone treatments, however, more well-controlled treatment outcome studies must be conducted before the true effectiveness of these treatments can be determined (Bradford, 2000).

GENDER IDENTITY DISORDER

The DSM-IV describes gender identity disorder as a persistent and strong cross-gender identification and a persistent unease with one's sex. Gender identity disorder is not diagnosed if these symptoms co-occur with a physical intersex condition. As with the sexual disorders, a diagnosis is only made if the symptoms produce marked distress or impairment. According to the DSM-IV, gender identity disorder can occur in childhood, adolescence, and adulthood. Sexually mature individuals may be heterosexual, homosexual, bisexual, or may feel little sexual attraction to either men or women (American Psychiatric Association, 1994). Gender identity disorder is often confused with transvestism (cross-dressing) although the two are distinct.

When biological males and females feel a cross-gender identification, it is termed male-to-female transsexualism (MF) and female-to-male transsexualism (FM), respectively. Prevalence estimates suggest that MF transsexualism is more common than FM transsexualism although a few studies have found a 1 : 1 ratio. Prevalence estimates range from 1 : 10,000 to 1 : 100,000 for MF and 1 : 30,000 to 1 : 400,000 for FM (Cohen-Kettenis and Gooren, 1999; Zucker and Green, 1992).

Studies examining the biological causes of gender identity disorder have typically examined the effects of prenatal hormones on prenatal brain development. During normal prenatal development, the presence of testosterone leads to the development of external male genitalia and to a male differentiated brain. It is hypothesized that for individuals with gender identity disorder, a discrepancy may exist between prenatal genital differentiation and brain differentiation such that the external genitals develop, for example, as male while the brain develops as female. The evidence to support this hypothesis is mixed. Genetic females exposed to high levels of testosterone *in utero* (e.g., congenital adrenal hyperplasia), rarely develop gender identity disorder. Similar prenatal exposure to antiandrogenic, androgenic, and oestrogenic drugs rarely leads to gender identity disorder in either genetic females or males although some of these individuals display abnormal gender role behaviour (Cohen-Kettenis and Gooren, 1999). The strongest evidence to suggest that abnormal prenatal brain differentiation may lead to gender identity disorder comes from a recent study examining hypothalamic brain nuclei in men with gender identity disorder. Zhou *et al.* (1995) found that the central subdivision of the bed nucleus of the stria terminalis (a region of the hypothalamus) was smaller in MF transsexuals compared to normal males but similar in size to normal females, a difference that was not accounted for by hormone therapy. Sadeghi and Fakhrai (2000) recently reported a case of 18-year-old monozygotic female twins requesting gender reassignment surgery. The twins had a childhood history of cross-dressing. Unfortunately they were lost to follow up after the initial evaluation but this case suggests that gender identity disorder may have a genetic component.

Recent studies indicate that, compared to controls, MF transsexuals have more older brothers (but not more older sisters) and a later birth order (Blanchard *et al.*, 1995; Zucker *et al.*, 1997). Conversely, FM transsexuals are more likely to have several younger sisters but not brothers compared to controls (Zucker *et al.*, 1998). The histocompatibility-Y antigen (H-Y antigen), which is responsible for the development of the male testes and brain differentiation, may be implicated in this process for males. With progressive male births, mothers may become immunized to the H-Y antigen, leading to increased production of H-Y antibodies, and a disruption in normal brain differentiation (Blanchard *et al.*, 1998).

Social, parental, or familial factors have been associated with mild gender disturbance. MF transsexuals often report over controlling, rejecting fathers. FM transsexuals often report mothers and fathers who were rejecting and mothers who were over protective. It is feasible, however, that these differences may have been the result of abnormal gender development, rather than the cause (Cohen-Kettenis and Gooren, 1999).

Childhood gender identity disorder may, in some cases, predict adult gender identity disorder. Fifty-five feminine boys with gender

identity disorder were followed into early adulthood. Five of the feminine boys were diagnosed with gender identity disorder, one as a transvestite, 21 as homosexual, 14 as heterosexual, and 14 that were not rated. This suggests that childhood gender identity disorder reflects a high likelihood of either adult gender identity disorder or homosexuality (Green, 1987).

In cases where gender identity disorder is present, if the individual displays only a mild tendency, displays serious psychopathology, or is not functioning well socially, psychotherapy rather than sex reassignment surgery may be advised. For those with extreme symptoms of gender identity disorder, who are free of from psychopathology, and who are functioning well in society, sex reassignment surgery is still not permitted until the person has lived full time as the preferred gender, often for a period of 2 years. During this period, candidates may be required to change their name, inform their family, boss, and co-workers, cross-dress full time, and receive hormone treatment. This period is considered to be essential for determining whether surgery is appropriate. The candidates have the opportunity to experience what it is like to live as the other gender and to determine whether they are fully prepared for and fully comprehend the impact of living the remainder of their lives as the other sex (Cohen-Kettenis and Gooren, 1999).

A review of sex reassignment surgery outcome studies suggests that in most cases, surgery resolves the gender identity disorder. Depending on the study, between 71% and 97% of subjects were successfully treated with surgery and less than 1% later took steps to reverse the sex reassignment. Factors that predict a poor outcome include: misdiagnosed transvestism, poor surgery outcome, poor social or work functioning, suicidal tendencies, and sex reassignment surgery late in life. This suggests that the current procedure for determining appropriateness of sex reassignment surgery is effective, when applied strictly (Cohen-Kettenis and Gooren, 1999). Male to female transsexuals who are attracted to men (MF homosexuals) seem to have a better post-surgery outcome compared to MF transsexuals who are attracted to women (MF heterosexuals). MF heterosexuals may have a poorer post-surgery outcome because of the added stigma of becoming homosexual after surgery, and because they typically present for surgery much later in life than MF homosexuals and thus are likely to have more male-role investments (e.g., husband, father). FM transsexuals in general have better post-surgery outcome than MF transsexuals (Cohen-Kettenis and Gooren, 1999).

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