**The Biology of Sex and Gender**

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After reading this chapter, you will be able to:

* Contrast sex with other motivated behaviors.
* Demonstrate the role of hormones and brain structures in sexual behavior.
* Identify hormonal and brain differences between females and males.
* Describe how behavioral differences between males and females are influenced by biology and environment.
* Explain the role of biological influences on gender identity.
* Assess the impact of biological influences on sexual orientation.

Bruce Jenner spent his early career as an athlete, playing football at Graceland College until a knee injury necessitated a change in sport to decathlon in 1968. After years of grueling training and competition, Jenner won a gold medal in decathlon at the 1976 Summer Olympics in Montreal and became a hero back home in the United States. Jenner even graced the cover of *Sports Illustrated* magazine and became the most widely known athlete to be on the cover of a Wheaties breakfast cereal box. Leaving athletics behind, he became better known as a sports commentator and occasional actor in films and television. During this time he had been married three times and fathered six children. Throughout the successes in athletics and as an actor, Jenner was struggling with a psychological disorder called gender dysphoria, which is the distress that people feel when their gender identity does not match their sex at birth. At times he dressed as a woman, and he was taking female hormones to try to better match his feelings of being female.

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In April 2015, Jenner made news by coming out as a transgendered woman. Later that year, Jenner officially adopted her now-permanent feminine identity as Caitlyn Jenner, and in January 2017, she underwent gender reassignment surgery to replace the penis with a vaginal opening. Her memoir *The Secrets of My Life* and the documentary series *I Am Cait* detailed her gender transition; in recognition of her outspoken support for LGBT rights and the strength she demonstrated in discussing her gender identity struggles with the public, she received the Arthur Ashe Courage Award and *Out* magazine’s Newsmaker of the Year award. Quite possibly the most interesting thing about Caitlyn Jenner’s new gender and identity is that she still finds herself sexually attracted to women, underscoring the fact that gender identity does not always match a person’s sexual orientation.

Source: Valerie Macon/AFP/Getty Images.

Humans have a great affinity for dichotomies, dividing their world into blacks and whites with few grays in between. No dichotomy is more significant for human existence than that of male and female: One’s sex is often the basis for deciding how the person should behave, what the person can do, and with whom the person should fall in love. Not only are many of the differences between males and females imposed on them by society, but Caitlyn’s experience suggests that typing people as male or female may not be as simple or as appropriate as we think. We will encounter even more puzzling cases later as we take a critical look at the designation of male versus female and the expectations that go with it. In the meantime, we need to continue our discussion of motivation by considering how sex is like and unlike other drives.

**Sex as a Form of Motivation**

To say that sex is a motivated behavior like hunger may be stating the obvious. But theorists have had difficulty categorizing sex with other physiological drives because it does not fit the pattern of a homeostatic tissue need. If you fail to eat or if you cannot maintain body temperature within reasonable limits, you will die. But no harm will come from forgoing sex; sex ensures the survival of the species but not of the individual.

There are, however, several similarities with other drives like hunger and thirst. They include arousal and satiation, the involvement of hormones, and control by specific areas in the brain. We will explore these similarities as well as some differences in the following pages.

**Arousal and Satiation**

The cycle of arousal and satiation is the most obvious similarity between sexual motivation and other motivated behaviors such as hunger and thirst. In the 1960s, William Masters and Virginia Johnson conducted groundbreaking research on the human sexual response. Until then, research had been limited to observing sexual behavior in animals or interviewing humans about their sexual activity. Masters and Johnson (1966) observed 312 men and 382 women and recorded their physiological responses during 10,000 episodes of sexual activity in the laboratory. This kind of research was unheard of at the time; in fact, the researchers had trouble finding journals that would publish their work. Their work on human sexual behavior was the subject of the recent Showtime cable series *Masters of Sex.*

How is sex like and unlike other drives?

Masters and Johnson identified four phases of sexual response ([Figure 7.1](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2518)). The *excitement phase* is a period of arousal and preparation for intercourse. Both sexes experience increased heart rate, respiration rate, blood pressure, and muscle tension. The male’s penis becomes engorged with blood and becomes erect. The female’s clitoris becomes erect as well, her vaginal lips swell and open, the vagina lubricates, her breasts enlarge, and the nipples become erect.

While hunger is mostly a function of time since the last meal, sexual arousal is more influenced by opportunity and sexual stimuli such as explicit conversation or the presence of an attractive person. In contrast to humans, sexual arousal in most mammal species is triggered by a surge in hormones. Another difference between sex and other drives is that we usually are motivated to reduce hunger, thirst, and temperature deviations, but we seek sexual arousal. This difference is not unique, though; for example, we might skip lunch to increase the enjoyment of a gourmet dinner.

During the *plateau phase,* the increase in sexual arousal levels off. Arousal is maintained at a high level for seconds or minutes, though it is possible to prolong this period. The testes rise in the scrotum in preparation for ejaculation; vaginal lubrication increases and the vaginal entrance tightens on the penis. During *orgasm,* rhythmic contractions in the penis are accompanied by ejaculation of seminal fluid containing sperm into the vagina. Similar contractions occur in the vagina. This period lasts just a few seconds but involves an intense experience of pleasure. Orgasm is similar to the pleasure one feels after eating or when warmed after a deep chill, but it is unique in its intensity; the *resolution* that follows is reminiscent of the period of quiet following return to homeostasis with other drives.

After orgasm, males have a *refractory phase,* during which they are unable to become aroused or have another orgasm for minutes, hours, or even days, depending on the individual and the circumstances. Females do not experience a refractory period and can have additional orgasms anytime during the resolution phase. When comparing the sex drive with other kinds of motivation, the male refractory period has an interesting parallel with sensory-specific satiety (see [Chapter 6](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2175.xhtml)); it is called the Coolidge effect. According to a popular but probably questionable story, President Coolidge and his wife were touring a farm when Mrs. Coolidge asked the farmer whether the flurry of sexual activity among the chickens was the work of one rooster. The farmer answered yes, that the rooster copulated dozens of times each day, and Mrs. Coolidge said, “You might point that out to Mr. Coolidge.” President Coolidge, so the story goes, then asked the farmer, “Is it a different hen each time?” The answer again was yes. “Tell that to Mrs. Coolidge,” the president replied. Whether the story is true or not, the ***Coolidge effect—*a quicker return to sexual arousal when a new partner is introduced**—has been observed in a wide variety of species. We will visit the subject again shortly.

**Figure 7.1** Phases of the Sexual Response Cycle.

Source: From Psychology: The Adaptive Mind (2nd ed.), by J. S. Nairne, 2000, Wadsworth, a part of Cengage Learning, Inc.

**The Role of Testosterone**

As important as sex is to humans, it is ironic that so much of what we know about the topic comes from the study of other species. One reason is that research into human sexual behavior was for a long time considered off-limits, and funding was hard to come by. Another reason is that sexual behavior is more “accessible” in nonhuman animals; rats have sex as often as 20 times a day and are not at all embarrassed to perform in front of the experimenter. In addition, we can manipulate their sexual behavior in ways that would be considered unethical with humans. Hormonal control in particular is more often studied in animals because hormones have a clearer role in animal sexual behavior.

***Castration*, or removal of the gonads (testes or ovaries)**, is one technique used to study hormonal effects because it removes the major source of sex hormones; castration results in a loss of sexual motivation in nonhuman mammals of both sexes. Sexual behavior may not disappear completely, because the adrenal glands continue to produce both male and female hormones, though at a lesser rate than the gonads. The time course of the decline is also variable, ranging from a few weeks to five months in male rats (J. M. Davidson, 1966); across several species, animals who are sexually experienced are impaired the least and decline the slowest (B. Hart, 1968; Sachs & Meisel, 1994). Humans are less at the mercy of fluctuating hormone levels than other animals, but when they are castrated (usually for medical reasons, such as cancer), sexual interest and functioning decrease in both males and females (Bremer, 1959; Heim, 1981; Sherwin & Gelfand, 1987; Shifren et al., 2000). The decline takes longer in humans than in rats, but the rate is similarly variable.

Castration has been elected by some male criminals in the hope of controlling aggression or sexual predation, sometimes in exchange for shorter prison sentences. Castration is an extreme therapy; drugs that counter the effects of ***androgens* (a class of hormones responsible for a number of male characteristics and functions**) are a more attractive alternative. Those that block the production of the androgen ***testosterone,* the major sex hormone in males**, have been 80%–100% effective in eliminating deviant sexual behavior such as exhibitionism and pedophilia (sexual contact with children), along with sexual fantasies and urges (A. Rösler & Witztum, 1998; Thibaut, Cordier, & Kuhn, 1996). Chemical castration is either allowed on a voluntary basis or mandated for some offenses in nine states in the United States (M. Park, 2012) and in several other countries. The effects of castration indicate that testosterone is necessary for male sexual behavior, but the amount of testosterone required appears to be minimal; men with very low testosterone levels can be as sexually active as other men (Raboch & Stárka, 1973).

Frequency of sexual activity does vary with testosterone levels *within* an individual, but the testosterone increases appear to be the *result* of sexual activity rather than the cause. For example, testosterone levels are high in males at the *end* of a period in which intercourse occurred, not necessarily before (J. M. Dabbs & Mohammed, 1992; Kraemer et al., 1976). A case report (which is anecdotal and does not permit us to draw conclusions) suggests that just the anticipation of sex can increase the testosterone level. Knowing that beard growth is related to testosterone level, a researcher working in near isolation on a remote island weighed the daily clippings from his electric razor. He found that the amount of beard growth increased just before planned visits to the mainland and the opportunity for sexual activity (Anonymous, 1970).

**Figure 7.2** Female-Initiated Activity During the Menstrual Cycle.

Source: From figure 2 from “Rise in Female-Initiated Sexual Activity at Ovulation and Its Suppression by Oral Contraceptives,” by D. B. Adams, A. R. Gold, and A. D. Burt, 1978, New England Journal of Medicine, 299(21), pp. 1145–1150.

In most mammals, females are unwilling to engage in sex except during ***estrus,* a period when the female is ovulating, sex hormone levels are high, and the animal is said to be in heat**. Human females and females of some other primate species engage in sex throughout the reproductive cycle. Studies of sexual frequency in women have not shown a clear peak at the time of ovulation. However, initiation of sex is a better gauge of the female’s sexual motivation than is her willingness to have sex; women do initiate sexual activity more often during the middle of the menstrual cycle, which is when ovulation occurs ([Figure 7.2](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2522); D. B. Adams, Gold, & Burt, 1978; S. M. Harvey, 1987). The researchers attributed the effect to ***estrogen,* a class of hormones responsible for a number of female characteristics and functions**. Their reasons were that estrogen peaks at midcycle and the women did not increase in sexual activity if they were taking birth control pills, which level out estrogen release over the cycle.

However, testosterone peaks at the same time, and the frequency of intercourse during midcycle corresponds to the woman’s testosterone level (N. M. Morris, Udry, Khan-Dawood, & Dawood, 1987). At menopause, when both estrogen and testosterone levels decline, testosterone levels show the most consistent relationship with intercourse frequency (McCoy & Davidson, 1985). How to interpret these observations is unclear, because testosterone increases in women as a *result* of sexual activity, just as it does in men ([Figure 7.3](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2527); J. M. Dabbs & Mohammed, 1992). However, studies in which testosterone level was manipulated demonstrate that it also contributes to women’s sexual behavior. Giving a dose of testosterone to women increases their arousal during an erotic film (Tuiten et al., 2000). More important, in women who had their ovaries removed, testosterone treatment increased sexual arousal, sexual fantasies, and intercourse frequency, but estrogen treatment did not (Sherwin & Gelfand, 1987; Shifren et al., 2000).

**Figure 7.3** Relationship Between Sexual Behavior and Salivary Testosterone Levels in Men and Women.

Source: From “Male and Female Salivary Testosterone Concentrations Before and After Sexual Activity,” by J. M. Dabbs, Jr. and S. Mohammed, Physiology and Behavior, 52, pp. 195–197, Fig. 1. © 1992 Reprinted with permission from Elsevier Science.

**Brain Structures and Neurotransmitters**

As neuroscientists developed a clearer understanding of the roles of various brain structures, motivation researchers began to shift their focus from drive as a tissue need to drive as a condition in particular parts of the brain. Sexual activity, like other drives and behaviors, involves a network of brain structures. This almost seems inevitable, because sexual activity involves reaction to a variety of stimuli, activation of several physiological systems, postural and movement responses, a reward experience, and so on. We do not understand yet how the sexual network operates as a whole, but we do know something about the functioning of several of its components. In this section, you will see some familiar terms, the names of hypothalamic structures you learned about in [Chapter 6](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2175.xhtml). This illustrates an important principle of brain functioning, that a particular brain area, even a very small one, often has multiple functions.

What is the role of testosterone in sexual behavior?

Two areas are important in sexual behavior in both sexes, the medial preoptic area of the hypothalamus and the medial amygdala. ***The medial preoptic area (MPOA)* is one of the more significant brain structures involved in male and female sexual behavior**. (Be careful not to confuse the medial preoptic area with the median preoptic nucleus, discussed in [Chapter 6](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2175.xhtml). They are both in the preoptic area, which you can locate in [Figure 6.2](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2175.xhtml#s9781506349183.i2238).) Stimulation of the MPOA increases copulation in rats of both sexes (Bloch, Butler, & Kohlert, 1996; Bloch, Butler, Kohlert, & Bloch, 1993), and the MPOA is active when they copulate spontaneously (Pfaus, Kleopoulos, Mobbs, Gibbs, & Pfaff, 1993; Shimura & Shimokochi, 1990). The MPOA appears to be more responsible for performance than for sexual motivation; when it was destroyed in male monkeys, they no longer tried to copulate, but instead they would often masturbate in the presence of a female (Slimp, Hart, & Goy, 1978).

What brain structures are involved in sexual behavior?

**The *medial amygdala* also contributes to sexual behavior in rats of both sexes. Located near the lateral ventricle in each temporal lobe, the *amygdala* is involved not only in sexual behavior but also in aggression and emotions**. The medial amygdala is active while rats copulate (Pfaus et al., 1993), and stimulation causes the release of dopamine in the MPOA (Dominguez & Hull, 2001; Matuszewich, Lorrain, & Hull, 2000). The medial amygdala’s role apparently is to respond to sexually exciting stimuli, such as the presence of a potential sex partner (de Jonge, Oldenburger, Louwerse, & Van de Poll, 1992).

**Figure 7.4** The Sexually Dimorphic Nuclei of the Rat.

Source: From “The Neuroendocrine Regulation of Sexual Behavior,” by R. A. Gorski, pp. 1–58, in G. Newton and A. H. Riesen (Eds.) Advances in Psychobiology (Vol. 2), 1974, New York: Wiley. Reprinted with permission of John Wiley & Sons, Inc.

There are other areas that are involved in sexual behavior but only in the behaviors of a single sex. Especially significant for males is the ***sexually dimorphic nucleus (SDN),* located in the preoptic area (de Jonge et al., 1989). The SDN is three to four times larger in male rats than in females (**[**Figure 7.4**](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2540)**; He, Ferguson, Cui, Greenfield, & Paule, 2013), and a male’s level of sexual activity is related to the size of the SDN**, which in turn depends, at least in part, on protection by testosterone from the cell death that occurs during the pruning stage shortly after birth (He et al., 2013). Destruction of the SDN reduces male sexual activity (de Jonge et al., 1989). The SDN’s connections to other sex-related areas of the brain suggest that it integrates sensory and hormonal information and coordinates behavioral and physiological responses to sensory cues (Roselli, Larkin, Resko, Stellflug, & Stormshak, 2004). Two other hypothalamic structures are also important. The paraventricular nucleus (PVN; see [Figure 6.2](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2175.xhtml#s9781506349183.i2238)) is important for male sexual performance and, particularly, for penile erections (Argiolas, 1999). The ***ventromedial hypothalamus*is active in females during copulation (Pfaus et al., 1993), and its destruction reduces the female’s responsiveness to a male’s advances**(Pfaff & Sakuma, 1979).

**Figure 7.5** Dopamine Levels in the Nucleus Accumbens During the Coolidge Effect.

Source: From “Dynamic Changes in Nucleus Accumbens Dopamine Efflux During the Coolidge Effect in Male Rats,” by D. F. Fiorino, A. Coury, and A. G. Phillips, 1997, Journal of Neuroscience, 17, p. 4852. © 1997 Society for Neuroscience. Reprinted with permission.

For obvious reasons, we know much less about the brain structures involved in human sexual behavior. Functional MRI (fMRI) recording during masturbation has confirmed the involvement of the medial amygdala and PVN in human sexual activity (Komisaruk et al., 2004). PVN neurons are known to secrete **the hormone/neuromodulator *oxytocin*, which contributes to male and female orgasm and the intensity of its pleasure**(Carmichael, Warburton, Dixen, & Davidson, 1994). We will see additional results from human research in the discussion of neurotransmitters. We also know that a few brain structures in humans differ in size between males and females. Because their contribution to sexual behavior is not clear and the size differences may also distinguish homosexuals from heterosexuals, we will defer discussion of these structures until we take up the subject of sexual orientation.

Sexual behavior involves several neurotransmitters, but dopamine has received the most attention. You saw in [Chapter 5](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i1928.xhtml) that dopamine level increases in the nucleus accumbens during sexual activity, and in this chapter that stimulation of the medial amygdala releases dopamine in the MPOA. Dopamine activity in the MPOA contributes to sexual motivation in males and females of several species (E. M. Hull et al., 1999). In males, dopamine is critical for sexual performance: Initially, it stimulates D1 receptors, activating the parasympathetic system and increasing motivation and erection of sexual tissues; as dopamine level increases, it activates D2 receptors, which shifts autonomic balance to the sympathetic system, resulting in orgasm and ejaculation. D2 activity also inhibits erection, which probably accounts in part for the sexual refractory period in males. Interestingly, dopamine release parallels sexual behavior during the Coolidge effect. As you can see in [Figure 7.5](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2545), it increased in the male rat’s nucleus accumbens in the presence of a female, dropped back to baseline as interest waned, and then increased again with a new female (Fiorino, Coury, & Phillips, 1997). The changes occurred even when the male and female were separated by a clear panel, so the dopamine level reflects the male’s interest rather than the effects of sexual behaviors.

Our knowledge about the role of dopamine in human sexual behavior is less precise but nevertheless intriguing. Drugs that increase dopamine levels, such as those used in treating Parkinson’s disease and stimulants, increase sexual activity in humans (Evans, Haney, & Foltin, 2002; Meston & Frolich, 2000). The dopamine system has been reported to be active in the ventral tegmental area in males during ejaculation (Holstege et al., 2003) and in the nucleus accumbens in females during orgasm (Komisaruk et al., 2004). This activity likely reflects a reward response, but, significantly, the activated areas also have strong motor output to the pelvic floor muscles, which are important in orgasmic activity. Variations in the gene for the D4 receptor (*DRD4*) are associated with sexual arousal and functioning (Ben Zion et al., 2006), and one variant is correlated with promiscuity and sexual infidelity (Garcia et al., 2010).

Ejaculation is also accompanied by serotonin increases in the lateral hypothalamus, which apparently contributes further to the refractory period (E. M. Hull et al., 1999). Injecting a drug that inhibits serotonin reuptake into the lateral hypothalamus increases the length of time before male rats will attempt to copulate again and their ability to ejaculate when they do return to sexual activity. Humans take serotonin reuptake blockers to treat anxiety and depression, and they often complain that the drugs interfere with their ability to have orgasms. The antianxiety drug buspirone, by contrast, decreases the release of serotonin and facilitates orgasms (Komisaruk, Beyer, & Whipple, 2008).

An interesting model for the regulation of gender-related aggressive and bonding behaviors has been proposed in the steroid/peptide theory of social bonds (van Anders, Goldey, & Kuo, 2011). According to this theory, the balance among testosterone, oxytocin, and vasopressin determine behaviors such as aggression and intimacy ([Figure 7.6](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2551)). As you probably guessed, a high testosterone level in either sex increases aggression, but it also impairs the formation of close social bonds. Oxytocin (involved in muscle contractions of sexual tissue and in social bonding) and vasopressin (a potent neuromodulator of brain activity) modulate the form of intimacy and aggression. Antagonistic aggression (which includes social dominance, partner acquisition, and defense of partners and territory) is seen in those with low levels of vasopressin, whereas protective aggression (such as defending children or partners) is seen in those with high levels of vasopressin. Intimacy increases oxytocin, but its interaction with testosterone levels determines whether that intimacy is sexual (if testosterone is high) or nurturing (if testosterone is low). Therefore, testosterone levels determine the relative amount of competitive versus nurturing behaviors an individual expresses, whereas oxytocin determines the relative amount of social bonding versus social isolation.

**Figure 7.6** The Steroid/Peptide Theory of Social Bonds.

Source: From “The Steroid/Peptide Theory of Social Bonds: Integrating testosterone and peptide responses for classifying social behavioral contexts,” by S. M. van Anders, K. L. Goldey, & P. X. Kuo. Psychoneuroendocrinology, 36(9). © Elsevier. Reprinted with permission.

**Odors, Pheromones, and Sexual Attraction**

Sexual behavior results from the interplay of internal conditions, particularly hormone levels, with external stimuli. Sexual stimuli can be anything from brightly colored plumage or an attractive body shape to particular odors. Here we will examine the role of odors and pheromones in sexual attraction, with emphasis on how important they might be for humans.

Before we launch into this discussion, we need to have a basic understanding of the olfactory (smell) system. Olfaction is one of the two chemical senses, along with taste. Airborne odorous materials entering the nasal cavity must dissolve in the mucous layer overlying the receptor cells; the odorant then stimulates a receptor cell when it comes in contact with receptor sites on the cell’s dendrites ([Figure 7.7](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2556)). Axons from the olfactory receptors pass through openings in the base of the skull to enter the olfactory bulbs, which lie over the nasal cavity. From there, neurons follow the olfactory nerves to the nearby olfactory cortex tucked into the inner surfaces of the temporal lobes.

**Figure 7.7** The Olfactory and Vomeronasal Systems.

By varying the number of components in odor mixtures, researchers have calculated that humans can distinguish a trillion odors (Bushdid, Magnasco, Vosshall, & Keller, 2014). But we do not have a different receptor for each odor, and an individual neuron cannot produce the variety of signals required to distinguish among so many different stimuli. Researchers have discovered that humans have around 400 different receptor genes that produce an equal number of receptor types, but additional alleles of some of these genes brings the total to about 600 (Olender et al., 2012). Variation in these alleles among individuals suggests considerable variation in what different people can smell. We’re pikers compared with dogs (800); mice (1,100); and the African elephant, which has 2,000 different receptor genes (Niimura, Matsui, & Touhara, 2016). Research has shown that elephants can distinguish people from different tribes by odor and can recognize up to 30 different family members.

There is a good argument to be made for the nose as a sexual organ. The most convincing evidence comes from the study of ***pheromones,*airborne chemicals released by an animal that have physiological or behavioral effects on another animal of the same species**. Most pheromones are detected by the ***vomeronasal organ (VNO)*, a cluster of receptors also located in the nasal cavity**. The VNO is illustrated in [Figure 7.7](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2556), although you will soon see that most researchers believe that it is no longer functional in humans, the victim of evolution as our ancestors developed color vision and came to rely on visual sexual signals (I. Rodriguez, 2004). However, a VNO may not be entirely necessary, because some pheromones and pheromone-like odors can be detected by the olfactory system when an animal’s VNO has been blocked or eliminated surgically (Wysocki & Preti, 2004). The VNO’s receptors are produced by a different family of genes, and the VNO and olfactory systems are separate neurally (P. J. Hines, 1997). Not surprisingly, in animals the VNO’s pathway leads to the MPOA and the ventromedial nucleus of the hypothalamus, as well as to the amygdala (Keverne, 1999).

Pheromones can be very powerful, as you know if your yard has ever been besieged by all the male cats in the neighborhood when your female cat was “in heat.” The female gypsy moth can attract males from as far as two miles away (Hopson, 1979). Pheromones provide cues for kin recognition in animals, influence cycling of sexual receptivity in female mice, initiate aggression in both males and females, and trigger maternal behavior in adults and suckling in infants (Wysocki & Preti, 2004). In pigs, the boar exudes androstenone, which elicits sexual posturing and receptivity in sows. In fact, pig farmers use androstenone as an aid in artificial insemination.

So, do pheromones play a role in human behavior? In spite of the eagerness with which the media and fragrance industry have embraced the topic, the best answer appears to be “maybe . . . maybe not.” We certainly don’t see pheromones controlling sexual behavior as powerfully as they do in animals; in fact, the best candidate for pheromone control of human behavior is the sucking and searching movements in infants in response to breast odors of a nursing woman (Wyatt, 2016; Wysocki & Preti, 2004). Early interest in the possibility of human pheromones was spurred by reports that women living together in dorms tended to have synchronized menstrual periods and that this was caused by sweat-borne compounds that altered the frequency of luteinizing hormone release (Preti, Cutler, Garcia, Huggins, & Lawley, 1986; Preti, Wysocki, Barnhart, Sondheimer, & Leyden, 2003; K. Stern & McClintock, 1998). Later studies have failed to demonstrate menstrual synchrony almost as often as they have succeeded, and the results have been questioned on methodological grounds (Z. Yang & Schank, 2006).

Is there evidence for pheromones in human sexual behavior?

Several other studies have claimed evidence for an influence of pheromones, or at least body odors, on human behavior. This includes amygdala activation from smelling the sweat of first-time skydivers (Mujica-Parodi et al., 2009); increased intercourse opportunities when using aftershave or perfume containing underarm extracts that enhanced the person’s sex-characteristic body odor (Cutler, Friedman, & McCoy, 1998; McCoy & Pitino, 2002); higher alcohol consumption and sociability in males after exposure to fertile female odors (Tan & Goldman, 2015); and men’s higher attractiveness ratings of the scent of women’s T-shirts when women were ovulating (Kuukasjärvi et al., 2004).

Application: Of Love, Bonding, and Empathy

Source: Todd Ahern/Emory University.

Prairie voles are a rare exception among mammals; they mate for life, and if they lose a mate they rarely take another partner. The bonding process (as reviewed in L. J. Young & Wang, 2004) begins with the release of dopamine in reward areas during mating. If dopamine activity is blocked by a receptor antagonist, partner preference fails to develop. Sexual activity also releases the neuropeptides oxytocin and vasopressin, which are likewise required for bonding to take place. Either can facilitate bonding in males or females, but oxytocin is more effective with females and vasopressin with males.

So does any of this apply to humans, who are also monogamous (more or less)? The most apparent parallel involves oxytocin. Oxytocin not only facilitates bonding but also causes smooth muscle contractions, such as those involved in orgasm and in milk ejection during breastfeeding. Blood levels of oxytocin increase dramatically as males and females masturbate to orgasm (M. R. Murphy, Checkley, Seckl, & Lightman, 1990). Oxytocin also contributes to social recognition, which is necessary for developing mate preferences. Male mice without the oxytocin gene fail to recognize females from one encounter to the next (J. N. Ferguson et al., 2000), and human males are better at recognizing previously seen photos of women after receiving oxytocin (Rimmele, Hediger, Heinrichs, & Klaver, 2009). Men given oxytocin also had more activity in the nucleus accumbens while viewing photos of their partners, and they increased their ratings of their partners’ attractiveness, but not of other women they knew (Scheele et al., 2013).

Oxytocin’s bonding effects are not limited to mates and sex partners. Mother-infant bonding is correlated with oxytocin levels during pregnancy and following birth (Feldman, Weller, Zagoory-Sharon, & Levine, 2007), and a gene for the oxytocin receptor is related to parenting sensitivity (Bakermans-Kranenburg & van IJzendoorn, 2008). Oxytocin also apparently explains empathetic behavior in prairie voles. Though we can’t speculate about what the rodents are “feeling,” they respond to a cagemate’s earlier, unobserved stress with increased grooming, and they match the cagemate’s fear response, anxiety-related behaviors, and corticosterone increase (Burkett et al., 2016). Consoling behavior did not occur if the animals received an infusion of an oxytocin receptor antagonist into the lateral ventricles.

But there is no shortage of critics. They point out that no human secretion has been identified as a pheromone, including the “putative human pheromones” regularly used in research studies (Wyatt, 2016; Wysocki & Preti, 2004). Although these compounds may have physiological effects, so do plant odors. Pepper oil, fennel oil, and rose oil can change blood pressure and catecholamine levels (including adrenaline), and the scent of lemon oil increases positive mood. In addition, pheromone studies are criticized for their small sample size, lack of statistical power, lack of replication, and publication bias—the tendency to publish positive results and shelve negative ones. In spite of these concerns, at least one of the critics agrees that we’re able to identify family members by odor and that smell may influence our choice of sexual partner, but he attributes these abilities to a finely tuned sense of smell rather than pheromones (Wyatt).

In most animals, attraction is fleeting, lasting only through copulation or, at best, till the end of the mating season. For a few species, though, pair bonding occurs for years or for a lifetime, as we see in the accompanying Application.

**Concept Check**

**Take a Minute to Check Your Knowledge and Understanding**

* What change in thinking helped researchers see sex as similar to other biological drives?
* What roles do estrogen and testosterone play in sexual behavior in humans?
* In what ways do sensory stimuli influence sexual behavior?

**The Biological Determination of Sex**

Now we need to talk about differences between the sexes and the anomalies (exceptions) that occur. ***Sex* is the term for the biological characteristics that divide humans and other animals into the categories of male and female. *Gender* refers to the behavioral characteristics associated with being male or female.** For our purposes, it will be useful to make two further distinctions: ***Gender role* is the set of behaviors society considers appropriate for people of a given biological sex**, whereas ***gender identity* is the person’s subjective feeling of being male or female**. The term *sex* cannot be used to refer to all these concepts, because the characteristics are not always consistent with each other. Thus, classifying a person as male or female can sometimes be difficult. You might think that the absolute criterion for identifying a person’s sex would be a matter of chromosomes, but you will soon see that it is not that simple.

**Chromosomes and Hormones**

You may remember from [Chapter 1](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i785.xhtml) that when cells divide to produce sex cells, the pairs of chromosomes separate, and each gamete—the sperm or egg—receives only 23 chromosomes. This means that a sex cell has only one of the two sex chromosomes. In mammals, an egg will always have an X chromosome, but a sperm may have either an X chromosome or a Y chromosome. The procreative function of sexual intercourse is to bring the male’s sperm into contact with the female’s egg, or *ovum*. When the male ejaculates into the female’s vagina, the sperm use their tail-like flagella to swim through the uterus and up the fallopian tubes, where the ovum is descending. As soon as one sperm penetrates and enters the ovum, the ovum’s membrane immediately becomes impenetrable so that only that sperm is allowed to fertilize the egg. The sperm makes its way to the nucleus of the ovum, where the two sets of chromosomes are combined into a full complement of 23 pairs. After fertilization, the ovum begins dividing, producing the billions of cells that make up the human fetus. If the sperm that fertilizes the ovum carries an X sex chromosome, the fetus will develop into a female; if the sperm’s sex chromosome is Y, the child will be a male ([Figure 7.8](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2582)).

For the first month, XX and XY fetuses are identical. Later, the primitive ***gonads* (testes and ovaries, the primary reproductive organs)** in the XX individual develop into ***ovaries,* where the ova (eggs) develop**. The ***Müllerian ducts* develop into the uterus, the fallopian tubes, and the inner vagina**, while the Wolffian ducts, which would become the male organs, wither and are absorbed ([Figure 7.9](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2584)). The undifferentiated external genitals become the clitoris, the outer segment of the vagina, and the labia, which partially enclose the entrance to the vagina ([Figure 7.10](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2587)).

What makes a person male or female?

**Figure 7.8** Female and Male X and Y Chromosomes.

If the fetus receives a Y chromosome from the father, the *SRY* (sex-determining region Y) gene on that chromosome produces a protein that causes the primitive gonads to develop into ***testes,* the organs that will produce sperm**. The testes begin secreting two types of hormones (Haqq et al., 1994). ***Müllerian inhibiting hormone* defeminizes the fetus by causing the Müllerian ducts to degenerate**. Testosterone, the most prominent of the androgens, masculinizes the internal organs: **The *Wolffian ducts* develop into the seminal vesicles, which store semen, and the vas deferens, which carry semen from the testes to the penis. A derivative of testosterone, *dihydrotestosterone,* masculinizes the external genitals**. The same structures that produce the clitoris and the labia in the female become the penis and the scrotum, into which the testes descend during childhood.

**Figure 7.9** Development of Male and Female Internal Organs.

Source: Adapted from Our Sexuality (7ed.), by R. Crooks and K. Baur, 1999, Fig. 3.2 p. 46. Stamford, CT: Cengage Learning.

In the absence of a Y chromosome (and *SRY* gene), the primitive gonads of the XX fetus develop into ovaries. The ovaries won’t begin producing estrogens until later, but in humans *default sex* is female, and the uterus, vagina, clitoris, and labia will all develop without benefit of hormones. You should understand that it is not entirely accurate to speak of hormones as being “male” or “female.” The testes and ovaries each secrete both androgens and estrogens, although in differing amounts; the adrenal glands of the kidney also secrete small amounts of both kinds of hormones.

**Figure 7.10** Differentiation of Male and Female Genitals.

Source: Based on Netter (1983).

The hormonal effects we have been discussing are called organizing effects. ***Organizing effects* mostly occur prenatally and shortly after birth; they affect structure and are lifelong in nature**. Organizing effects are not limited to the reproductive organs; they include sex-specific changes in the brains of males and females as well, at least in nonhuman mammals. ***Activating effects* can occur at any time in the individual’s life; they are reversible changes that can come and go as hormone levels change**. Some of the changes that occur during puberty are examples of activating effects.

During childhood, differences between boys and girls other than in the genitals are relatively minimal. Boys tend to be heavier and stronger, but there is considerable overlap. Boys also are usually more active and more aggressive, and interests diverge at an early age. Marked differences appear about the time the child enters puberty, usually during the preteen years. At puberty, a surge of estrogens from the ovaries and testosterone from the testes completes the process of sexual differentiation that began during prenatal development. Organizing effects include maturation of the genitals and changes in body size. Activating effects include breast development in the girl and muscle increases and hair growth in the boy. In addition, the girl’s ovaries begin releasing the ova that have been there since birth (i.e., she begins to *ovulate*), and she starts to menstruate. Boys’ testes start producing sperm, and ejaculation becomes possible. More important from a behavioral perspective, sexual interest increases dramatically, and in the majority of cases, preference for same-sex company shifts to an attraction to the other sex, along with an interest in sexual intimacy.

**Prenatal Hormones and the Brain**

Several characteristics and behaviors can be identified as male typical and others as female typical. This does not mean that the behaviors are somehow more appropriate for that sex but simply that they occur more frequently in one sex than in the other. These differences are not absolute. For example, consider the stereotypical sexual behavior of rats: The male mounts the female from behind, while the female curves her back and presents her hindquarters in a posture called *lordosis*. However, females occasionally mount other females, and males will sometimes show lordosis when approached by another male.

The same hormonal influence responsible for the development of male gonads and genitals affects behavior as well. A male rat will display lordosis (arching of the back) and accept the sexual advances of other males if he was castrated shortly after birth or if he was given a chemical that blocks androgens just before birth and for a short time postnatally (after birth). Similarly, a female rat given testosterone during this critical period will mount other females at a higher rate than usual as an adult ([Figure 7.11](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2595); Gorski, 1974). These behaviors apparently result from the influence of testosterone on the size and function of several brain structures; in other words, the presence of testosterone masculinizes certain brain structures. That statement is somewhat misleading, though, because it is *estradiol,* the principal estrogen hormone, that carries out the final step of masculinization. When testosterone enters a neuron, it is converted to estradiol by a chemical process called *aromatization*. At the critical time when brain masculinization occurs, aromatase increases in the areas that are to be masculinized (Horvath & Wikler, 1999).

What is the effect of “sexualizing” the brain?

**Figure 7.11** A Female Rat Mounting a Male.

Source: From “Sex-Hormone-Dependent Brain Differentiation and Sexual Functions,” in G. Dörner (Ed.), Endocrinology of Sex (pp. 30–37). Leipzig: J. A. Barth. Copyright © 1974 Gunther Dörner. Used with permission.

Until recently, it was believed that feminization of the brain, like the sex organs, required only the absence of testosterone; now we know that just as masculinization of the male brain requires estradiol, so does feminization of the female brain. Female knockout mice born unable to produce estradiol display less sexual interest and receptivity toward males or females as adults than do other mice, even when they are given replacement estrogens (Bakker, Honda, Harada, & Balthazart, 2002, 2003). Just as the male brain must be masculinized and the female brain feminized, the male brain must also be defeminized. Again, estrogens are necessary; male knockout mice lacking the estrogen receptor showed normal male sexual behavior but also were receptive to advances of other males (Kudwa, Bodo, Gustafsson, & Rissman, 2005).

This sexualization of the brain is reflected in behavioral differences, affecting not only sexual activity but also play behavior, spatial activity, and learning performance (see Collaer & Hines, 1995). Do hormones have a similar influence in humans? In the following pages we will try to answer that question.

**Concept Check**

**Take a Minute to Check Your Knowledge and Understanding**

* How is the sex of a fetus determined, and what affects prenatal and postnatal sexual development?
* What effect do sex hormones have on differentiation of the brain and behavior?

**Gender-Related Behavioral and Cognitive Differences**

In his popular book *Men Are From Mars, Women Are From Venus*, John Gray (1992) said that men and women communicate, think, feel, perceive, respond, love, and need differently, as if they are from different planets and speaking different languages. How different are men and women? This question is not easily answered, but it is not for lack of research on the topic. The results of studies are often ambiguous and contradictory. One reason is that different researchers measure the same characteristic in different ways. Also, the research samples are often too small to yield reliable results, and the subjects are usually not selected in a manner that ensures accurate representation of the population. Whether the differences that do exist are influenced by biology or are solely the product of experience is controversial. Contemporary parents often make efforts to rear their children equally, but it can be difficult to separate oneself from ingrained societal expectations; parents who claimed equality as a childrearing value were found to verbalize differently and play differently with a child dressed as a girl than when the same child was dressed as a boy (Culp, Cook, & Housley, 1983). Differential rearing, of course, could account for marked differences in behavior, temperament, and self-expectations.

**Some Demonstrated Male-Female Differences**

Back in 1974, Eleanor Maccoby and Carol Jacklin reviewed more than 2,000 studies and concluded that the evidence firmly supported three differences in cognitive performance and one difference in social behavior: (1) Girls have greater verbal ability than boys, (2) boys excel in visual-spatial ability, (3) boys excel in mathematical ability, and (4) boys are more aggressive than girls. Later research has supported these differences to some extent, but with qualifications. First, there is considerable overlap between males and females in these characteristics. Second, the differences are rather specific. For example, females excel in verbal fluency and writing but not in reading comprehension or vocabulary (Eagly, 1995; Hedges & Nowell, 1995), and male spatial performance exceeds females’ most on tasks requiring mental rotation of a three-dimensional object (like the one in [Figure 7.12](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2604)) and less on other spatial tasks (Hyde, 1996). More significantly, the differences have narrowed during the ensuing four-plus decades, particularly for the cognitive abilities.

**Figure 7.12** A Spatial Rotation Task.

Stories of Brain & Behavior

**Origins of Male-Female Differences**

The best evidence that the three cognitive differences mentioned earlier are partially the result of experience is that they have decreased over the years, presumably as gender roles and expectations have changed (Hedges & Nowell, 1995; Hyde, 1996; Voyer, Voyer, & Bryden, 1995). In fact, testing of 7 million students indicates that the gender difference in average mathematical performance has disappeared in the United States, although boys are slightly overrepresented at both the lower and higher extremes (Hyde, Lindberg, Linn, Ellis, & Williams, 2008). Similar trends were found in a study of 89 countries, and data suggested that progress is due to increasing gender equality (Kane & Mertz, 2012). In addition, the dramatic variation in murder rate in different countries suggests there is also a strong cultural influence on aggression; for example, the murder rate in 2014/2015 was 57.15 per 100,000 of population in Venezuela, 4.88 in the United States, 0.92 in the United Kingdom, and 0.31 in Japan (“List of Countries,” n.d.).

Although environmental influences play a significant role, gender differences in cognition and behavior also owe a great deal to biology. Most often, researchers attribute the differences to the effects of estrogen and testosterone, particularly on the organizational development of the brain during gestation. Supporting this view is the fact that gender differences in the volume of different brain areas correspond to the density of sex hormone receptors in those areas (J. M. Goldstein et al., 2001). Because the effects of sex hormones on brain development are most evident in people with atypical sexual development, we will hold that discussion for the [next section](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2626) and focus here on activating effects occurring after birth.

How do we explain the differences in verbal and spatial abilities and in aggression?

Males who produce low amounts of testosterone during the developmental years are impaired later in spatial ability (Hier & Crowley, 1982), and testosterone replacement in older men improves their spatial functioning (Janowsky, Oviatt, & Orwoll, 1994). Individuals born men who take estrogens because they identify as females (transgender women) increase their scores on verbal fluency tasks, but they lose spatial performance; transgender men taking testosterone lose verbal ability but improve in spatial performance (for references, see Hulshoff Pol et al., 2006). Men kill 30 times as often as women do (Daly & Wilson, 1988), and testosterone is usually blamed for this difference. However, whether testosterone is the cause or the result of aggression is questioned because a variety of studies show, for example, that winning a sports competition increases testosterone and losing decreases it (Archer, 1991). Aggression in males is partly inheritable; genetic effects account for about half the variance in aggression, and aggression is moderately correlated in identical twins even when they are reared apart (Rushton, Fulker, Neale, Nias, & Eysenck, 1986; Tellegen et al., 1988). The source of aggression is a complex subject, and we will deal with it more thoroughly in [Chapter 8](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2763.xhtml).

In The News: GTEx Project Provides New Insight Into the Differences Between the Sexes

In the years since completion of the Human Genome Project, researchers have focused on understanding how differences in genes’ ability to create proteins, or *gene expression,* can provide insight into typical versus atypical human functioning. The Genotype-Tissue Expression (GTEx) Project, which began in 2010, aims to identify differences in gene expression in tissues and organs that have been donated from hundreds of different people, with the hope of better understanding diseases (National Human Genome Research Institute, 2016). Researchers at the Weizmann Institute of Science have recently used some of the GTEx data to determine why human males and females differ in disease risk, behavior, and body type in spite of sharing most genes (Gershoni & Pietrokovski, 2017). Of the approximately 20,000 genes that they examined, the researchers identified more than 6,500 that were expressed differently in at least one tissue of males compared with females. Sex-based gene expression differences were found not just in the reproductive organs and mammary glands but also in the heart, the skin, the liver, and parts of the brain. These differences in gene expression likely relate to sex differences not only in fat storage, body hair, and milk production and release but also in medical issues such as drug responsiveness, heart muscle disease, and risk for Parkinson’s disease. Of particular interest, there were more mutations in male-specific genes than female-specific genes, suggesting that natural selection acts less strongly on males than females, possibly since males can create many more offspring in a lifetime (due to both gestation time and the Coolidge effect).

**Thought Questions**

1. Why might so many genes be expressed differently in males and females?
2. What sorts of sex differences in behavior might be explained by gene expression differences?

For the news story, visit edge.sagepub.com/garrett5e and select the [Chapter 7](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml) study resources.

Differences in brain functioning are also cited as bases for gender differences. Jerre Levy (1969) hypothesized that women outperform men on verbal tests because they are able to use both hemispheres of the brain to solve verbal problems rather than mostly the left hemisphere. This idea has obviously been controversial; there has been some support from fMRI studies, but there have also been negative findings, and a meta-analysis of fMRI studies during language tasks found no lateralization differences between males and females (Sommer, Aleman, Bouma, & Kahn, 2004). This doesn’t settle the issue, according to Harrington and Farias (2008), who claim that many fMRI studies are not as methodologically rigorous as they could be, and point out that gender differences should be expected only on some types of tasks. A more recent strategy has been to measure functional connectivity. Some support for Levy’s hypothesis comes from studies that found greater connectivity within hemispheres in males and greater connectivity between the hemispheres in females (Ingalhalikar et al., 2014; Tomasi & Volkow, 2012).

A meta-analysis of spatial performance did confirm gender differences, with males relying primarily on the right hemisphere and females showing no hemisphere preference (J. M. Vogel, Bowers, & Vogel, 2003). Imaging studies indicate that men use parietal areas to perform spatial rotations, whereas women rely more on frontal areas (reviewed in Andreano & Cahill, 2009), and that men’s scores on the task are correlated with the amount of cortical surface in the parietal lobes (Koscik, O’Leary, Moser, Andreasen, & Nopoulos, 2009). In an fMRI study, men performed a spatial memory task by activating the right hippocampus, whereas women relied on the left hippocampus and reported using a verbal strategy (Frings et al., 2006).

There are also several other indications that male and female brains work differently. Males and females have different patterns of brain activation during learning (Andreano & Cahill, 2009), pain (Naliboff et al., 2003), and stress (J. Wang et al., 2007). Males are genetically more resistant to pain, and males and females respond differently to different pain medications (J. Bradbury, 2003). Men are less affected by stress (Matud, 2004), and, as you will see in subsequent chapters, males are more susceptible to autism, Tourette syndrome, and attention-deficit/hyperactivity disorder, whereas women are more likely to suffer from depression and Alzheimer’s disease. Research described in the accompanying In the News feature suggests that some of these differences may be due to male-female differences in expression of their genes.

The value of studying these differences is not to determine whether one sex is smarter or more aggressive or healthier than the other but to understand what contributes to the characteristics in either sex. Keep in mind that aside from physical strength and possibly aggressiveness, the differences are small and do not justify discrimination in society or in the workplace. We are far more alike than we are different; this is a reason to use the term *other sex* instead of *opposite sex*. There are real differences, though, and an understanding of their origins could help us enhance intellectual development, reduce violence, and cure or manage diseases. From a scientific perspective, that knowledge also helps us understand how the brain develops, an issue that we will continue to pursue in the next two sections.

**Concept Check**

**Take a Minute to Check Your Knowledge and Understanding**

* What are the origins of male-female differences in verbal and spatial abilities?
* What are the arguments for environmental origins and for biological origins of male-female differences in cognitive abilities and behaviors?

**Biological Origins of Gender Identity**

For decades, sex researchers have argued about what shapes an individual’s gender identity, with some believing it is formed in the first few years of life by a combination of rearing practices and genital appearance (Money & Ehrhardt, 1972) and others claiming that chromosomes and hormones are more important (M. Diamond, 1965). Our earlier discussion of the effects of XY and XX chromosomes was the simple version of the sex-determination story; in reality, development sometimes takes an unexpected turn. As you will soon see, the results not only challenge our definition of what is male and what is female but also tell us a great deal about the influence of biology on gender.

*There is no one biological parameter that clearly defines sex.*

*—Eric Vilain*

**Gender Dysphoria**

**Individuals who believe they have been born into the wrong sex are referred to as *transgender***. They may dress and live as the other sex, take hormones to feminize or masculinize their bodies, or undergo surgery for sex reassignment. It is estimated that 0.1%–0.5% of people in the United States and the United Kingdom have taken some steps to transition to the other sex (Gates, 2011). **Distress that people may feel due to the perception that their sex does not match their gender is called *gender dysphoria***. The best therapeutic outcomes have resulted from the requested hormonal and surgical treatment rather than psychotherapy alone (Saraswat, Weinand, & Safer, 2015). Gender dysphoria is estimated to occur in 0.005%–0.014% of adult males and in 0.002%–0.003% of adult females (American Psychiatric Association, 2013). Gender dissatisfaction that appears as early as three or four years of age appears to be due mostly to family environmental influences (Knafo, Iervolino, & Plomin, 2005); among adolescents the heritability is estimated at 62%, with the remainder due to nonfamily environmental influence (Coolidge, Thede, & Young, 2002). Specific genes that have been identified are alleles of the *CYP17* gene and the *AR* gene. The first increases testosterone levels in female-to-male transgender individuals (Bentz et al., 2008) and the second reduces sensitivity to androgen in male-to-female transgender individuals (Hare et al., 2009); the researchers believe these genes lead to masculinization of the female brain and a failure to masculinize the male brain.

What influences affect gender identity and gender-related behavior?

**Figure 7.13** BSTc Size in a Male-to-Female Transgender Individuals.

Source: Figure 2, “A Sex Difference in the Human Brain and Its Relation to Transsexuality,” by J.-N. Zhou, M. A. Hofman, L. J. Gooren, and D. F. Swaab, 1995, Nature, 378, pp. 68–70. Reprinted by permission of Nature, copyright 1995.

How could an individual’s genitals and brain develop at variance with each other? They differentiate sexually at different times during gestation, the genitals during the first two months and the brain during the last half, presumably allowing them to fall under the influence of independent processes (Bao & Swaab, 2011). Several studies suggest that the brains of transgender individuals have followed a different developmental path, finding that various brain structures are more like those of their identified sex than of their birth sex in size, shape, or patterning (reviewed in Saraswat et al., 2015). **The *third interstitial nucleus of the anterior hypothalamus (INAH3)* is larger in men than in women and is thought to be the human counterpart of the sexually dimorphic nucleus**. It and the bed nucleus of the stria terminalis (BSTc, which controls autonomic, neuroendocrine, and behavioral responses) are larger in males than in females, but they have been reported to be female sized in male-to-female transgender individuals ([Figure 7.13](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2635); Garcia-Falgueras & Swaab, 2008; Kruijver et al., 2000; J. N. Zhou, Hofman, Gooren, & Swaab, 1995). INAH3 is believed to correspond to the sexually dimorphic nucleus in animals. Female-typical brain responses have also been reported in male-to-female transgender individuals. In a study with presumed pheromones, an androgen derivative found in male sweat (4, 16-androstadien-3-one, or AND) activated the anterior hypothalamus, whereas an estrogen-like compound found in female urine (estratetraenol, or EST) did not (Berglund, Lindström, Dhejne-Helmy, & Savic, 2008). In a second study, when male-to-female transgender individuals viewed an erotic video, their pattern of brain responses resembled those of women rather than men (Gizewski et al., 2009). The studies are vulnerable to a variety of criticisms, however: Some, though not all, of the studies are potentially contaminated because the subjects were undergoing hormone treatment; studies have rarely been replicated, and results from attempts have been inconsistent; and “data-snooping” brain scans—measuring a large number of structures because the researcher doesn’t know what to look for—increases the probability of incorrectly identifying one of them as different, especially in the small samples that are typical of research on transgender individuals.

For a variety of reasons, obtaining reliable data on gender identity has not been easy; the sexual variations described in the sections that follow serve as “natural experiments” that provide valuable, and sometimes dramatic, additional information.

**46 XY Difference in Sexual Development**

At puberty Jan had failed to develop breasts or to menstruate; instead, her voice deepened, and her body became muscular, while her clitoris grew to a length of 4 centimeters (1½ inches) and her vaginal lips partially closed, giving the appearance of a male scrotum. Once comfortable with her tomboyishness, she was now embarrassed by her appearance and increasingly masculine mannerisms; she withdrew from peers, and her school performance began to suffer. To everyone’s surprise, her doctor discovered two undescended testes in her abdomen and no ovaries. After a psychiatric evaluation, Jan’s parents and doctors offered Jan the opportunity to change to a male sexual identity. She immediately went home and changed into boy’s clothing and got a boy’s haircut. The family moved to another neighborhood where they were unknown. At the new high school, Jack became an athlete, excelled as a student, was well accepted socially, and began dating girls. Surgeons finished closing the labia and moved the testes into the newly formed scrotum. He developed into a muscular, 6-foot-tall male with a deep voice and a heavy beard. At the age of 25 he married, and he and his wife reported a mutually satisfactory sexual relationship (Imperato-McGinley, Peterson, Stoller, & Goodwin, 1979).

Today Jack would be diagnosed with ***46 XY difference in sexual development* (46 XY DSD), meaning that he had a typical number of chromosomes, including an X and a Y chromosome, but his sexual development was atypical for those chromosomes**. The term adopted by practitioners in 2006 used the word *disorder* in place of *difference.* Some observers contend that sexual development is a continuum, and prefer the term *difference* in place of *disorder*; in respect for that view, we will use DSD here to mean “difference in sexual development.” These variations in development have a variety of causes. The reason for Jack’s unusual development was a deficiency in an enzyme (17α-hydroxysteroid) that converts testosterone into dihydrotestosterone; dihydrotestosterone masculinizes the external genitalia before birth. The large surge of testosterone at puberty enabled his body partially to carry out that process.

A deficiency in another enzyme, 5α-reductase, also reduces dihydrotestosterone levels and delays genital development; this deficiency is genetic and is most likely to occur when there is frequent intermarriage among relatives. Of 18 such individuals in the Dominican Republic who were reared unambiguously as girls, all but one made the transition to a male gender identity after puberty, and 15 were living or had lived with women (Imperato-McGinley, Peterson, Gautier, & Sturla, 1979). The men said they realized they were different from girls and began questioning their sex between the ages of 7 and 12. Although their transition argues for the influence of genes and hormones on gender identity, such a conclusion must be tentative because the individuals had a great deal to gain from the switch in a society that puts a high premium on maleness (Cohen-Kettenis, 2005).

Eden Atwood ([Figure 7.14](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2641)) is a widely acclaimed jazz singer. She has recorded and performed all over the world and with the biggest names in jazz. Ms. Atwood is also remarkable for having been born with XY chromosomes and two testes. Her ***androgen insensitivity syndrome,* a form of 46 XY DSD, is caused by a genetic absence of androgen receptors, which results in insensitivity to androgen**. Müllerian inhibiting hormone suppresses development of most of the female internal organs, but because the individual is unaffected by androgens, the testes do not descend and the external genitals develop as more or less feminine (depending on the degree of insensitivity), with a shallow vagina. If the genitals are mostly feminine, the child is reared as a girl, and at puberty her body is further feminized by estrogen from the testes and adrenal glands. The condition may not be recognized until menstruation fails to occur at puberty or when unsuccessful attempts to become pregnant lead to a more complete medical examination. In the absence of testosterone’s influence, androgen-insensitive individuals tend to have well-developed breasts and a flawless complexion. Because these characteristics are often combined with long, slender legs, androgen-insensitive males repeatedly turn up among female fashion models (J. Diamond, 1992).

**Figure 7.14** Eden Atwood.

ZUMA Press, Inc. / Alamy

**46 XX Difference in Sexual Development**

**A female fetus may be partially masculinized by excess androgen and by some hormone treatments during fetal development, resulting in *46 XX difference in sexual development***. The internal organs are female, because no Müllerian inhibiting hormone is released, but the external genitals are virilized to some extent; that is, they have some degree of masculine appearance. In extreme cases, the clitoris is as large as a newborn male’s penis, and the external labia are partially or completely fused to give the appearance of an empty scrotum.

[Figure 7.15](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2646) illustrates one cause of 46 XX DSD; ***congenital adrenal hyperplasia (CAH),* which results from an enzyme defect that causes the individual’s adrenal glands to produce large amounts of androgen during fetal development and after birth until the problem is treated**. Postnatal hormone levels can be normalized by administering corticosteroids, and the parents often choose reconstructive surgery to reduce the size of the clitoris and eliminate labial fusion, giving the genitals a more feminine appearance. If masculinization is more pronounced, the parents may decide to rear the child as a boy; in that case, the surgeons usually finish closing the labia and insert artificial testes in the scrotum to enhance the masculine appearance. Recent work indicates that the most common cause of CAH, 21-hydroxylase deficiency, can be detected in the womb and treated with a synthetic corticosteroid to reduce genital ambiguity (Nimkarn & New, 2010).

**Figure 7.15** Female Infant With Congenital Adrenal Hyperplasia.

Source: Used with permission of Thomas A. Wilson, MD, The School of Medicine at Stony Brook University Medical Center.

Obviously, sex cannot always be neatly divided between male and female. Some experts believe that two categories are not sufficient to describe the variations in masculinity and femininity. Anne Fausto-Sterling (1993) advocates at least five sexual categories. The ones between male and female are often referred to as *intersex conditions,* a term that is not used in the medical profession but is preferred by many individuals. It would be easy to get caught up in the unusual physical characteristics of these individuals and to be distracted from our question: What makes a person male or female? This question is the topic of the accompanying Application as well as the next few pages.

**Cognitive and Behavioral Effects**

As mentioned earlier, reversing the sex hormone balance during prenatal development changes the brain and later behavior in nonhuman animals. Is it possible that masculinization and feminization of the developing brain account for sex differences in behavior and cognitive abilities in humans as well? If so, then we would expect the behavior and abilities of individuals who have experienced an excess or a deficit of androgen during prenatal development to be at odds with their chromosomal sex.

That is indeed the case. Androgen-insensitive males are like females in that their verbal ability is higher than their spatial performance, and their spatial performance is lower than that of other males (Imperato-McGinley, Pichardo, Gautier, Voyer, & Bryden, 1991; Masica, Money, Ehrhardt, & Lewis, 1969). And though there have been contradictory results, evidence favors increased spatial ability in CAH women (Puts, McDaniel, Jordan, & Breedlove, 2008). Androgen-insensitive 46 XY individuals also are typically feminine in behavior, have a strong childbearing urge, and are decidedly female in their sexual orientation (M. Hines, 1982; Money, Schwartz, & Lewis, 1984; J. M. Morris, 1953). Although 95% of CAH women reared as girls accept a female identity, they also show behavioral shifts in the masculine direction (Dessens, Slijper, & Drop, 2005). They have been reported to be tomboyish in childhood (M. Hines); to prefer boys’ toys, such as trucks and building blocks (Berenbaum, Duck, & Bryk, 2000); and to draw pictures more typical of boys, using darker colors and including mechanical objects and excluding people (Iijima, Arisaka, Minamoto, & Arai, 2001). They also more often report male-dominated occupational choices (30% vs. 13%), interest in rough sports (74% vs. 50%), and interest in motor vehicles (14% vs. 0%; Frisén et al., 2009). There is evidence that these effects are due to androgen levels before birth rather than during postnatal development (Berenbaum et al., 2000). Homosexual or bisexual orientation has been reported to be as high as 37% (Money et al., 1984) and at 19% in a recent larger study (Frisén et al.).

What are the behavioral implications of 46 XX and 46 XY DSD?

Some critics claim that humans are sexually neutral at birth, and they attribute the cognitive and behavioral effects we have just seen to feminine or ambiguous rearing in response to the child’s genital appearance. (You may be beginning to appreciate the deficiencies of natural experiments.) However, some of the findings are difficult to explain from a socialization perspective. For example, the anti-miscarriage drug diethylstilbestrol (DES) given to women in the 1950s and 1960s has an androgen-like effect in the brain but does not virilize the genitals, yet DES-exposed daughters reported increased homosexual fantasy and behavior (Ehrhardt et al., 1985; Meyer-Bahlburg et al., 1995). In another study, girls exposed to a similar drug and who had nonvirilized genitals scored higher in aggression than their unexposed sisters (Reinisch, 1981). In addition, the fact that androgen-insensitive 46 XY individuals perform even lower on spatial tests than their unaffected sisters and female controls (Imperato-McGinley et al., 1991) can be explained by insensitivity to androgens but not by “feminine rearing.”

*The evidence accumulated so far strongly suggests that man is no exception with regard to the influence of sex steroids on the developing brain and subsequent behavior.*

*—Anke Ehrhardt and Heino Meyer-Bahlburg*

Application: Sex, Gender, and Sports

Cameron Spencer/Getty Images Sport/Getty.

When Caster Semenya of South Africa won the gold medal in the 800-meter race at the 2009 World Championships in Athletics, her strong performance and masculine physique aroused suspicions about her gender. Fueled by years of media reports that some female competitors might actually be men, the International Association of Athletics Federations (IAAF) and the International Olympic Committee (IOC) had introduced routine gender testing in the 1960s (J. L. Simpson et al., 1993). However, physical examination was soon rejected as unacceptable to many women, and chromosome testing turned out to be inadequate as a measure of performance advantage. For example, Barr body analysis, which identifies cells with XX chromosomes, rejects androgen-insensitive XY individuals though they receive no benefit from testosterone, but it would pass XXY males, who do benefit. The IOC and the IAAF ended routine testing in the 1990s, though both reserved the authority to request gender identification on an individual basis.

Semenya agreed to an IAAF request to undergo extensive gender testing. In the meantime, reports were leaked that Semenya had two testes and triple the normal level of testosterone for a female. The IAAF said that if the reports turned out to be accurate, it would pay for corrective surgery; the surgery would remove the internal testes, which have a high risk for cancer, and eliminate the source of the extra testosterone. When the IAAF received the report, it did not reveal the results to the public, but nearly a year after the championships, Semenya was cleared to compete again—as a woman. Some sexual activists argue that if society would place less emphasis on gender, whether a person is male or female wouldn’t matter, but this case suggests there is a need for better understanding of what it means to be male or female.

The IOC tried to skirt the gender issue in 2012 by announcing it would bar athletes from competing as females if they had normal male levels of androgens and were responsive to androgens. However, this policy on hyperandrogenism was suspended in 2015 when female Indian sprinter Dutee Chand, who had a high testosterone level, brought suit in the Court of Arbitration for Sport (Branch, 2015). The court decided that evidence demonstrating testosterone-related increased athletic performance in women was lacking and gave the IAAF until 2017 to provide data supporting its claim for hormone-induced enhanced female performance.

Sources: “Caster Semenya Must Wait . . . ,” 2010; S. Hart, 2009; Macur, 2012; O’Reilly, 2010; Powers, 2010.

**Ablatio Penis and Other Natural Experiments**

The “neutral-at-birth” theorists claim that individuals reared in opposition to their chromosomal sex generally accept their sex of rearing and that this demonstrates that rearing has more effect on gender role behavior than chromosomes or hormones (studies reviewed in M. Diamond, 1965). Diamond, who advocates a “sexuality-at-birth” hypothesis, argues that the reason individuals with ambiguous genitals accept their assigned gender is that sex of rearing is usually decided by whether the genital appearance is predominantly masculine or feminine, which in turn reflects the influence of prenatal hormones. According to Diamond, there was no case in the literature where an *unambiguously* male or female individual was successfully reared in opposition to the biological sex. He and others (such as Money, Devore, & Norman, 1986) have described several instances in which individuals assigned as one sex successfully shifted to their chromosomal and gonadal sex in later years, long after the assumed window for forming gender identity (the first few years of life) had closed. Failures in predicting the later gender identity of a child with ambiguous genitals has led several experts (along with the advocacy group Accord Alliance) to advocate waiting until the child can give informed consent, or at least indicates a clear gender preference; others are reluctant to see the child subjected to the social difficulties that result from an ambiguous appearance.

In 1966, an eight-month-old boy became the most famous example of resistance to sexual reassignment when the surgeon using electrocautery to perform a circumcision turned the voltage too high and destroyed the boy’s penis (called *ablatio penis*). After months of consultation and agonizing, Bruce’s parents decided to let surgeons transform his genitals to feminine-appearing ones. While sounding radical even today, this has remained the recommended practice for *ablatio penis* cases. The neutral-at-birth view was widely accepted then, and the psychologist John Money counseled the parents that they could expect their son to adopt a female gender identity (M. Diamond & Sigmundson, 1997). Bruce would be renamed Brenda, and “she” would be reared as a girl. This case study had two characteristics lacking in other “natural experiments”: The child was normal before the accident, and he happened to have an identical twin brother who served as a control.

Over the next several years, Money (1968; Money & Ehrhardt, 1972) reported that Brenda was growing up feminine, enjoying her dresses and hairdos, and choosing to help her mother in the house, while her “typical boy” brother played outside. But developmental progress was not nearly as smooth as Money claimed (M. Diamond & Sigmundson, 1997). Brenda was in fact a tomboy who played rough-and-tumble sports and fought, preferred her brother’s toys and trucks over her dolls, and even preferred to urinate in a standing position. She had private doubts about her sex beginning in the second grade, decided she was a boy at age 11, and decided to switch to living as a male at 14. Only then did Brenda’s father tell her the story of her sexual transition in infancy. Then, said Brenda, “everything clicked. For the first time things made sense and I understood who and what I was” (p. 300).

*Gender identity is sufficiently incompletely differentiated at birth as to permit successful assignment of a genetic male as a girl.*

*—John Money*

*An extensive search of the literature reveals no case where a male or female without some sort of biological abnormality . . . accepted an imposed gender role opposite to that of his or her phenotype.*

*—Milton Diamond*

Brenda changed her name to David and requested treatment with testosterone, removal of the breasts that had developed under estrogen treatment, and construction of a penis. The person who was isolated and teased as a girl was accepted and popular as a boy, and he attracted girlfriends. At age 25, he married Jane and adopted her three children. Although he was limited in his sexual performance, he and Jane engaged in sexual play and occasional intercourse.

But life was still not ideal. He brooded about his childhood and was often angry or depressed; after 14 years, Jane told him they should separate for a while. Troubled by his past and his present, and perhaps a victim of heredity—his mother had attempted suicide, his father became an alcoholic, and his twin brother died of an overdose of antidepressants—one spring day in 2004 David Reimer took his own life ([Figure 7.16](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2666); Colapinto, 2004).

**Figure 7.16** David Reimer, 1965–2004.

Source: © STR/Reuters/Newscom.

Although only seven cases of ablatio penis have been examined in the literature, there are numerous instances of male infants born with a missing or underdeveloped penis, not involving hormonal causes. These include malformation of the pelvic area (cloacal exstrophy) and absence of a penis (penile agenesis). All of these conditions require decisions about surgical intervention and gender rearing (Meyer-Bahlburg, 2005). In 311 such individuals reared as male, all accepted that role, with only one indicating possible gender dysphoria. Of the 77 reared as females, 22% transitioned later to male and 13% more exhibited possible gender dysphoria. In another study of individuals with cloacal exstrophy who were assigned a female gender shortly after birth, most exhibited male-like behaviors and had switched to a male persona later in life (Reiner & Gearhart, 2004). The accompanying Application describes a recent case that highlights the need for better understanding when a child’s gender must be chosen.

**Concept Check**

**Take a Minute to Check Your Knowledge and Understanding**

* How do the sexual anomalies require you to rethink the meaning of male and female?
* What reasons can you give for thinking that the brains of people with sexual anomalies have been masculinized or feminized contrary to their chromosomal sex?

**Sexual Orientation**

Sex researchers, along with the rest of us, spend a great deal of time arguing about why some people are attracted to members of the same sex. Whether we know it or not, we are also asking why most people are heterosexual. The answer to that question may seem obvious, but the fact that a behavior is nearly universal and widely accepted does not mean that it requires no explanation. People who are attracted to members of their own sex may be able to tell us not only about homosexuality but also about the basis for sexual orientation in general.

Why is it difficult to measure sexual orientation rates?

It is difficult to estimate how many people are homosexual; the numbers vary from study to study and from one country to another, due to differences in definition and sampling methods, as well as reluctance to admit membership in a stigmatized group. In a review of nine studies, the average rate in the United States was 3.5%, with Canada, Australia, the United Kingdom, and Norway ranging from 1.2% to 2.1% (Gates, 2011). In the United States, prevalence was equally divided between men and women. Although almost two thirds of the nonheterosexual men identified themselves as exclusively homosexual, only one third of nonheterosexual women did so. Interestingly, when Gallup asked Americans what percentage of the U.S. population they thought was gay or lesbian, the average answer was 23% (Newport, 2015). That response hasn’t changed significantly since 2002, in spite of increasing acceptance of homosexuality and access to greater information. Homosexual experiences are fairly common, especially during adolescence and in the absence of heterosexual opportunities. Almost 8% of the population have had at least one same-sex sexual encounter, and as many as 11% report same-sex attraction (Gates, 2011). As Ellis and Ames (1987) point out, these experiences do not make a person homosexual any more than occasional heterosexual activity makes a person heterosexual. About 1% of people express no interest in sex at all (Bogaert, 2004). *Asexuality* is gaining acceptance as an additional category of preference.

Research does not support the belief that gay men are necessarily feminine and lesbians are masculine; only about 44% of gays and 54% of lesbians fit those descriptions (Bell, Weinberg, & Hammersmith, 1981). Even then, they usually identify with their biological sex, so gender role, gender identity, and sexual orientation are somewhat independent of each other and probably have different origins.

Application: Who Chooses a Child’s Sex?

Source: Barcroft/Barcroft Media/Getty Images.

M.C. was born diagnosed with ovotesticular DSD; he had a normal penis and a scrotum and testosterone levels were high, but there was a small vaginal opening below his penis. His physicians recommended corrective surgery, but even though he was identified as male at birth, four months later they decided he would be reassigned as a girl. Because M.C. was a ward of the state, the South Carolina Department of Social Services approved the surgery, which was performed at the age of 16 months, just 3 months before he was adopted by Pam and Mark Crawford. M.C. behaved more like a boy than a girl; he wanted a haircut like his dad, and he wanted to use the men’s restroom and to be referred to as a boy. At the age of 7 he chose to begin living as a boy, an identity supported by his family, friends, school, pediatrician, and religious leaders. M.C.’s adoptive parents filed a legal suit against the Medical University of South Carolina, the Greenville Health System, and the Department of Social Services, on the grounds that the decision should have been left to the boy. Defense attorneys got the case dismissed from federal court by arguing that the doctors had no way of knowing at the time that they were violating M.C.’s constitutional rights, but the state case is proceeding. (For sources, see On the Web at the end of the chapter.)

It is not clear what causes homosexuality, which means that we do not know how to explain heterosexuality either. There is considerable evidence for biological influences on sexual orientation, or else the topic would not appear in this chapter. But because social influences are commonly believed to be more important, we will consider this position first.

**The Social Influence Hypothesis**

It has been argued that homosexuality arises from parental influences or is caused by early sexual experiences. Bell and his colleagues (1981) expected to confirm these influences when they studied 979 gay and 477 heterosexual men. But they found no support for frequently hypothesized environmental influences, such as seduction by an older male or a dominant mother and a weak father.

Several developmental experiences do seem to differentiate homosexuals from heterosexuals, and these have been considered evidence for a social learning hypothesis (Van Wyk & Geist, 1984). But these experiences—such as spending more time with other-sex playmates in childhood, learning to masturbate by being masturbated by a member of the same sex, and homosexual contact by age 18—can just as easily be interpreted as reflecting an early predisposition to homosexuality. In fact, Bell and his associates (1981) concluded that adult homosexuality “is just *a continuation of the earlier homosexual feelings and behaviors from which it can be so successfully predicted*” (p. 186; italics in the original). However, they did find more evidence for an influence of learning on bisexuality than on exclusive homosexuality. This suggests that there might be a biological influence that varies in degree, with experience making the final decision in the individuals with weaker predispositions for homosexuality.

Among homosexuals, 70% remember feeling “different” as early as four or five years of age (Bell et al., 1981; Savin-Williams, 1996). Memories of feelings are suspect, because they are easily distorted in light of today’s circumstances. Memories of behavior are somewhat more reliable, and home videos from childhood are better yet. These behavioral measures show a high rate of ***gender nonconformity,* including mannerisms and dress typical of the other sex, a tendency to engage in activities usually preferred by the other sex, and an atypical preference for other-sex playmates and companions while growing up** (Bell et al., 1981; Rieger, Linsenmeier, Gygax, & Bailey, 2008). If we are to entertain a biological hypothesis of sexual orientation, though, we must come up with some reasonable explanation for how it is formed and how it is altered. There are three biological approaches to the question: *genetic, hormonal,* and *neural*.

**Genetic and Epigenetic Influences**

Twin and family studies provide the most documented evidence for a biological basis for sexual orientation. Homosexuality is seen two to seven times more often among the siblings of homosexuals than it is in the general population (J. M. Bailey & Bell, 1993; J. M. Bailey & Benishay, 1993; Hamer, Hu, Magnuson, Hu, & Pattatucci, 1993). Studies in the early 1990s reported concordances of about 50% in identical twins for both men and women (J. M. Bailey & Pillard, 1991; J. M. Bailey et al., 1993). However, when subjects were recruited without regard to their sexual preference, concordances for men fell to 37% in one study and 18% in another, and women’s concordances dropped to 30% and 22% (J. M. Bailey, Dunne, & Martin, 2000; Långström, Rahman, Carlström, & Lichtenstein, 2010). Presumably the earlier data suffered from volunteer bias, due to homosexual individuals’ greater willingness to volunteer if they had a homosexual sibling.

**Figure 7.17** Possible Locations of Genes for Male Homosexuality.

Source: Based on data from “Genetic and Environmental Effects on Same-Sex Sexual Behavior: A Population Study of Twins in Sweden,” by N. Långström, Q. Rahman, E. Carlström, and P. Lichtenstein, 2010, Archives of Sexual Behavior, 39, pp. 75–80.

The search for specific genes has been frustrating, which is not uncommon when multiple genes are involved; because any number of combinations of the genes can produce the behavior, a particular gene can have a significant effect in one study and go undetected in the next. The most successful research on male homosexuality has involved a stretch of DNA on the X chromosome. Dean Hamer and his associates (1993) focused their attention there because gay men have more gay relatives on the mother’s side of the family than on the father’s side, and the mother contributes only X chromosomes to her sons. They found that 64% of the pairs of gay brothers they studied shared identical genetic material at one end of the X chromosome, in the region designated as Xq28 ([Figure 7.17a](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2685)). The location received additional confirmation in a meta-analysis of the five studies available in 1999 (Hamer, 1999), a whole-genome study in 2005 (Mustanski et al., 2005), and a recent unusually large study of 409 pairs of homosexual brothers (Sanders et al., 2015). The gene itself has not been identified yet, however. The Sanders study also supported linkage at 8q12, which had been tentatively identified a decade earlier. A whole-genome study implicated a stretch of DNA on chromosome 7 in the 7q36 region (Mustanski et al.), and a later study of Chinese homosexual men implicated an allele of the *SHH* gene there ([Figure 7.17b](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2685); B. Wang et al., 2012). *SHH* contributes to the patterning of organ development, from growth of fingers to organization of the brain, but it is also involved in male-male sexual activity, at least in fruit flies. Female sexual orientation, by contrast, does not appear to be linked to 7q36 (S. Hu et al., 1995; Ngun & Vilain, 2014).

What is the evidence for a biological basis for homosexuality?

Evidence that homosexuality is influenced by genes presents a Darwinian contradiction; how could homosexuality survive when its genes are unlikely to be passed on by the homosexual individual? Italian researchers have offered an intriguing proposal; the birth rate is higher in women on the mother’s side of the family of male homosexuals, so they conclude that genes responsible for homosexuality also increase the women’s birth rate—compensating for the homosexual’s lack of productivity (Camperio Ciani, Corna, & Capiluppi, 2004; Iemmola & Camperio Ciani, 2009). A later analysis indicated that this effect is best explained by two genes, at least one of which is on the X chromosome; the researchers also suggested that the genes increase attraction to men, in both men and women (Camperio Ciani, Cermelli, & Zanzotto, 2008).

In the face of recent data indicating that environmental influences are stronger than previously thought, we need to look further for the sources. As you can see in [Figure 7.18](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2694), unique environmental factors are much greater than shared (family) influences (Långström et al., 2010). At the age of three or four, when prehomosexual feelings and behaviors typically appear, there has been little opportunity for social influences outside the family to come into play; this means that the prenatal environment is the more likely source of these unique influences. A variety of epigenetic mechanisms have been proposed as a prenatal factor. In females, one of each pair of X chromosomes in every cell is turned off by methylation; which of the two X chromosomes gets turned off usually varies randomly from cell to cell. However, inactivation of the same chromosome occurred in 90% of the cells in 13% of women with a homosexual son and 23% of the mothers of two or more gay sons, compared with only 4% of women with no gay sons (Bocklandt, Horvath, Vilain, & Hamer, 2006). Another suggestion is that an epigenetic modification of testosterone sensitivity that occurs in a parent as compensation for an atypical testosterone level could be passed on to the offspring (W. R. Rice, Friberg, & Gravilets, 2012). If a man with low testosterone transfers his increased sensitivity to a daughter, or a woman with high testosterone transfers her decreased sensitivity to a son, it could have significant effects on the offspring’s sexual and gender development. In the most promising effort so far, researchers at UCLA studying identical twins discordant for homosexuality identified a pattern of methylation in five DNA regions; the pattern predicted which individuals were homosexual with 70% accuracy (Ngun et al., 2015). The researchers cautioned that it is too early to know how well this predictive ability will generalize outside the sample.

**Figure 7.18** Genetic and Environmental Contributions to Sexual Orientation.

Source: Based on data from “Genetic and Environmental Effects on Same-Sex Sexual Behavior: A Population Study of Twins in Sweden,” by N. Långström, Q. Rahman, E. Carlström, and P. Lichtenstein, 2010, Archives of Sexual Behavior, 39, pp. 75–80.

**Prenatal Influences on Brain Structure and Function**

For early researchers, the most obvious biological explanation was that homosexuality is due to atypical sex hormone levels. Their attempts to reverse male homosexuality by administering testosterone not only were not successful but often increased homosexual activity (for references, see A. C. Kinsey, Pomeroy, Martin, & Gebbard, 1953). Later studies measured hormonal levels and found no evidence of either a deficit or an excess of sex hormones (Gartrell, 1982; Meyer-Bahlburg, 1984). However, by manipulating hormonal levels during gestation and shortly after birth, researchers were able to produce same-sex preference in rats, hamsters, ferrets, pigs, and zebra finches (for references, see LeVay, 1996).

Which brain structures are different in homosexual males?

Critics say this effect has no bearing on human behavior, claiming that spontaneous homosexual behavior occurs in animals only when members of the other sex are unavailable and that it does not represent a shift in sexual orientation. However, about 10% of male sheep prefer other males as sex partners, and some form pair bonds in which they take turns mounting and copulating anally with each other (Perkins & Fitzgerald, 1992). A few female gulls observed on Santa Barbara Island off the coast of California form “lesbian” pairs—courting, attempting copulation, taking turns sitting on their nest, and sharing parenting if some of the eggs were fertilized during an “unfaithful” interlude with a male (Hunt & Hunt, 1977; Hunt, Newman, Warner, Wingfield, & Kaiwi, 1984). A shortage of males could be a contributing factor, but the gulls’ behavior is atypical of opportunistic homosexuality in that the majority of birds stay paired for more than one season.

If sex hormones play a role in human sexual orientation, they probably do so by altering brain development during gestation. It is difficult to measure prenatal hormone levels in humans, so researchers have looked for evidence for hypo- or hyper-masculinization of homosexuals’ brains by examining differences known or thought to be influenced by sex hormones during development. A few differences in brain structures have been reported, including equal-sized cerebral hemispheres in gay males and straight women, with a larger right hemisphere in lesbian females and heterosexual men (Savic & Lindström, 2008); and a female-sized INAH3 ([Figure 7.19](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2704); LeVay, 1991) and suprachiasmatic nucleus (Swaab & Hofman, 1990) in gay men. In rats, blocking the effects of testosterone in male rats during the prenatal period and shortly after birth increased the number of vasopressin-secreting cells; as adults, the rats preferred the company of a sexually active male rather than an estrous female, and they showed lordosis and accepted mounting from the male (Swaab, Slob, Houtsmuller, Brand, & Zhou, 1995). From a functional perspective, a few studies have reported that homosexual males perform better on verbal tests and poorer on spatial tests than do heterosexual men ([Figure 7.20](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2709); Collaer, Reimer, & Manning, 2007; C. M. McCormick & Witelson, 1991; Rahman, Abrahams, & Wilson, 2003). Evidence that homosexual women perform like men has been inconsistent (Collaer et al., 2007; Gladue, Beatty, Larson, & Staton, 1990; Rahman et al., 2003). Also, some studies have reported that homosexual men and women respond to presumed pheromones similarly to members of the other sex ([Figure 7.21](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2712); Savic, Berglund, & Lindström, 2005; W. Zhou et al., 2014). Finally, relative finger length is considered a marker of fetal androgen exposure, and the ratio of index finger to ring finger length has been reported to be male typical in lesbian women (T. J. Williams et al., 2000).

**Figure 7.19** INAH3 in a Heterosexual Man (Left) and a Homosexual Man (Right).

Source: From “A Difference in Hypothalamic Structure Between Heterosexual and Homosexual Men,” by S. LeVay, Science, 253, pp. 1034–1047. © 1991, American Association for the Advancement of Science (AAAS). Reprinted with permission from AAAS.

You will notice that most of the differences relate to males; this is partly due to an unfortunate research focus on male homosexuality and partly because the results have more often been negative when studying females. The latter may be because homosexual women are more often bisexual, which could lead to disparate results; for that reason, studies should divide nonheterosexual women into two groups. Something else you might have picked up on is that these studies are several years old. That is not damning in itself, but it should be disturbing that most of these lines of research—on such a socially controversial topic—have not seen replication. We are faced with the dilemma whether to draw conclusions based on either a single study or a few studies with inconsistent results, or to suspect that subsequent studies have yielded negative results, which often don’t find their way into print. At this point we lack a clear idea of what forces determine human sexual preference. What does seem likely is that the answer will turn out to be a multiplicity of factors, rather than any single influence.

*The most powerful sex organ is between the ears, not between the legs.*

*—Milton Diamond*

**Figure 7.20** Sex-Atypical Cognitive Performance in Homosexual Men and Women.

Sources: (a) Based on data from “Sexual-Orientation-Related Differences in Verbal Fluency,” by Q. Rahman, S. Abrahams, and G. D. Wilson, 2003, Neuropsychology, 17, pp. 240–246. (b) Based on data from “Visuospatial Performance on an Internet Line Judgment Task and Potential Hormonal Markers: Sex, Sexual Orientation, and 2D:4D,” by M. L. Collaer, S. Reimers, and J. T. Manning, Archives of Sexual Behavior, 36, pp. 177–192.

**Figure 7.21** Responses of Heterosexual Women, Homosexual Men, and Heterosexual Men to a Presumed Male Pheromone.

Source: From “Smelling of Odorous Sex Hormone-Like Compounds Causes Sex-Differentiated Hypothalamic Activations in Humans,” by I. Savic et al., Neuron, 31, pp. 661–668, fig. 1. © 2002.

**Social Implications of the Biological Model**

As is often the case, the research we have been discussing has important social implications. If homosexuality is a choice, as argued by some, then U.S. civil rights legislation does not apply to homosexuals, because protection for minorities depends on the criterion of unalterable or inborn characteristics (Ernulf, Innala, & Whitam, 1989). About 75% of homosexuals believe that homosexuality is inborn, and that they have no choice (Leland & Miller, 1998). When Congressman Barney Frank of Massachusetts ([Figure 7.22](https://jigsaw.vitalsource.com/books/9781506349220/epub/OEBPS/s9781506349183.i2495.xhtml#s9781506349183.i2717)) was asked if he ever considered whether switching to the straight life was a possibility, he replied, “I wished it was. But it wasn’t. I can’t imagine that anybody believes that a 13-year-old in 1953 thinks, ‘Boy, it would be really great to be a part of this minority that everybody hates and to have a really restricted life’” (Dreifus, 1996, p. 25).

But some people in the gay community think that promoting this view is not in their best interest. For them, the biological model is associated too closely with the old medical “disease” explanation of homosexuality. They fear that homosexuals will be branded as defective, or even that science may find ways to identify homosexual predisposition in fetuses and that parents will have the “problem” corrected through genetic manipulation or abortion. Emotions are so strong among some homosexuals that the researcher Dick Swaab was physically attacked in Amsterdam by members of the Dutch gay movement, who felt threatened by his biological findings (Swaab, 1996).

**Figure 7.22** Former U.S. Congressman Barney Frank.

Other gay and lesbian rights activists welcome the biological findings because they think that belief in biological causation will increase public acceptance of homosexuality. Polls indicate they are right; in a survey of four different cultures, 56%–85% of people who believed homosexuals are “born that way” held significantly more positive views (Ernulf et al., 1989). In the United States, moral acceptance of homosexuality has risen from 40% in 2001 to 60% in 2016, and in the past 20 years belief that same-sex marriage should be legal has increased from 27% to 61% (“Gay and Lesbian Rights,” 2016). The nature-nurture debate will not be settled to everyone’s satisfaction anytime soon, but most researchers believe that when we understand the origins of homosexuality and heterosexuality, they will include a combination of heredity, hormones, neural structures, and experience (LeVay, 1996).