

Behavioral Genetics and Homosexuality

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Abstract

Behavioral genetics is a branch of science that investigates the genetic influence on human behavior. The science of behavioral genetics, however, is often misunderstood by the typical layperson and even by the knowledgeable social scientist. The purpose of this paper is to explain basic concepts of behavioral genetics and its application to understanding the possible causes of homosexuality. The author concludes that although homosexuality may be influenced by genetic factors (as are all complex psychosocial behaviors), it is not determined solely or even primarily by genetic factors. Homosexuality is a complex psycho-social-biological phenomenon with possible genetic, environmental, and freewill influences.

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In order to explore the position that homosexuality is innate and has a direct genetic cause, it is imperative to understand basic concepts and assumptions about behavioral genetics (Bazzett, 2008). This paper will explain fundamental principles of behavioral genetics and will explore how behavioral genetics can lead to an understanding of homosexuality.

Before proceeding, an explanation of terms is required. *Homosexuality* is usually defined as a complex phenomenon involving thoughts, feelings, and behaviors. The *thinking* aspect includes the self-definition of one's sexual orientation as heterosexual, homosexual, bisexual (or some other variation of these labels, such as mostly heterosexual or bi-curious); the thinking part also includes fantasies and other related cognitions. The *feeling* aspect is one's emotional and physical attraction to people of the same sex, to those of the opposite sex, or to those of both sexes. It includes both romantic feelings and sexual attraction, which is sometimes referred to as *same-sex attraction* (SSA). The third element of homosexuality is *behavior*—the act of having intimate sexual interaction with another person or persons or the act of self-masturbation with or without pornography.

Not all individuals have a clear and seamless interface between the thinking, feeling, and behavioral elements of homosexuality. A person may self-identify as “heterosexual” and have romantic and erotic feelings for people of the opposite sex, but may have engaged in same-sex relations. Or a person may self-identify as “homosexual” and have romantic and erotic feelings for people of the same sex, but may not have actually engaged in same-sex relations.

These three aspects of homosexuality complicate the issue of how genes influence homosexuality. We must then ask the following: *Which aspects of homosexuality are influenced by genes and which elements are more influenced by environment?*

Same-gender sexual attraction may be more connected to heredity than is self-identity as gay or lesbian. However, for the sake of simplicity, the *genetic influence on homosexuality* refers to the way in which genes affect the total complex of thinking,

feeling, and behavior. Furthermore, in this paper a homosexual is defined as one who is consciously and persistently sexually attracted to those of the same gender, has engaged in homosexual behavior and intends to do so in the future, and who self-identifies as gay or lesbian.

Many people believe that genes cause complex psychosocial behavior (Ridley, 2003), yet this is not the case. In most instances, behavior results from genetic influence interacting with environmental inputs and with self-determination, such as moral agency (Lerner, 2006). Genetic influence, however, can be misunderstood by the educated layperson and by the naïve social scientist, especially when the newspaper headline reads, “Gene X Has Been Found to Cause Behavior Y” (Jang & Vernon, 2005). In almost all cases, such a headline is a gross overstatement. Genes do not directly cause behavior, but rather genes create the code for proteins that, through a long series of biochemical processes, eventually have some influence on behavior (Bazzett, 2008; Norgate & Richardson, 2006). The leap from an identified gene to a specific behavior is very complex and convoluted (Jang & Vernon, 2005; Rutter, 2006). As Wine (2000) stated, “It is very difficult to jump from gene to behavior, or more generally to bridge the chasm between genotype and phenotype” (p. 1).

Part 1: Overview of Basic Genetics

Each body cell contains forty-six chromosomes, twenty-three inherited from the mother and twenty-three from the father. Chromosomes are tiny, coiled strings of DNA—deoxyribonucleic acid—that microscopically look something like a tightly twisted ladder with rungs in the middle supported by sidebars (Carey, 2003). An average chromosome has about 100 million nucleotides or nitrogenous bases (Klug, Cummings, Palladino, & Spencer, 2009).

There are four types of nucleotides: thymine linked to adenine (TA links) and its reverse (AT), and cytosine linked to guanine (CG links) and its reverse (GC). The

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nucleotides form two chains and are connected by sugar-phosphate molecules, which give structural support to the nucleotides; the nucleotides form the DNA molecule, which in turn makes up the chromosome. A very long string of nucleotides, made up of thousands of base pairs, comprise a single gene.

A gene is a short segment of DNA in a particular location on a specific chromosome (Bazzett, 2008). Geneticists believe there are 20,000 to 25,000 genes in human DNA (Carey, 2003). Alternative forms of genes are known as alleles. For example, there are three distinct alleles that result in blood types A, B, and O.

An average length of about 1,500 nucleotides makes a gene. But the typical chromosome only has 2,000–3,000 genes, so most of the chromosome is non-coding DNA. This means the majority of our DNA (about 97 percent) does not code for proteins but has other functions—such as structural support or gene regulation—in other words, turning genes off and on (Kolb & Whishaw, 2004).

If you unwind and string out a single chromosome, revealing all its base pairs (AT, TA, GC, and CG), the string would be almost identical in every person. But in every few thousand nucleotides, there would be a small difference that would make the chromosome unique—for example, a TA might be replaced with a CG. While there are approximately three billion nucleotides in the human genome, human DNA varies from one person to another by only a few million nucleotides! These minor variations in DNA segments that do not result in diseases or disability are known as polymorphisms.

Genes carry instructions, much in the way a building construction blueprint does; they instruct the body to manufacture proteins or to activate or deactivate other genes (Plomin, DeFries, & McClearn, 1980). Genes also spell out the “order in which amino acids should be assembled to construct a certain protein” (Kolb & Whishaw, 2004, p. 94). Single genes usually make one protein or part of one protein; for example, the DNA sequence AAC-GTA-TCG-CAT would be read as a polypeptide chain of four amino acids: leucine-histidine-serine-valine (Cary, 2003).

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Genes can influence behavior in three ways: (1) by the action of a single gene of major effect, such as phenylketonuria (PKU); (2) by a small number of genes of moderate effect, called *oligogenic*, such as those that cause celiac disease; or (3) by many genes of small effect, called *polygenic*, such as those that cause heart disease or diabetes.

Abnormal genes (or gene mutations) have alterations in their base sequence—such as additions, deletions, or substitutions of nucleotides—that then affect protein synthesis. Three outcomes of gene mutation are possible. First, an abnormal protein may be produced that has little or no noticeable effect on normal functioning. Second, the abnormal protein may have a significant negative effect on organism functioning. Third, the abnormal protein may have little effect on the organism under normal living conditions but may have a significant adverse effect under certain stressful environmental conditions, such as malnutrition, abuse, disease, toxic chemicals, or abnormal hormonal changes. “This last category of abnormal gene function is considered a *genetic predisposition*” (Bazzett, 2008, p. 49).

The word *protein* comes from the Greek “*proteos*,” meaning “of primary importance.” A protein is a long chain of amino acids folded up into a specific three-dimensional shape. There are twenty common amino acids, but these can combine in various ways to make thousands of proteins. Proteins made in cells may remain in the cell to support cell structure and function, or they may be excreted and exported to other parts of the body. Examples of proteins include enzymes, antibodies, and some hormones and neurotransmitters.

The action of genes is sequential. First, a DNA segment is transcribed into messenger RNA. The messenger RNA is then translated by ribosomes (small biochemical factories in the cell) into a chain of amino acids. As chromosomes move about the nucleus, they change shape and expose new segments of DNA to intracellular fluid, which triggers the process of transcription and translation of a different gene into a new protein.

Four Facts about Genes

Fact 1. Thus far, scientists have identified only a few physical disorders that are caused by the action of a single gene (Quarrell, 2007). These include Huntington's disease, cystic fibrosis, early-onset familial-type Alzheimer's disease, and PKU. As an example, PKU is caused by a single gene mutation on Chromosome 12 that causes the inability to digest the amino acid phenylalanine—an amino acid that is part of most protein-rich foods, including meat, milk products, and nuts. If not treated, PKU can lead to mental retardation, stunted growth, and emotional-behavioral problems.

Another example of a single gene action is Huntington's disease, which is caused by overrepetition (also called a repeat expansion) of the three-base sequence CAG on the short arm of Chromosome 4. Instead of the usual five to twenty-five triplets of CAG (cytosine, adenine, and guanine), people with Huntington's have from forty to almost two hundred CAG triplets in a row. "The abnormally expanded CAG segment leads to the production of a defective Huntington protein that contains a long stretch of the amino acid glutamine. This elongated protein disrupts the normal function of nerve cells in certain parts of the brain, and ultimately leads to the death of those cells" (*Genetics Home Reference*, 2008). The loss of brain cells causes the devastating symptoms of Huntington's disease, including uncontrolled movements, emotional disturbance, and dementia.

Still another example is fragile X syndrome, which is caused by a similar process but results from the expansion of the triplet CGG on the long arm of the X chromosome; instead of the normal twenty-five to fifty repeats, there are a hundred to four hundred repetitions. The normal gene produces codes for a protein (FMRP) that regulates other proteins involved in learning and memory. When this gene doesn't work, the brain produces too many synapses too quickly, and the synapses are immature and fragile, thus the name "fragile X syndrome." Children with fragile X often suffer from autism, mental retardation, and ADHD.

Fact 2. Complex behaviors (such as homosexuality) probably involve multiple genes that are affected by a variety of environmental events (Human Genome Project Information, 2008). If many genes influence sexual orientation, then the phenomenon we call homosexuality is polygenic. Researcher D. H. Hamer and colleagues have identified one gene on the X chromosome that may be implicated in homosexuality in some men (Hamer, Hu, Magnuson, Hu, & Pattatucci, 1993). However, the existence, number, and location of candidate genes affecting sexual orientation have not been determined, and efforts to identify any that may exist continue to be unsuccessful (Byne, 1995, 2007). In some psychopathological conditions, such as autism and depression, more progress has been made in identifying multiple genes and their mechanisms on biochemistry (Jang & Vernon, 2005).

In addition to the possible polygenic nature of homosexuality, this trait or condition is also multifactorial—in other words, it has many aspects or elements, including physical, psychological, social, and even political (Carey, 2003). Each element or aspect of homosexuality may have a different genetic and environmental basis (Ridley, 2003).

In summary, homosexuality appears to be a polygenic and multifactorial phenomenon composed of several elements, and each element is probably influenced by many genes.

Fact 3. Single-gene traits usually produce discrete phenotypes. In the case of genetic diseases, either one has the disease or does not have the disease. Environment usually has little influence on these single-gene “qualitative” conditions. On the other hand, a polygenic trait results in a range of behavioral outcomes. Clinical depression is an example of a polygenic trait with a range of phenotypes. Depression has many symptoms, such as feelings of hopelessness, trouble concentrating, fatigue, feeling restless and irritable, and insomnia. Each of these symptoms is probably influenced by a different gene or gene combination, and each gene probably has lesser or greater sensitivity to environmental actions (Jang & Vernon, 2005). In addition, there is a wide range of depression phenotypes that can result in conditions ranging from mild chronic depression to severe acute depression.

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Numerous studies have demonstrated that homosexuality (especially in men) is not a singular phenomenon but has a range of mental, emotional, and physical outcomes. Many homosexual men are not exclusively homosexual, and their sexual thoughts and behavior may vary and change over time. A homosexual man may have sex mostly with men, but also may have sex with women on occasion. A person who is currently bisexual may later self-identify as exclusively heterosexual or as exclusively homosexual. This fluidity of sexual thoughts, feelings, and behaviors is additional evidence that homosexuality is not a single-gene trait.

Another characteristic of polygenic traits is that they are more likely to be influenced by environmental inputs than single genes. Most mental illnesses are thought to be polygenic conditions, and there are many effective therapies for most acute and chronic conditions. In like manner, if homosexuality is polygenic, then unwanted homosexual desire and behavior should show some susceptibility to change due to education, therapy, other types of intervention, or other factors. In some cases, this has been shown to be true (Diamond, 2008; Jones & Yarhouse, 2007; Nicolosi, 2009; Spitzer, 2003).

Fact 4. Environments can and do affect the operation (or expression) of genes (Hubbard & Wald, 1999). Environments influence the functioning of DNA by turning protein-coding genes on and off. The social milieu can modify the proteins produced in various tissues and organs (Meaney, 2001). The choices we make, the lives we live, and the actions of those who love us or refuse to love us can “alter the very chemistry of our DNA” by turning genes on and off (Begley, 2007, p. 180). “Genes store information coding for amino acid sequences of proteins. That is all. They do not code for parts of the nervous system and they certainly do not code for particular behavior patterns” (Bateson & Martin, 2001, p. 34).

The relatively new science of epigenetics has demonstrated unequivocally that physical and psychosocial environments can and do turn specific genes on and off (Cabej, 2008; Church, 2009; Ridley, 2004). “At its most basic, epigenetics is the study of changes in gene activity that do not involve alterations to the genetic code but still get passed on to at least one successive generation” (Cloud, 2010, p. 2). The epigenome sits on top of and is

entangled with the genome, much like a series of light switches along a string of Christmas lights; the epigenome turns genes off or on due to other genes or environmental stimuli such as disease, stress, diet, or prolonged feelings of love or loneliness. “Lifestyle choices can change the epigenetic marks atop your DNA in ways that cause genes to express themselves too strongly . . . or too weakly” (Cloud, 2010; cf. Arai, Li, Hartley, & Feig, 2009).

In his book *The Genius in All of Us*, David Shenk (2010) states that the new science of epigenetics “obliterates the long-standing metaphor of genes as blueprints with elaborate predesigned instructions” (p. 16). Shenk declares we now have a more accurate metaphor:

Rather than [genes as] finished blueprints, genes are more like volume knobs and switches [in a recording studio].....Many of those knobs and switches can be tuned up/down/on/off at any time by another gene or by any minuscule environmental input. This flipping [of switches] and turning [of knobs] takes place constantly” in the human genome. (p. 16)

Shenk summarizes:

We do not inherit traits directly from our genes. Instead, we develop traits through the dynamic process of gene-environment interaction. In the GxE [genes and environment act together] world, genetic differences still matter enormously. But, on their own, they don’t determine who we are. (p. 18)

Thus any trait, condition, or behavioral outcome results from the interaction of genes and the environment.

Suomi (2004) provides an exemplary example of how environment can impact genes. The researchers bred rhesus monkeys that were born with various temperaments. Some showed high emotional reactivity—they became extremely excited and agitated

when separated from their mothers or challenged by a novel experience—and some displayed low reactivity. The differences appeared to be due to levels of serotonin in the brain. Suomi then placed high- and low-reactive baby monkeys with foster mothers who were high or low in reactivity. When the high-reactive babies were raised for six months by the low-reactive foster mothers, they displayed normal emotional reactivity even when separated from these mothers and put in cages with peers and unknown adult monkeys.

Suomi (2004) concluded that even heritable characteristics, such as fear and aggression in monkeys, can be shaped and modified by the environment, and “this is especially true of early attachments” (p. 43). Monkeys with a genetic proclivity to be timid and afraid can, with good mothering, overcome those developmental deficiencies. Suomi’s research makes a strong argument that DNA is not destiny and that behavior is a result of gene-environment interaction.

Examples of environmental events affecting the brain and biochemistry of humans have also been found (Haviland et al., 2006). Cohen and colleagues (2002) have data to suggest that early childhood sexual abuse in boys leads to abnormalities in the temporal regions of the brain that may increase one’s risk for becoming a pedophile.

These four facts about genes are uncontroversial and articulate the consensus of the scientific community. When considered together, these facts lead to the conclusion that homosexuality, like any other complex psycho-social-biological behavior, is not absolutely determined by a single gene or even by a group of genes. Environmental influences must be considered.

Part 2: Key Concepts in Behavioral Genetics

Behavioral geneticists (BGs) try to determine the unique and independent contribution of genetic and environmental influences to individual differences in behavior (DiLalla, 2004). Three key terms in this definition—*behavior*, *genetic influence*, and *environment*—are explained below.

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1. **Behavior** refers to “observable actions, or even emotions and moods”; it can be “unconscious, automatic, or instinctual.” In a broader sense, even “personality” is a behavior (Baker, 2004, pp. 2–3). Behavior includes one’s outward appearance and actions, but also includes emotions, moods, and mental states (Bazzett, 2008). Behavior may also be referred to as a phenotype.

2. **Genetic influence** as a term is usually misunderstood. People interpret the phrase to mean that there is a powerful path directly from genes to behavior. This is not true. Genes produce proteins and enzymes that pass through multiple biochemical processes; these processes may eventually produce small changes in cell structure or functioning that may, in turn, influence behavior within a certain environmental context.

3. **Environment** is any nongenetic influence, including internal biological entities such as nutrients, bacteria, viruses, and medicines (Baker, 2004). Environment includes any forces that impinge upon the person, such as family and neighborhood, peers, schools, the media, and even the climate and geography. Likewise, natural disasters, disease, and war are environmental factors.

BGs acknowledge that both environment and genes influence behavior and rarely assume “that one or the other is omnipotent” (Plomin et al., 1980, p. 374). Yet BGs also emphasize the powerful influence of genes on all types of human behavior, including intelligence, personality, criminality, and even belief in God (Owen, McGuffin, & Gottesman, 2001; Plomin, DeFries, Craig, & McGuffin, 2003). The ultimate goal of many BGs is to find specific genes that cause harmful physical or psychological conditions, and then to figure out ways to change the genetic influence (Owen et al., 2001). This could be done by creating a drug that counteracts (or blocks) harmful protein synthesis or by replacing the defective gene with a functional gene that will produce the correct protein at the right time and place in the body.

Now that the three terms have been explained, their interaction will be described. Gene-environment interaction (GEI) implies that genes act differently under different

environmental conditions. Environmental conditions, such as prenatal or postnatal influences, make possible the expression or suppression of various genes at different times in development. Because of GEI, it is nearly impossible to disentangle the separate and unique effects of genes and environment on complex psychosocial behaviors such as personality, intelligence, and sexual orientation.

Heritability is a critical concept in behavioral genetics, but is one that is difficult to understand, and in the end it has little value in understanding the etiology of homosexuality (Ofstedal, 2005). A technical definition is as follows: Heritability is the proportion of phenotypic variation that is attributable to genotypic variation. Said another way, “Heritability describes the extent to which genetic differences among individuals in a population make a difference phenotypically” (Plomin et al., 1980, p. 224). Put simply, heritability refers to traits that are similar in parents and offspring ([Stanford Encyclopedia of Philosophy](#), 2009). It means that physical characteristics or behavioral traits among kin are probably due to genetic variations in that particular family group. Heritability is an estimate of the relative contributions of genetic and environmental factors to a particular expressed trait, condition, or behavior.

Heritability is calculated using a complex series of mathematical formulas and is indicated by a numerical value that varies from 0 to 1. A heritability quotient of 0 indicates no genetic contribution to individual differences in phenotype, while a quotient of 1 indicates the behavior (the phenotype) is completely determined by genetic variation. Red hair would have a heritability of 1; a preference for red hybrid Honda automobiles probably has heritability near 0. Heritability is a population parameter and tells us nothing about individuals. For instance,

a heritability of .40 informs us that, on average, about 40% of the individual differences that we observe in say, shyness [in a particular population] *may* in some way be attributable to genetic individual difference. It does not mean that

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40% of any person's shyness is due to his or her genes and the other 60% is due to his or her environment. (Heritability: Introduction, 2009; Sesardic, 2005)

Michael Rutter (2006) illustrates heritability using the example of schizophrenia. Based on his meta-analyses of twin studies, he asserts: "The proband-wise concordance rate for schizophrenia in monozygotic twin pairs was [on average] 41–65 percent, as compared with 0–28 percent for dizygotic twin pairs, giving rise to a heritability estimate of appropriately 80 to 85 percent" (p. 65, also Footnote 1). This means that 80 percent of the variation in schizophrenia—in a specific sample at a specific point in time—is due to genetic variation in the sample population. This does not indicate that 80 percent of the reason why a particular person is schizophrenic is genetic. It simply means that there is probably "something" passed down from parents to children through genetic mechanisms that increase the offspring's chance of developing schizophrenia. But what that "something" is—the elusive gene or genes, as well as the dynamics of the GEI—has not been identified.

Now suppose that in a family study, heritability for homosexuality was found to be .30. This means that 30 percent of the variation in sexual orientation among a particular family group is assumed to be due to genetic variation, and 70 percent of the variation in sexual orientation is assumed to be a result of environmental experiences, including both shared and non-shared environments. Shared environments are those conditions that members experience equally, such as the family socioeconomic status or parenting style. Non-shared environments are the unique experiences that one member has but that other family members do not share, such as sexual abuse, exposure to pornography, or rejection by same- or opposite-sex peers.

A heritability estimate of .30 does not predict that three out of ten brothers of homosexual men will become homosexual. Nor would this estimate indicate that, for a specific person, 30 percent of his homosexuality is due to genetics and 70 percent is due

to environment. Heritability “is a relative percentage only—relative to the contributions from common environment and non-shared environments. In twin studies, heritability “is a measure of the balance between genetic and environmental influence on a trait at a [specific] place and point in time” (Whitehead & Whitehead, 2008, p. 15). In other words, heritability estimates only indicate that there is probably something in the genetic pool *of this sample at this time* that may be related—in other words, a correlation, not a cause—to the likelihood of a person expressing homosexuality. Heritability estimates say nothing about any individual’s likelihood of engaging in homosexual behavior.

Heritability quotients have serious limitations (Sesardic, 2005; Tabery, 2006). First, a heritability estimate only applies to the sample from which it was derived. Second, heritability will rise or fall due to environmental conditions. In a society intolerant of homosexuality, as exists in many Muslims countries, the heritability quotient would be much smaller than in Denmark or Holland, where homosexuality is tolerated and widely accepted. Third, many large samples are needed to obtain valid heritability estimates applicable to a wider population, and this has not been done. Fourth, heritability is a population parameter like the mean for height; the average height of a population tells you nothing about the tallness or shortness of any particular individual. Likewise, the mean for height does not explain why a particular person is short or tall. A heritability quotient cannot be used to predict who will be the lanky basketball player or who will be the petite gymnast.

William Byne (2007) summarizes well some of the problems with using the concept of heritability to understand the appearance and prevalence of a given trait, like homosexuality:

Heritability reflects only the degree to which a given trait is associated with genetic factors. It says nothing about the specific genetic factors involved or about the mechanisms through which they exert their influence. Furthermore,

heritability gives no information about how a particular trait might change under different environmental conditions. (p. 82)

Part 3: Studies of Genetic Influence on Homosexuality

“There are basically three kinds of inquiry used to demonstrate a genetic basis for [homosexuality]: family studies [also called gene linkage studies], twin studies, and adoption studies” (Lewontin, Rose, & Kamin 1984, p. 213). The simple idea behind all these studies is that if relatives of homosexuals report same-sex attraction and/or homosexual behavior at a higher rate than a comparison sample, then homosexuality must have a genetic component (Pattatucci, 1998).

A typical twin study works this way. Identical twins (monozygotic or MZ) and fraternal twins (dizygotic or DZ) are recruited where at least one of the twins is homosexual. The usual sample is a convenience sample recruited through gay and lesbian publications, websites, or homosexual support groups (Bailey & Dawood, 1998). The twins are asked to identify their sexual orientation in various ways, such as the gender of the objects of their physical and emotional attractions, their self-reported sexual orientation, and their number of ever or recent same-sex partners. Past studies, whose results have not been replicated by other studies, had shown that if an identical twin is homosexual, his identical brother had a 40 to 50 percent chance of also self-identifying as homosexual (Bailey & Pillard, 1991; Whitam, Diamond, & Martin, 1993). If one fraternal twin is homosexual, his brother has only a 9 to 19 percent chance of also being homosexual (Dawood, Bailey, & Martin, 2009). In adoption studies where a biological child identifies as homosexual, an adopted brother has only a 2 percent to 3 percent chance of being homosexual—about the same as the incidence of male homosexuality in the general population (see Dawood et al., 2009).

In other adoption studies, a child who is adopted at birth is compared to his or her biological parents for similarity of traits. It is assumed that the adoptive child shares genes but no environment with the biological parents and shares environment but no genes with

the adoptive parents. If the adoptive child turns out to be more like a biological parent—both self-identify as homosexual—then that trait is assumed to be genetic.

Such studies appear to show that genes exert some influence on the development of homosexuality. The closer the blood ties, and thus the more genes in common, the more likely that a homosexual boy will have a homosexual brother or a lesbian will have a lesbian sister. However, such studies have limitations. First and foremost is the use of biased samples. A typical sample is composed of homosexuals who know they have a brother or sister who is homosexual, so they readily volunteer for twin research. Thus, the pair-wise concordance rate in such samples may be greatly inflated. Even advocates such as J. Michael Bailey admit that sample bias can be a problem. Bailey suggests: “A homosexual twin who sees an advertisement for a [twin] study may be less likely to call if his twin is heterosexual, [and] this would cause concordance-dependent bias” (Bailey & Dawood, 1998, p. 10).

The problem with sampling bias has been remedied by more recent research that uses national twin registries like the one in Australia. For instance, Bailey, Dunne, and Martin (2000) used this registry of 25,000 twin-pairs and found only a 14 percent probandwise concordance for MZ twins and 11 percent probandwise concordance for DZ twins. This means that if one twin is homosexual, there is only a one in eight chance the brother will be homosexual. This rate is a far cry from the 40 percent to 50 percent concordance rates found in earlier studies using biased samples (also see Hershberger, 1997). Bearman and Bruckner (2002) used a large national sample of American adolescent twins and found only a 7.5 percent concordance rate for MZ twins and a 5.3 percent rate for DZ. Given error estimates of plus or minus 20 percent, such findings suggest that any similarity of sexual orientation between siblings is probably just a chance occurrence.

Another limitation is that “twin studies also tend to eliminate the effect of family life and upbringing” on sexual preference (Whitehead & Whitehead, 2008, p. 15). Yet “there is abundant evidence that the environments of MZ twins are much more similar than those of DZs” (Lewontin et al., 1984, p. 214). Identical twins are often dressed

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alike and play together more than fraternal brothers; they are also treated more alike by teachers and peers and are therefore more likely to share the same environment. Thus, MZ twins are more alike not just because they share genes, but because they also share an environment. If pure genetics were the dominant factor in homosexuality, then the pairwise concordance rate for MZ twins should be close to 100 percent, but it is not. For this reason, these more recent twin studies indicate that the genetic influence is very weak.

The third type of study is known as gene linkage research. The name *gene linkage* comes from the fact that some genes are so close to each other on a chromosome that during meiosis (cell division) they are passed on together to the offspring. Such close-proximity genes are considered linked. If one of the genes has previously been identified, it is called the marker gene (MG); the other gene is called the trait gene (TG) or the candidate gene. Because the MG is linked to the TG and is passed on from parents to children, the MG can then be used to track the inheritance of a TG.

Dean Hamer and his colleagues (1993) noticed that some homosexual men had more homosexual uncles and homosexual cousins on the mother's side of the family than on the father's side. No homosexuals were found among the fathers of the homosexual men or among their paternal relatives. This result would be expected if the TG (e.g., for homosexuality) was carried on the X chromosome inherited from mothers. This phenomenon is known as pedigree analysis.

Hamer et al. (1993) then selected a subgroup of thirty-eight families in which each family had two homosexual brothers and some homosexuals on the maternal side but none on the paternal side. Blood samples were taken from all the homosexual brothers and a gene linkage analysis was done. Hamer's data indicated "a statistically significant correlation between the inheritance of genetic markers on the chromosomal region called Xq28 and sexual orientation" (p. 321). The results, however, were not strong. Brothers will normally have 50 percent of their genes in common, but in Hamer's study there was a 64.5 percent chance that the homosexual brothers would be similar in the Xq28 region of the X

chromosome. With this rather weak result, Hamer et al. claimed that at least one subtype of homosexuality was inherited from the mother and linked to the X chromosome.

Hamer et al.'s (1993) findings, however, have been criticized by several authors (see Baron, 1993). First, if homosexuality were a simple Mendelian trait (like eye color), then Hamer et al. should have found a higher incidence of homosexuality among brothers (it was only 13.5 percent). Second, there is no evidence that the Xq28 section of the chromosome is directly related to sexual behavior. The Xq28 region of the X chromosome may be related to some other yet unidentified trait common in the sample families. Rice, Anderson, Risch, and Ebers (1999) did a similar study with a larger sample (N = 52) and found no support for an X-linked gene underlying male homosexuality. Hamer et al.'s research methodology has been criticized by Risch, Squires-Wheeler, and Keen (1993) and, despite attempts, the results of Hamer et al.'s study have yet to be replicated (Dawood et al., 2009).

Another limitation of linkage studies is the absence of detailed socialization information, such as the family's sexual values and social and political ideology; the amount of exposure to erotic and pornographic media; the occurrence of mental illness; the incidence of abuse, incest, or neglect; and a detailed examination of the emotional health of the parent-child relationship. Unless environmental factors have been adequately measured, one cannot rule out family upbringing as a contributor to homosexuality. Research continues to fail to show that genetics is either a necessary or sufficient cause of homosexual behavior; rather, such behavior, like other complex human behavior, appears to result from nature-nurture interaction.

Hormones and Homosexuality

The effects of hormones on sexual orientation have also been studied to assess the possibility of genetic determinism (Odent, 2005). Hormones (from the Greek "to spur on") are powerful chemicals produced by endocrine glands—the hypothalamus, pituitary, thyroid,

ovaries, and testes—that circulate freely in the bloodstream and affect a wide range of cell structure and functioning (Johnson, 2007). The prenatal hormone hypothesis suggests that if a fetus is exposed to too many or too few sex hormones during prenatal (or even perinatal) development, this exposure will affect not only the internal and external genitalia, but also the brain—which may in turn influence gender identity and sexual orientation (Byne, 2007; LeVay, 1991). The theory suggests that a female fetus exposed to too much endogenous or exogenous androgens will have a higher chance of becoming lesbian, and a male fetus exposed to too little androgens will more likely self-report as homosexual than those with normal hormones levels (Veridiano, Vann, & Neuwalder, 1995).

The prenatal hormone hypothesis is impossible to test directly because hormone experimentation with humans is both unethical and illegal. Researchers can only take advantage of “natural experiments” in which pregnant women were inadvertently exposed to sex hormones or in which children were born with endocrine disorders, such as congenital adrenal hyperplasia (CAH) or androgen insensitivity syndrome (see Kaplan & Owett, 1993; Meyer-Bahlburg et al., 1995). In an example of research involving such children, Berenbaum and Snyder (1995) examined playmate preferences of twenty-four girls and nineteen boys with CAH. Girls with CAH preferred boys’ toys and activities, but boys with CAH did not differ from the controls. While CAH may have some influence on sexual orientation, the possible mechanism for this effect is too complex to disentangle. Meyer-Bahlburg (1979) studied the hormone levels of lesbian and transsexual women; most had normal female hormone levels, but a third had slightly elevated androgen levels. The author concluded that prenatal or post-pubertal hormone levels “do not determine sexual orientation,” but a “neuroendocrine predisposition cannot be ruled out” (p. 59).

However, there is ample evidence in animal studies (most often using mice) where androgen treatment of female fetuses in utero will produce male-type behavior in females, and that removal of normal fetal androgen secretion in male fetuses will produce female-type behavior (Habr-Alencar, Dias, Teodorov, & Bernardi, 2006). Birke (1981) has

questioned the validity of manipulating hormones in animal studies and then applying such findings to humans. He has concluded that there is insufficient support for the hypothesis that homosexuality is caused by endocrine abnormalities. Endocrinologist Louis Gooren (2006) concluded, “The mechanism of sexual differentiation in laboratory animals is clearly orchestrated by gonadal steroids; in humans the mechanism of brain sexual dimorphism [is] not yet certain . . . [and] we are far away from any comprehensive understanding of hormonal imprinting on [human] gender identity formation” (pp. 589, 593).

Various studies that have attempted to test the prenatal hormone hypothesis have proven inconclusive (Gooren, 2006). Banks and Gartrell (1995) concluded that “studies of testosterone levels have not shown a deficiency in male homosexuals or an excess in lesbians” (p. 263). Others disagree and interpret existing research as supportive of the prenatal hormone theory (see Rahman & Wilson, 2003; Wilson & Rahman, 2005).

To summarize this section, twin studies, adoption research, and linkage studies are inconclusive in demonstrating a direct deterministic link between a gene or genes and human homosexual behavior. The evidence suggests that while there may be some type of hereditary influence on homosexuality, the nature or degree of such influence is not known. Studies of hormone influence on sexual orientation are more suggestive of a biological—which is not the same as a genetic—link, but even these findings, as a whole, appear unconvincing at present (Byne, 2007; Gooren, 2006).

The American Psychological Association (APA) issued a new statement on the etiology of homosexuality in 2008. In a 1998 announcement, the APA had stated: “There is considerable evidence to suggest that biology, including genetic or inborn hormonal factors, play a significant role in a person’s sexuality” (see Byrd, 2009). But in 2008, APA’s pronouncement read:

There is no consensus among scientists about the exact reasons that an individual develops a heterosexual, bisexual, gay, or lesbian orientation. Although much

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research has examined the possible genetic, hormonal, developmental, social, and cultural influences on sexual orientation, no findings have emerged that permit scientists to conclude that sexual orientation is determined by any particular factor or factors. Many think that nature and nurture both play complex roles. (APA, 2008, p. 2)

Thus, even the APA has backed off their earlier claim of direct genetic or hormonal causation of homosexual behavior.

Scientific Proof: Is It Possible?

All of this leads to a critical question: *If homosexuality were directly genetically or hormonally caused, how could science show this?*

To prove that genes cause homosexuality, scientists would first have to isolate candidate genes and then determine what proteins these genes manufacture. The action of these proteins on brain tissue, brain chemistry, or on some part of the endocrine system would then have to be established. Finally, if differences in brain or endocrine chemistry are consistently found between homosexuals and heterosexuals, then the potency (or strength) of those changes to predict homosexuality would need to be determined.

Two genetic concepts help explain gene potency: penetrance and expressivity. Gene penetrance is the probability that a gene will be expressed in a recognizable phenotype in the population. In other words, penetrance refers to how often a trait is expressed in people who have the gene for that trait. “Complete” penetrance means that everyone who has the gene will show the trait or behavior. “Incomplete” penetrance means that only some people who have the gene will show the trait or behavior. Gene expressivity is how much of a trait will be expressed in a particular person—whether the person is greatly, moderately, or only mildly affected by the gene. Expressivity means a trait may appear very pronounced, barely noticeable, or somewhere in between.

If genes for homosexuality were ever identified, these genes would probably demonstrate incomplete penetrance and mild expressivity. This means that some individuals who carry the suspected homosexual alleles would not become homosexual; others would show only minor to moderate symptoms of homosexual thoughts, feelings, and behaviors. In either case, the influence of environmental events and self-determination would also be needed to explain the development and expression of homosexuality.

Dr. Richard M. Lerner (2004) of Tufts University champions the inclusion of self-determination (agency and choice) as part of our understanding of any complex human behavior. “Humans are neither passive recipients of genes that compel their actions nor passive recipients of [environmental] stimuli that impel their behavior. Humans are active, acting, goal oriented effective shapers of the complex ecology of human development” (p. 64). There is no logical or empirical reason why the development of homosexual thoughts, feelings, or behaviors would be exceptions to Lerner’s observation.

Part 4: The Threshold Model of Homosexuality

Another way that genetics has been hypothesized to play a role in homosexuality is called the *threshold model of homosexuality*. In general, the expression of a trait or condition requires a certain number of genetic influences and a certain number of environmental events to push the individual “over the threshold” from a common or typical to an uncommon or atypical physical or mental state. This situation has been described as an “accumulation of genetic and environmental liabilities” (DiLalla, 2004, p. 10; Gottesman & Goldsmith, 1994). In other words, some genes may be “susceptibility genes,” which increase the chance of expressing the condition but which by themselves are not sufficient to produce the condition without some environmental trigger (Pericak-Vance, 2003).

Likewise, some environmental factors, like smoking, may put the individual at risk for developing a disease such as lung cancer but may not be enough to cause the sickness in a genetically robust person. For example, for a person to get lung cancer, he

or she may need a genetic vulnerability to cancer as well as environmental irritants such as smoking, secondhand smoke, severe air pollution, and/or high life stress.

Similarly, homosexuality may result from an accumulation of both genetic and environmental risks (Satinover, 1996). Suppose that at some time in the future scientists identify thirteen genes common among those who self-identify as homosexual. These may be genes related to emotional sensitivity, lack of physical prowess, artistic creativity, unconventional thinking, and late onset of puberty. We would then assume that these traits or behaviors appear as factors that likely increase the chances that a child may become homosexual (Satinover, 1996).

In addition, assume that there are some environmental factors that may increase the likelihood of a child becoming homosexual. For example, in a national cohort study of two million Danes, homosexual marriage was more likely in men with divorced parents or otherwise absent fathers (Frisch & Hviid, 2006). Several studies have shown that both boys and girls who have experienced sexual abuse or incest are more likely to become homosexual (Bradford, Ryan, & Rothblum, 1994; Paul, Catania, Pollack, & Stall, 2001; Tomeo, Templer, Anderson, & Kotler, 2001; Zucker & Bradley, 1995). Some research suggests that prolonged exposure to pornography may increase homosexual experimentation in some men (Morrison, Morrison, & Bradley, 2007; Parsons, Kelly, Bimbi, Muench, & Morgenstern, 2007). Research on the effect of media on behavior, however, is correlational, and direction of effect is ambiguous: does pornography increase sexual experimentation, or does sexual experimentation lead to use of pornography? But for illustrative purposes, father absence, sexual abuse, and pornography—singularly or in combination, may increase the likelihood of a boy's experiencing homosexual feelings or expressing homosexual behaviors. Other environmental traumas also may increase the chances of a homosexual outcome (Broman, 2003).

Now consider some fictional examples. Continuing the illustration above, imagine that Teen A (a male) has seven of the thirteen genes hypothetically found to be related to

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homosexuality, and that he has experienced three of seven possible environmental factors associated with homosexuality (father absence, sexual abuse, and pornography). His total “risk” for homosexuality could be computed as $7 + 3 = 10$. This total may be just enough to nudge Teen A over the threshold of a normative heterosexual orientation to a homosexual orientation.

Another person, Teen B (a female), has five genes correlated to homosexual behavior but has only experienced one environmental event, childhood sexual abuse. Her total risk would be computed as $5 + 1 = 6$. In the case of Teen B, the accumulative risk of 6 may not be large enough to push her over the threshold from heterosexuality to homosexuality. This scenario is oversimplified and assumes that genes correlated with various elements of homosexual behavior will be found. However, it provides an illustration of how a threshold model might apply to a complex psycho-social-biological behavior like homosexuality.

In essence, the threshold theory of homosexuality is similar to Daryl Bem’s (1996) interactional theory of homosexuality, a theory that combines the indirect effects of genetics with powerful environmental events. Bem hypothesized that genetic factors do not directly cause sexual orientation but do influence a child’s temperament and activity level, which in turn influence the child’s preferred friends, activities, and emotional responses. Such children may exhibit gender-nonconforming behaviors and may find themselves more comfortable with opposite-sex playmates. Yet they also may have a craving and longing for acceptance from and companionship with same-sex friends (Stein, 1999). Over time, same-sex peers are seen first as “exotic” and then as “erotic.” Eventually, such youths may develop a sexual attraction to same-sex peers.

Conclusions

Except for rare physical abnormalities such as Huntington’s disease, there is no evidence of a direct causative link between a single gene and complex psychosocial

behaviors such as homosexuality (Bazzett, 2008). This conclusion is supported by geneticists, molecular biologists, neuroscientists, and behavioral psychologists (Plomin, McClearn, McGuffin, & Defries, 2000). Behavioral psychologist Catherine Baker (2004) explained:

Many people think that a gene controls a behavioral trait. This is genetic determinism, the belief that the development of an organism is determined solely by genetic factors. *Genetic determinism is a false belief.* It comes from misunderstandings of scientific research.The fact is that so far, scientific research has not confirmed any one-to-one correspondence between a gene and a [complex] human behavior. Behavior results from the activity of multiple genes amidst the influence of multiple environmental factors. (pp. 17–18; my emphasis)

Many people take a simplistic view of behavioral genetics: they believe that one gene controls or determines one specific behavior. This false belief has led many people to think that there is an alcoholism gene, a manic-depression gene, an obesity gene, and a homosexuality gene. Such is not the case. Hubbard and Wald (1999) explained, “It is an oversimplification to say that any gene is ‘the gene for a trait.’ Each gene simply specifies one of the proteins involved in the complex process [of gene-environment interaction]” (p. 44). Valenstein (1998) neatly summarizes this idea:

Most recent claims that a gene has been discovered that causes alcoholism, schizophrenia, [or] homosexuality . . . have proven illusory Genes do not produce behavioral or mental states. Genes carry the instructions and templates for producing and assembling amino acids and proteins into anatomical structures. Behavior and mental traits, however, are the product of an interaction between anatomical structure and experience. Even where there is compelling evidence

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that some behavioral or mental trait is influenced by genetic factors it is almost always a predisposition, not a certainty A predisposition is not a cause. (pp. 140–141, 224)

At this time, the complex psycho-social-biological condition of homosexuality cannot be directly traced to the activity of a single gene or even to a group of genes (Parens, Chapman, & Press, 2006; Rutter, 2006). Geneticist Robert Plomin comments: “Genes do not act as master puppeteers within us. They are chemical structures that control the production of proteins, thereby indirectly affecting behavior..... Genes do not determine one’s destiny” (Plomin et al., 1980, p. 13).

Baker (2004) makes the incontrovertible statement that “behavior results from the activity of multiple genes amidst the influence of multiple environmental factors” (p. 18). There is no direct path from a gene to a behavior; environment always intervenes (Turkheimer, 2002). And lest it be overlooked, human agency—free will and choice—plays a significant role in expression of complex psychosocial behaviors (Abbott & Bryd, 2009).

At present there is insufficient evidence to support the hypothesis that homosexuality is exclusively or primarily genetically determined. As F. S. Collins (2006), head of the Human Genome Project, states:

There is an inescapable component of heritability to many human behavioral traits. For virtually none of them is heredity ever close to predictive. An area of particularly strong public interest is the genetic basis of homosexuality. Evidence [indicates] that sexual orientation is genetically influenced but not hardwired by DNA, and that whatever genes are involved represent predispositions, not predeterminations. (p. 281)

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Homosexuality is a very complex behavior that results from the dynamic interaction of multiple biological and environment influences that change over time (Diamond, 2008). Thus, any explanation of its etiology must involve the intricate interaction between genetic influences, environmental events, and self-determination or free will (cf., Allen, 2007; Garcia, Lerner, & Bearer, 2003; Pelle, 1995). Psychologist David Moshman (2005) concluded: “There is evidence that hereditary variations influence sexual orientation, but no evidence that any gene or set of genes causes a person to be homosexual” (p. 108). Edward Stein, author of *The Mismeasure of Desire: The Science, Theory, and Ethics of Sexual Orientation* and a pro-gay activist, concluded:

Genes in themselves cannot directly specify any behavior or psychological phenomenon. Instead, genes direct a particular pattern of RNA synthesis, which in turn may influence the development of psychological dispositions and the expression of behaviors. There are many intervening pathways between a gene and a disposition or a behavior, and even more intervening variables between a gene and a pattern [of behavior] that involves both thinking and behaving. The terms “gay gene” and “homosexual gene” are, therefore without meaning. . . . No one has.....presented evidence in support of such a simple and direct link between genes and sexual orientation. (1999, p. 221)

To this author, the scientific evidence is clear and unequivocal: *Homosexual behavior is not directly caused by genetic processes*. No matter how strongly some people want to believe that homosexuality is genetically determined, science fails to support this belief. Those who continue to push and prod science to discover a genetic explanation of homosexual behavior fail to recognize that in almost all instances of complex psychosocial behavior, *DNA is not destiny* (Barr, 2003; Church, 2009; Cloud, 2010; Garcia et al., 2003; Shenk, 2010).

Footnotes

¹ Some additional definitions may be required to understand twin studies. A *proband* is the person within a specific family who has a preselected trait or condition of interest (such as autism, schizophrenia, or homosexuality). Concordance is the probability that the second twin will have the same trait or condition as the first twin. In concordant twins, both have the trait while in discordant pairs only one twin has the trait. Suppose you have twenty preselected identical twin pairs where one twin self-identifies as homosexual, and in ten pairs the second twin self-identifies as heterosexual. The pairwise concordance rate is equal to the number of concordant pairs (ten, designated as C) divided by the number of concordant pairs (designated as D) plus the number of discordant pairs (10 / 10 + 10), so the pairwise rate is $C/(C + D)$ or $10/20$, which equals 50 percent. Probandwise concordance rate is different; it estimates an individual's probability of having a specific trait or condition if the person's twin has the trait or condition. It is preferred by most geneticists as a more accurate estimate of genetic influence on the trait and is calculated differently as $2C/(2C + D)$. In this case it would be $2 \times 10 / (2 \times 10 + 10)$, which equals 66 percent.

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