The trumpets were left at home and the parades were canceled. The press releases and campaign signs were quietly forgotten. The news was big, but it did not contain what some had hoped for. On April 14, 2003, the International Human Genome Consortium announced the successful completion of the Human Genome Project—two years ahead of schedule. The press report read: “The human genome is complete and the Human Genome Project is over” (see “Human Genome Report…,” 2003, emp. added). Most of the major science journals reported on the progress in the field of genetics, but also speculated on how the information would now be used. The one piece of information that never materialized from the Human Genome Project was the identification of the so-called “gay gene.”

Homosexuality has been practiced for thousands of years. Simply put, homosexuality is defined as sexual relations between like genders (i.e., two males or two females). It was Sigmund Freud who first postulated that parental relationships with a child ultimately determine the youngster’s sexual orientation. But this “nurturing” aspect has effectively given way to the “nature” side of the equation. Can some behaviors (e.g., alcoholism, homosexuality, schizophrenia) be explained by genetics? Are these and other behaviors influenced by nature or by nurture? Are they inborn or learned? Some individuals believed that the answer would be found hiding amidst the chromosomes analyzed in the Human Genome Project.

The human X and Y chromosomes (the two “sex” chromosomes) have been completely sequenced. Thanks to work carried out by labs all across the globe, we know that the X chromosome contains 153 million base pairs, and harbors a total of 1168 genes (see NCBI, 2004). The National Center for Biotechnology Information reports that the Y chromosome—which is much smaller—contains “only” 50 million base pairs, and is estimated to contain a mere 251 genes. Educational institutions such as Baylor University, the Max Planck Institute, the Sanger Institute, Washington University in St. Louis, and others have spent countless hours and millions of research dollars analyzing these unique chromosomes. As the data began to pour in, they allowed scientists to construct gene maps—using actual sequences from the Human Genome Project. And yet, neither the map for the X nor the Y chromosome contains any “gay gene.”

What is the truth regarding homosexuality? Too often, speculation, emotions, and politics play a major role in its assessment. The following is a scientific investigation of human homosexuality.
The argument goes like this: “Just as a person cannot help being black, female, or Asian, I cannot help being homosexual. We were all born this way, and as such we should be treated equally.” However, this argument fails to comprehend the true “civil rights” movements. The law already protects the civil rights of everyone—black, white, male, female, homosexual, or heterosexual. Homosexuals enjoy the same civil rights everyone else does. The contention arises when specific laws deprive all citizens of certain behaviors (e.g., sodomy, etc.). We should keep in mind that these laws are the same for all members of society. Because of certain deprivations, homosexuals feel as though “equal” rights have been taken away (i.e., marriage, tax breaks, etc.).

Skin color and other genetic traits can be traced through inheritance patterns and simple Mendelian genetics. Homosexuals are identified not by a trait or a gene, but rather by their actions. Without the action, they would be indistinguishable from all other people. It is only when they alter their behavior that they become a group that is recognized as being different. If we were to assume momentarily that homosexuality was genetic, then the most one could conclude is that those individuals were not morally responsible for being homosexual. However, that does not mean that they are not morally responsible for homosexual actions! Merely having the gene would not force one to carry out the behavior. For instance, if scientists were able to document that a “rape gene” existed, we certainly would not blame an individual for possessing this gene, but neither would we allow him to act upon that rape disposition. Neil Risch and his coworkers admitted:

There is little disagreement that male homosexual orientation is not a Mendelian trait. In fact, a priori, one would expect the role of a major gene in male homosexual orientation to be limited because of the strong selective pressures against such a gene. It is unlikely that a major gene underlying such a common trait could persist over time without an extraordinary counterbalancing mechanism (1993, 262-2064).

Evan S. Balaban, a neurobiologist at the Neurosciences Institute in San Diego, noted that

the search for the biological underpinnings of complex human traits has a sorry history of late. In recent years, researchers and the media have proclaimed the “discovery” of genes linked to alcoholism and mental illness as well as to homosexuality. None of the claims...have been confirmed (as quoted in Horgan, 1995).

Charles Mann agreed, stating: “Time and time again, scientists have claimed that particular genes or chromosomal regions are associated with behavioral traits, only to withdraw their findings when they were not replicated” (1994, 264:1687). It appears that the gay gene will be added to this category of unreplicated claims.

The real issue here is homosexual actions that society has deemed immoral and, in many instances, illegal. Since no study has firmly established an underlying genetic cause for homosexuality, arguments suggesting “equal rights” are both baseless and illogical.

**REAL STATISTICS**

Anyone who has tuned into prime-time television within the past few years has observed an increasing trend of shows featuring characters who are homosexual—and proud of it. It seems as though modern sitcoms require “token” homosexuals in order to be politically correct. The perception is that these individuals share the same apartment buildings, offices, clubs, etc., with heterosexual people, and that we need to realize just how prevalent homosexuality is. So, exactly what fraction of the population do homosexuals actually represent?

The famous Kinsey Institute report often is cited as evidence that 10% of the population is homosexual. In his book, *Is It a Choice?: Answers to 300 of the Most Frequently Asked Questions About Gays and Lesbians*, Eric Marcus used the Kinsey studies to demonstrate that one in ten people is homosexual (1993). In truth, Kinsey never reported figures that high. The Kinsey Report clearly stated that: “Only about 4 percent of the men [evaluated] were exclusively homosexual throughout their entire lives... Only 2 or 3 percent of these women were exclusively homosexual their entire lives” (see Reinisch and Beasley, 1990, p. 140). However, there is good reason to believe that the real percentage is not even this high.

While no one has carried out a door-to-door census, we do have a fairly accurate estimate. Interestingly, these statistics came to light in an *amicus curiae* (“friend of the court”) brief filed with the U.S. Supreme Court on March 26, 2003, in the Lawrence vs. Texas case (commonly known as the Texas sodomy case). On page 16 of this legal brief, footnote 42 revealed that 31 homosexual and pro-homosexual groups admitted the following:

The most widely accepted study of sexual practices in the United States is the National Health and Social Life Survey (NHLS). The NHLS found that 2.8% of the male, and 1.4% of the female population identify themselves as gay, lesbian, or bisexual (Laumann, et al., 1994).

The study also found that only 0.9% of men and 0.4% of women reported having only same-sex partners since age 18—a figure that would represent a total of only 1.4 million Americans as homosexual (based on the last census report, showing roughly 292 million people living in America). The resulting accurate figures demonstrate that significantly less than one percent of the American population claims to be homosexual. The NHLS results are similar to a survey conducted by the Minnesota Ad-
The landmark Kinsey report was produced using the sexual histories of thousands of Americans. While that report consisted of a diverse sample, it was not a representative sample of the general population (Kinsey, et al., 1948, 1953). In 1994, Richard Friedman and Jennifer Downey published a review on homosexuality in The New England Journal of Medicine. In reviewing Kinsey's work, they noted:

Kinsey reported that 8 percent of men and 4 percent of women were exclusively homosexual for a period of at least three years during adulthood. Four percent of men and 2 percent of women were exclusively homosexual after adolescence (1994, 331:923).

With this "statistical information" in hand, some sought to change the way homosexuality was viewed by both the public and the medical community. Prior to 1973, homosexuality appeared in the Diagnostic and Statistical Manual of Mental Disorders (DSM), the official reference book used by the American Psychiatric Association for diagnosing mental disorders in America and throughout much of the rest of the world. Homosexuality was considered a sickness that doctors routinely treated. In 1973, however, it was removed as a sexual disorder, based on the claim that it did not fulfill the "distress and social disadaptability" criteria that were used to define a disorder. Today, there is no mention of homosexuality in the DSM-IV (aside from a section describing gender identity disorder), indicating that individuals with this condition are not suitable candidates for therapy (see American Psychiatric Association, 2000).

Physicians treating patients for homosexuality (to bring about a change in sexual orientation) frequently are reported to ethics committees in an attempt to have them cease. Robert Spitzer lamented:

Several authors have argued that clinicians who attempt to help their clients change their homosexual orientation are violating professional ethical codes by providing a "treatment" that is ineffective, often harmful, and reinforces in their clients the false belief that homosexuality is a disorder and needs treatment (2003, 32:403). Thus, the stage was set for the appearance of a "gay gene."

**SIMON LEVAY—BRAIN DIFFERENCES**

The first "significant" published study that indicated a possible biological role for homosexuality came from Simon LeVay, who was then at the Salk Institute for Biological Studies in San Diego, California. In 1991, Dr. LeVay reported subtle differences between the brains of homosexual and heterosexual men (1991). LeVay measured a particular region of the brain (the interstitial nuclei of the anterior hypothalamus—INAH) in postmortem tissue of three distinct groups: (1) men; (2) women who were presumed to be heterosexual; (3) and homosexual men.

LeVay’s Reported Findings

LeVay reported that clusters of these neurons (INAH) in homosexual men were the same size as clusters in women, both of which were significantly smaller than clusters in heterosexual men. LeVay reported that the nuclei in INAH 3 were “more than twice as large in the heterosexual men as in the women. It was also, however, more than twice as large in the heterosexual men as in the homosexual men” (1991, 253:1034). This difference was interpreted as strong evidence of a biological link to homosexuality. LeVay’s assumption was that homosexual urges can be biologically based—so long as cluster size is accepted as being genetically determined.

**Problems with LeVay’s Study**

When looking at the methodology of the LeVay study, one of the key problems is that the study has never been reproduced. As William Byrne noted, LeVay’s work has not been replicated, and human neuroanatomical studies of this kind have a very poor track record for reproducibility. Indeed, procedures similar to those LeVay used to identify nuclei have previously led researchers astray (1994, 270[5]:53, emp. added).

Additionally, of nineteen homosexual subjects used in the study, all had died of complications of acquired immunodeficiency syndrome (AIDS). AIDS has been shown to decrease testosterone levels, so it should be expected that those who suffered from that condition would have smaller INAH. Byrne continued his comments on LeVay’s work. His inclusion of a few brains from heterosexual men with AIDS did not adequately address the fact that at the time of death, virtually all men with
AIDS have decreased testosterone levels as the result of the disease itself or the side effects of particular treatments. To date, LeVay has examined the brain of only one gay man who did not die of AIDS (270:53).

Furthermore, in a scientific environment where controls and standards are a necessity, LeVay did not possess a complete medical history of the individuals included in his study. He therefore was forced to assume the sexual orientation of the non-AIDS victims as being heterosexual, when some may not have been. In addition, bear in mind that he had no evidence regarding the sexual orientation of the women whose brains he examined. LeVay has admitted:

“It’s important to stress what I didn’t find. I did not prove that homosexuality is genetic, or find a genetic cause for being gay. I didn’t show that gay men are born that way, the most common mistake people make in interpreting my work. Nor did I locate a gay center in the brain (as quoted in Byrd, et al., 2001, empl. added).

Many have argued that what LeVay discovered in the brains of those he examined was only a result of prior behavior, not the cause of it. Mark Breedlove, a researcher at the University of California at Berkeley, has demonstrated that sexual behavior has an effect on the brain. In referring to his own research, Breedlove commented: “These findings give us proof for what we theoretically know to be the case—that sexual experience can alter the structure of the brain, just as genes can alter it. [...] It is possible that differences in sexual behavior cause (rather than are caused by) differences in the brain” (as quoted in Byrd, et al., parenthetical item in orig.). Considering this type of research, it makes sense that a homosexual lifestyle (and/or the AIDS condition) could alter the size of the nuclei LeVay was measuring.

What exactly did LeVay find? In actuality, not much. He did observe slight differences between the groups—if you accept the method he used for measuring the size of the neuron clusters (and some researchers do not). When each individual was considered by himself, there was not a significant difference; only when the individuals involved in the study were considered in groups of homosexuals vs. heterosexuals did differences result. Hubbard and Wald commented on this lack of difference:

“Though, on average, the size of the hypothalamic nucleus LeVay considered significant was indeed smaller in the men he identified as homosexual, his published data show that the range of sizes of the individual samples was virtually the same as for the heterosexual men. That is, the area was larger in some of the homosexuals than in many of the heterosexual men, and smaller in some of the heterosexual men than in many of the homosexuals. This means that, though the groups showed some difference as groups, there was no way to tell anything about an individual’s sexual orientation by looking at his hypothalamus (1997, pp. 95-96, empl. added).

Being homosexual himself, it is no surprise that LeVay observed: “...[P]eople who think that gays and lesbians are born that way are more likely to support gay rights.” In a Newsweek article, LeVay was quoted as saying, “I felt if I didn’t find any [difference in the hypothalami], I would give up a scientific career altogether” (as quoted in Gelman, et al., 1992, p. 49). Given how (poorly) twisted LeVay’s data are, and his own personal bias, his abandonment of science may have ultimately been of greater service.

Brain Plasticity—A Fact Acknowledged by All Neuroscientists

Today, scientists are keenly aware of the fact that the brain is not as “hard-wired” or permanently fixed as once thought—an important factor that LeVay failed to acknowledge. One of the properties of plasticity—many containers are made out of plastic so that they will not shatter when dropped. In a similar manner, the brain was once considered to be rigid, like Ball® jars used for canning—but we now know the brain is “plastic” and flexible, and able to reorganize itself. Research has shown that the brain is able to remodel its connections and grow larger, according to the specific areas that are most frequently utilized. Given that we know today that the brain exhibits plasticity, one must ask if the act of living a homosexual lifestyle itself might be responsible for the difference LeVay noted? Commenting on brain plasticity, Shepherd noted:

The inability to generate new neurons might imply that the adult nervous system is a static, “hard-wired” machine. This is far from the truth. Although new neurons cannot be generated, each neuron retains the ability to form new processes and new synaptic connections (1994).

Interestingly, since Shepherd’s textbook was published, additional research has even documented the ability of neurons to be generated within certain areas of the brain. This information must be considered when examining comparative anatomical experiments such as LeVay’s. These cortical re-arrangements that occur are not as simple as unplugging a lamp and plugging it into another socket. The changes observed by researchers indicate that if the brain were represented by a home electrical system, then many of the wires within the walls would be pulled out, rewired to different connections in different rooms, new outlets would appear, and some would even carry different voltages. Due to the colossal connectivity that takes place within the brain, any “rewiring” is, by its very nature, going to have an effect on several areas—such as INAH3. Scientists understand these things, yet LeVay’s work is still mentioned as alleged support for the so-called gay gene.
Their Reported Findings

- 52% of identical (monozygotic) twins of homosexual men were homosexual
- 22% of fraternal (dizygotic) twins were likewise homosexual
- 11% of adoptive brothers of homosexual men were homosexual
- 48% of identical twins of homosexual women were likewise homosexual
- 16% of fraternal (dizygotic) twins were likewise homosexual
- 6% of adoptive sisters of homosexual women were likewise homosexual (Bailey and Benishay, 1993, “Familial Aggregation of Female Sexual Orientation”)

Problems with Bailey & Pillard’s Study

While the authors acknowledged some of the flaws with their research, they still were quoted in Science News as saying: “Our research shows that male sexual orientation is substantially genetic” (as quoted in Bower, 1992, 14:16). However, the most glaring observation is that clearly not 100% of the identical twins “inherited” homosexuality. If there was, in fact, a “gay gene,” then all of the identical twins should have reported a homosexual orientation. And yet, in nearly half of the twins studied, one brother was not homosexual. In a technical-comment letter in Science, Neil Risch and colleagues pointed out: “The biological brothers and adoptive brothers showed approximately the same rates. This latter observation suggests that there is no genetic component, but rather an environmental component shared in families” (1993, 262:2063). In fact, more adoptive brothers shared homosexuality than non-twin biological brothers. If there was a genetic factor, this result would be counter to the expected trend. Byne and Parsons noted:

However, the concordance rate for homosexuality in nontwin biologic brothers was only 9.2—significantly lower than that required by simple genetic hypothesis, which, on the basis of shared genetic material, would predict similar concordance rates for DZ [dizygotic] twins and nontwin biologic brothers. Furthermore, the fact that the concordance rates were similar for nontwin biologic brothers (92%) and genetically unrelated adoptive brothers (11.0%) is at odds with a simple genetic hypothesis, which would predict a higher concordance rate for biological siblings (1993, 50:229).

A more recently published twin study failed to find similar concordance rates. King and McDonald studied 46 homosexual men and women who were twins. The concordance rates that they reported were 10%, or 25% with monoyzygotic twins—depending on whether or not the bisexuals were included along with the homosexuals. The rates for dizygotic twins were 8% or 12%, again, depending on whether bisexuals were included (King and McDonald, 1992). Byne and Parsons commented: “These rates are significantly lower than those reported by Bailey and Pillard; in comparison of the MZ [monozygotic] concordance rate, including bisexuals (25%), with the comparable figure from Bailey and Pillard (52%)” (p. 230). They went on to observe: “Furthermore, if the concordance rate is similar for MZ and DZ twins, the importance of genetic factors would be considerably less than that suggested by Bailey and Pillard” (p. 230, emp. added).

Another factor that may have had a drastic affect on the results of this study (and other similar studies) centers on methodology. Bailey and Pillard did not study a random sample of homosexuals. Instead, the subjects were recruited through advertisements placed in homosexual publications. This method can be deemed questionable because it is highly dependent on the readership of those publications and on the motives of those who respond. Thus, it may lead to skewed results—for example, inflated rates of concordance in identical twins owing to preferential participation (see Baron, 1993). Hubbard and Wald observed:

The fact that fraternal twins of gay men were roughly twice as likely to be gay as other biological brothers shows that environmental factors are involved, since fraternal twins are no more similar biologically than are other biological brothers. If being a fraternal twin exerts an environmental influence, it does not seem surprising that this should be even truer for identical twins, who the world thinks of as “the same” and treats accordingly, and who often share those feelings of sameness (1997, p. 97).

In summarizing their findings, Byne and Parsons stated: “Critical review shows the evidence favoring a biologic theory to be lacking” (50:228). Commenting on Bailey and Pillard’s report, researchers Billings and Beckwith wrote:

While the authors interpreted their findings as evidence for a genetic basis for homosexuality, we think that the data in fact provide strong evidence for the influence of the environment (1993, p. 60).

When evaluated scientifically, twin studies fail to provide any valid support for the longed-for “gay gene.”

DEAN HAMER—THE GAY GENE ON THE X CHROMOSOME

Two years after Simon LeVay’s report, a group led by Dean H. Hamer of the National Cancer Institute allegedly linked male homosexuality to a gene on the X chromosome. His team investigated 114 families of homosexual men. Hamer and his colleagues collected family history information from 76 gay male individuals and 40 gay brother pairs as they searched for incidences of homosexuality among relatives of gay men.

In many families, gay men had gay relatives through maternal lines. Thus, they concluded that a gene for homosexuality might be found on the X chromosome, which is passed from the mother alone. They then used DNA linkage analysis in an effort to find a correlation between inheritance and homosexual orientation.

Their Reported Findings

Because many of the families with a prevalence of homosexual relatives had a common set of DNA markers on the X chromosome, Hamer’s group assumed a genetic etiology. Of the 40 pairs of homosexual brothers he analyzed, Hamer found that 33 exhibited a matching DNA region called q28—a gene located at the tip of the long arm of the X chromosome. In summarizing their findings, Hamer and colleagues noted: “Our experiments suggest that a locus (or loci) related to sexual orientation lies within approximately 4 million base pairs of DNA on the tip of the long arm of the X chromosome” (1993, 261:326, parenthetical item in orig.). This discovery prompted Hamer and his colleagues to speculate:

The linkage to markers on Xq28, the subtelomeric region of the long arm of the sex chromosome, had a multipoint lod score of 4.0, indicating a statistical confidence level of more than 99 percent that at least one subtype of male sexual orientation is genetically influenced (261:321, emp. added).
It is important to note that Hamer did not claim to have found a “gay gene,” or even the set of genes, that might contribute to a propensity for homosexuality. According to Chicago Tribune staff writer, John Crewdson, what Hamer claimed to have found was “statistical evidence that such genes exist” (1995).

Problems with Hamer’s Study

One of the most significant problems with Hamer’s approach is that he and his colleagues did not feel that it was necessary to check whether any of the heterosexual men in these families shared the marker in question! Would it not be useful to know whether or not this “gay gene” is found in heterosexuals? Even if only a few of them possess the gene, it calls into question what the gene or the self-identification signifies. Additionally, Hamer never explained why the other seven pairs of brothers did not display the same genetic marker. If this is “the gene” for homosexuality, then one must assume all homosexual individuals would possess that particular marker—and yet that was not the case in Hamer’s study.

In a letter to Science, Anne Fausto-Sterling and Evan Balaban pointed out some of the additional problems with Hamer’s study. They noted:

Despite our praise for aspects of Hamer, et al.’s work, we feel it is also important to recognize some of its weaknesses. The most obvious of these is the lack of an adequate control group. Their study demonstrates cosegregation of a trait (which Hamer, et al. have labeled “homosexuality”) with X chromosome markers and the trait’s concordance in homosexual brothers. This cosegregation is potentially meaningful if the mother is heterozygous for the trait. In this case, segregating chromosomes without the markers should show up in nonhomosexual brothers, but Hamer, et al. present no data to that effect (1993, 261:1257, emp. added).

Fausto-Sterling and Balaban continued:

This sensitivity to assumptions about background levels makes Hamer, et al.’s data less robust than the summary in their abstract indicates.... Finally we wish to emphasize a point with which we are sure Hamer, et al. would agree: correlation does not necessarily indicate causation (261:1257).

In other words, Hamer’s methodology leaves something to be desired. One also should keep in mind that Hamer’s sampling was not random, and, as a result, his data may not reflect the real population.

George Rice and his colleagues from Canada looked intently at the gene Xq28. They then observed: “Allele and haplotype sharing for these markers was not increased over expectation. These results do not support an X-linked gene underlying male homosexuality” (1999, 284:665, emp. added). Rice, et al., included 182 families in their study. They noted:

It is unclear why our results are so discrepant from Hamer’s original study. Because our study was larger than that of Hamer et al., we certainly had adequate power to detect a genetic effect as large as was reported in that study. Nonetheless, our data do not support the presence of a gene of large effect influencing sexual orientation at position Xq28 (284:667).

That is a tactful way of saying that any claims of having found a “gay gene” were overblown, if not outright false, and that Hamer’s results are dubious at best. Commenting on the study of Rice and his colleagues, Ingrid Wickelgren remarked: “…the Ontario team found that gay brothers were no more likely to share the Xq28 markers than would be expected by chance.... Ebers interprets all these results to mean that the X linkage is all but dead” (1999, 284:571, emp. added).

In June of 1998, University of Chicago psychiatrist Alan Sanders reported at the meeting of the American Psychiatric Association that he, too, had been unable to verify Hamer’s results. Looking for an increase in Xq28 linkage, Sanders’ team studied 54 pairs of gay brothers. As Wickelgren indicated, Sanders’ team had found “only a weak hint—that wasn’t statistically significant—of an Xq28 linkage among 54 gay brother pairs” (284:571). Commenting on the validity of Hamer’s study, Wickelgren quoted George Rice: “Taken together, Rice says, the results ‘suggest that if there is a linkage it’s so weak it’s not important’ ” (1999, emp. added). Two independent labs failed to reproduce anything even remotely resembling Hamer’s results.

CHANGEABILITY OF HOMOSEXUALS—EVIDENCE AGAINST GENETICS

An individual born with diabetes has no hope of changing that condition. Likewise, a child born with Down’s syndrome will carry that chromosomal abnormality throughout his or her life. These individuals are a product of the genes they inherited from their parents. Homosexuality appears to be vastly different. Many people have been able to successfully change their sexual orientation. [Truth be told, some individuals experiment with a variety of sexual partners—male/female—often, going back and forth. One might inquire if the bisexuality denotes the existence of a “bisexual gene?”] Ironically, however, the removal of homosexuality as a designation from the Diagnostic and Statistical Manual of Psychiatric Disorders by the American Psychiatric Association has kept many physicians from attempting to provide reparative therapy to homosexuals.

Robert Spitzer conducted a study on 200 self-selected individuals (143 males, 57 females) in an effort to see if participants could change their sexual orientation from homosexual to heterosexual (2003, 32:403-417). He reported some minimal change from homosexual to heterosexual orientation that lasted at least five years (p. 403). Spitzer observed:

The majority of participants gave reports of change from a predominantly or exclusively homosexual orientation before therapy to a predominantly or exclusively heterosexual orientation in the past year (p. 403).

In summarizing his findings, Spitzer declared: “Thus, there is evidence that change in sexual orientation following some form of reparative therapy does occur in some gay men and lesbians.” He thus concluded: “This study provides evidence that some gay men and lesbians are able to also change the core features of sexual orientation” (p. 415).

Six years earlier, the National Association for Research and Therapy of Homosexuality (NARTH) released the results of a two-year study stating:

Before treatment, 68 percent of the respondents perceived themselves as exclusively or almost entirely homosexual, with another 22 percent stating that they were more homosexual than heterosexual. After treatment, only 13 percent perceived themselves as exclusively or almost entirely homosexual, while 33 percent described themselves as either exclusively or almost entirely heterosexual (see Nicolosi, 2000, 86:1071).

The study also reported:

Although 83 percent of respondents indicated that they entered therapy primarily because of homosexuality, 99 percent of those who participated in the survey said they now believe treatment to change homosexuality can be effective and valuable (p. 1071).

These data are consistent with the ongoing research project of Rob Goetz, who has identified 84 articles or books that contain some relevance to the possibility of sexual orientation change (2004). Of the data reported, 31 of the 84 studies showed a quantitative outcome of individuals able
to change sexual orientation. These are not studies that merely speculate on the ability to change; they actually have the numbers to back it up! All of these data on the heels of warnings from the Surgeon General, The American Academy of Pediatrics, and all of the major mental health associations, which have issued position statements warning of possible harm from such therapy, and have asserted that there is no evidence that such therapy can change a person’s sexual orientation. For instance, the 1998 American Psychiatric Association Position Statement on Psychiatric Treatment and Sexual Orientation noted:

...there is no published scientific evidence supporting the efficacy of reparative therapy as a treatment to change one’s sexual orientation. The potential risks of reparative therapy are great, including depression, anxiety, and self-destructive behavior (see American Psychiatric Association, 1999, p. 1131).

Thus, physicians are caught in a quandary of a double standard. On the one hand, they are told that it is “unethical” for a clinician to provide reparative therapy because homosexuality is not a diagnosable disorder, and thus one should not seek to change. Yet, they contend that not enough studies have been conducted to determine the effectiveness of reparative therapy. The message is loud and clear. “Do not do this because it is unethical to ask a homosexual person to change. However, truth be told, we have not collected enough data to know if a person can safely change his or her sexual orientation.”

In situations where sexual orientation is being measured, studies face serious methodological problems (i.e., follow-up assessment, possible bias, no detailed sexual history, random sampling, etc.). But even given these serious shortcomings from behavioral studies such as these, there are sufficient data to indicate that an individual can change his or her sexual orientation from homosexual to heterosexual—something that would be an impossibility if homosexuality were caused by genetics.

**CONCLUSION**

Consider the obvious problem of survival for individuals who allegedly possess a gay gene: individuals who have partners of the same sex are biologically unable to reproduce (without resorting to artificial means). Therefore, if an alleged “gay gene” did exist, the homosexual population eventually would disappear altogether. We now know that it is not scientifically accurate to refer to a “gay gene” as the causative agent in homosexuality. The available evidence clearly establishes that no such gene has been identified. Additionally, evidence exists which documents that homosexuals can change their sexual orientation. Future decisions regarding policies about, and/or treatment of, homosexuals should reflect this knowledge.

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INTRODUCING OUR SECOND SPANISH-LANGUAGE TRANSLATOR: MICHAEL CORTEZ

In my “Note from the Editor” in the June issue of *Reason & Revelation*, I had the pleasure of introducing to you Moisés Pinedo, our first new Spanish-language translator. Several months prior to that, we intentionally had set in motion a series of events, the end result of which was designed to allow us to eventually bring on board as full-time staff members two extremely talented bi-lingual young men who could work with us to get all of our products—everything from correspondence courses, tracts, books, and audio/video items, to the entire content of our immensely popular Web site—translated into Spanish.

Our research had established that Spanish-speaking people represent the fastest-growing minority in the United States, and we therefore felt it was time for us to address what was rapidly becoming a pressing need. For more years than I care to remember, Christians in the Hispanic community—in this country and abroad—have asked us if we had anything available in Spanish, and we always have had to say, “no.” Now, with the addition of our two new translators, that is about to change—permanently!

In this month’s “Note from the Editor,” it is my pleasure to introduce to you Michael Cortez, the second young man whom I have hired to assist us in translating all of our materials into Spanish. Michael, who is 24, holds a B.S. degree in Spanish from Valdosta (Georgia) State University. In addition, in June of this year he graduated from the Memphis School of Preaching. Unbeknownst to him, I had had my eye on Michael for quite some time. I knew of the work of his father, Lionel Cortez, who directs the Panama School of Preaching in Panama City, Panama, and I knew that Michael possessed immense talent—not just in the area of public speaking, but also in the area of Spanish. I had spoken to a number of people who knew Michael, and each of them gave him an unqualified recommendation.

Therefore, when the time was “just right” (about 3-4 months prior to his graduation in June—before anyone else discovered that he was available and could hire him), I wrote him a letter and invited him to come to Montgomery for a job interview. By the time that interview was over, I was convinced that Michael was the person we needed to serve as our second full-time Spanish translator. I offered him the job—and he accepted. He and his new wife Amelia joined us in early July, and Michael immediately began his translation work.

The first task I assigned him was to proofread all of the items that Moisés has spent the last three months translating into Spanish (specifically, our introductory-, intermediate-, and advanced-level *Christian Evidences Correspondence Course*). As soon as Moisés and Michael complete the translation of these materials and several others, including the popular A.P. tract series, they will begin work on a complete Spanish-language mirror-image of our Web site (which received a quarter of a million page-hits this past March!).

We are extremely proud—and excited—to have Michael (and Moisés) as a part of our staff. Both of these young men are incredibly talented, and incredibly dedicated. Aside from being fluent in Spanish, they also are well trained in biblical issues (in addition to being terrific speakers). All of this has turned out to be an amazing combination for, and contribution to, our work! In so many ways, we have been doubly blessed by having them in our midst as a part of the ever-growing A.P. Family.

As I close I would like to mention the fact that both Moisés and Michael are available for speaking engagements at Spanish-speaking congregations of the churches of Christ. If they can be of service to you in any way, please call on them. They—as all of us at Apologetics Press—are here to serve.

Bert Thompson