Epigenetics and Homosexuality: A Brief Survey and Analysis

Dr. J. Alan Branch, Midwestern Baptist Theological Seminary
Paper Presented at the 68th Annual Meeting of the Evangelical Theological Society
November 15, 2016, San Antonio, TX

One of the most fascinating and burgeoning fields of scientific research is epigenetics. *Epigenetics* refers to chemical modifications of the human genome that alter gene activity without changing the DNA sequence. While many are familiar with arguments regarding *genetics* and homosexuality, epigenetics is now a growing focus of research into possible avenues of biological determination regarding sexual identity.

In October, 2015, Dr. Tuck Ngun of UCLA presented a paper at the annual meeting of the American Society of Human Genetics which suggested epigenetics may have a major influence on sexual orientation. Ngun claimed applying certain algorithms to data gathered from a specific sample of identical male twins allowed him to achieve a high degree of predictive accuracy regarding a person's sexual orientation based on DNA methylation patterns. In other words, he claimed to have discovered a fairly accurate method of determining if someone is a homosexual by merely examining the epigenome. Ngun's research is related to previous suggestions by researchers associated with the National Institute for Mathematical and Biological Synthesis and led by William Rice of the

University of California, Santa Barbara. In 2012, Rice and his colleagues proposed epigenetics may explain the heritability of some forms of homosexuality. These claims are startling and debatable to some while they provide a satisfying explanatory force to others. However, a review of current research into epigenetics demonstrates certain epigenetic tags may possibly be a contributing, but not a causative, factor in the development of a homosexual orientation. To substantiate this claim, this paper will begin by defining and differentiating between genetics and epigenetics, then the work of Rice, et al and Ngun will be summarized, Finally, I will conclude with a brief critique of claims related to epigenetics and homosexuality and suggestions will be offered concerning how a robust understanding of epigenetics may possibly interact with Christian doctrine and ethics.

I. Genetics and Epigenetics

In order to understand how findings in epigenetics are being leveraged in moral debate, it is important to define and differentiate between genetics and epigenetics. We will begin by summarizing genetics and epigenetics, and then discuss ways epigenetics may possibly influence human health.

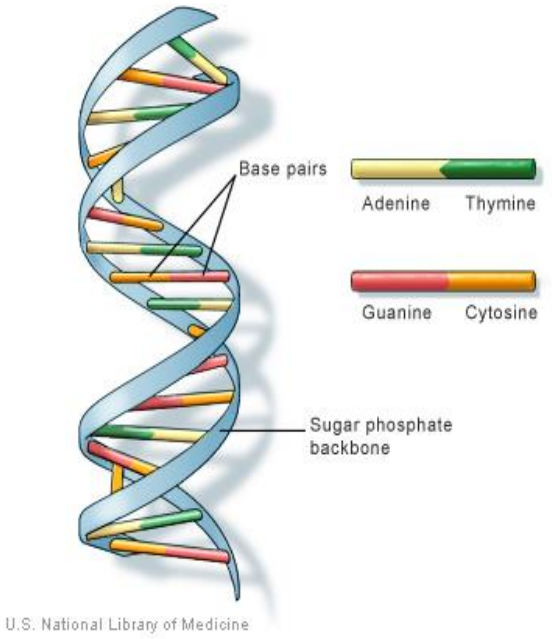
Genetics

Genetics is the study of heredity and variation in living organisms, especially the process of parents passing genes to their children. At the center of genetics is DNA, a long molecule stretched in a chain of nucleotides or "base pairs." Each human has around 6 billion nucleotides in each of the approximately 50 trillion cells in the human body.[1] Four types of bases are found in DNA – Adenine (A), Thymine (T), Cytosine (C) and Guanine (G): Adenine always bonds with Thymine and Cytosine always bonds with Guanine.[2] The base-pairs can be likened to letters, and DNA to a text or code that tells our bodies what to do.[3] The structure of DNA has been described as a "double helix" or "winding staircase." This diagram from the National Library of Medicine shows the structure:

[1] The estimates for the number of cells in the human body vary. Some of the difficulty relates to measuring by weight (higher number) versus volume (lower number). In 2013, one group of scientists suggested 37.2 trillion cells in the average human. Eva Bianconi, Allison Piovesan, Federica Facchin, Alina Beraudi, Raffaella Casadei, Flavia Frabetti, Lorenza Vitale, Maria Chiara Pelleri, Simone Tassani, Francesco Piva, Soledad Perez-Amodio, Pierluigi Strippoli, & Silvia Canaider, "An Estimation of the Number of Cells in the Human Body," *Annals of Human Biology* 40.6 (July 2013): 463 – 471. This number was derived by estimating the number of cells in each individual organ or area of the body. DNA is almost unimaginably small and is only 2 nanometers across or 2,000 times thinner than human hair

[2] Adenine and Guanine are members of a class of compounds called Purines. Thymine and Cytosine are members of a class of compounds called Pyrimidines. When two strands of polynucleotides associate to each other, bases form a hydrogen bond and stabilize the right-handed double-helix structure**.** Hydrogen bonds form when a hydrogen atom interacts with electron-attracting atoms, usually oxygen or nitrogen atoms. In RNA, uracil replaces thymine as the usual complement of adenine. Thus, thymine is usually seen only in DNA and uracil only in RNA.

[3] Ted Peters, *Playing God? Genetic Determinism and Human Freedom* (New York: Routledge, 1997), 3.



Nessa Carey suggests another way to think of DNA is to compare it to a zipper. While the zipper is not a perfect analogy, it gives us a basic understanding. One thing we all know about a zipper is that it is formed of two small strips of fabric facing each other: Similarly, DNA is composed of two strips facing each other. Likewise a zipper has "teeth" on each strip. The four bases of DNA are analogous to the teeth in a zipper. The bases on each side of the DNA "zipper" can link up to each other chemically and hold the zipper together. The two fabric strips of a zipper on which the teeth are attached are equivalent to the DNA backbones. The two sides of the DNA zipper are basically twisted around to form a spiral structure – the famous double-helix. The zipper analogy does have limits because the teeth of a DNA zipper aren't all equivalent. If one of the teeth is Adenine (A base), it

can only link up with Thymine (T base) on the opposite strand. Likewise, if one of the teeth is guanine (G base), it can only link up with Cytosine (C Base). Carey explains, "This is known as the base-pairing principle. If an A tried to link with a C on the opposite strand it would throw the whole shape of the DNA out of kilter, a bit like a faulty tooth on a zipper."[4]

DNA sends "messages" via Ribonucleic Acid (RNA). RNA functions to carry the 'genetic message' from nuclear DNA to the ribosomes. A specific form of RNA called "messenger" RNA (mRNA) leads to the development of proteins which form enzymes, hormones, structural proteins of cells and other vital components of the body. The central dogma of molecular biology explains that DNA is decoded to make RNA, and then RNA is used to make polypeptides that subsequently form proteins.[5] The flow of genetic information is almost always unidirectional: DNA to RNA to polypeptides to proteins. That is, the sequence of DNA specifies the synthesis and sequence of RNA by a process known as transcription. Messenger RNA in turn specifies the synthesis and sequence

[4] Nessa Carey, *The Epigenetics Revolution*: *How Modern Biology is Rewriting Our Understanding of Genetics, Disease, and Inheritance* (New York: Columbia University Press, 2012), 44. Carey is formerly a senior lecturer at Imperial College School of Medicine in London and is now director of exploratory research at CellCentric.

[5] Tom Strachan and Andrew Read, *Human Molecular Genetics*, 4th ed. (New York: Garland Science, 2011), 2.

of polypeptides, which are the building blocks of proteins by a process known as translation.[6]

Chromosomes are bundles of DNA. Humans have 23 pairs of chromosomes (46), one of each pair from each parent. Within each chromosome are genes, a sequence of DNA on a chromosome that is required for production of a functional product, which can either be a protein or a functional RNA molecule.[7] Genes vary in size, from just a few thousand base pairs to over two million base pairs. Genes tell each cell what to do and how to do it. To put it differently and in the broadest sense, genes are simply units of hereditary information. The Human Genome Project has revealed that there are probably about 20,500 human genes.[8]

But recent findings indicate genes are not quite as simple as was once thought. Until the advent of genome-wide analysis, a typical human gene was imagined to be well-defined and separated from its neighbors by identifiable intergenic spaces. We now know some genes overlap with others or are entirely imbedded within much larger genes. Furthermore, intergenic DNA – which makes up most of the genome – is now recognized

---

[6] William B. Dobyns, Susan L. Christian, and Soma Das, "Introduction to Genetics," in *Swaiman's Pediatric Neurology*, 5th ed., vol. 1, *Principles and Practice*, Kenneth Swaiman, Stephen Ashwal, Donna M. Ferriero, and Nina Schor, eds. (New York: Elsevier, 2012), 277.
[7] William B. Dobyns, et al, "Introduction to Genetics," in *Swaiman's Pediatric Neurology*, 5th ed., 281.
[8] This number is much lower than thought before the project began, with initial estimates ranging from 50,000 to 140,000 genes. The lower number of genes is a very surprising discovery.

as being much more functionally important for the correct expression of genes. As Strachan and Read say, "There is a growing awareness that the way in which our genome works is much more complex than it once appeared."[9]

The complexity and intricacy of DNA leaves us with many unanswered questions. For example, if every cell in the human body contains the entire DNA code, why do cells only perform specific functions? Furthermore, some genetic anomalies have posed questions regarding DNA and the inheritance of traits. For example, on rare occasions, identical twins have different hair color. But if they share identical DNA, how could the hair colors be different? The answers to these questions and others are found in *epigenetics*.

Epigenetics

Epigenetics – a word with a rough literal meaning of "on genes" – refers to chemical modifications of the human genome that alter gene activity without changing the DNA sequence.[10] DNA is wrapped around

[9] Tom Strachan and Andrew Read, *Human Molecular Genetics*, 4th ed., 346. This paragraph is summarized from Strachan and Read pages 276 and 346.

[10] Or stated slightly differently, epigenetics refers to all modifications to genes other than changes in the DNA sequence itself which alter gene expression. Joanna Downer, "Backgrounder: Epigenetics and Imprinted Genes," accessed April 24, 2014, http://www.hopkinsmedicine.org/press/2002/november/epigenetics.htm; and William B. Dobyns, Susan L. Christian, and Soma Das, "Introduction to Genetics," in *Swaiman's Pediatric Neurology*, 5th ed., vol. 1, *Principles and Practice*, Kenneth Swaiman, Stephen Ashwal, Donna M. Ferriero, and Nina Schor, eds. (New York: Elsevier, 2012), 277.

proteins called histones and both DNA and the histones are covered with chemical "tags." These histones and chemical tags (or "epi-marks") are part of each person's *epigenetics* and constitute an extra layer of information attached to our genes' backbones that regulates their expression.[11] As science has discovered more and more about genetic traits, we have learned that these epigenetic structures regulate genome activity and govern which genes in the DNA of any given cell will be active. These epigenetic structures can be thought of as switches and knobs which turns things "on or off" or "up and down." Perhaps the most fascinating difference between DNA and epigenetics is that the genome does not change during cell division throughout a person's lifetime, but the epigenome can change.

Another helpful analogy for understanding epigenetics is to think of actors reading a script for a movie. For example, Director Baz Luhrmann hands Leonardo DiCaprio his shortened version of Shakespeare's script for *Romeo and Juliet*, on which the director has written or typed various notes – such as directions for camera placements and other technical information. Whenever DiCaprio's copy of the script is photocopied, Luhrmann's additional information is copied along with it. Claire Danes,

[11] "Study Finds Epigenetics, Not Genetics, Underlies Homosexuality," National Institute for Mathematical and Biological Synthesis, December 11, 2012, accessed April 24, 2014, http://www.nimbios.org/press/FS_homosexuality.

playing the part of Juliet, also has a script for Romeo and Juliet. While the notes on her copy are different from those on DiCaprio's, Danes' notes will also survive photocopying. Nessa Carey explains the analogy and says, "That's how epigenetic regulation of gene expression occurs – different cells have the same DNA blueprint (the original author's script) but carrying varied molecular modifications (the shooting script) which can be transmitted from mother cell to daughter cell during cell division."[12]

Among several epigenetic mechanisms, perhaps the most important is *methylation*, an epigenetic signaling tool that can fix genes in the "off" position. During methylation, a quartet of atoms called a methyl (CH_3) group attaches to a gene at a specific point on the DNA strand itself.[13] There, the methyl group remodels the chromatin and affects how the gene is expressed.[14] The effect of this process is effectively to tell a particular

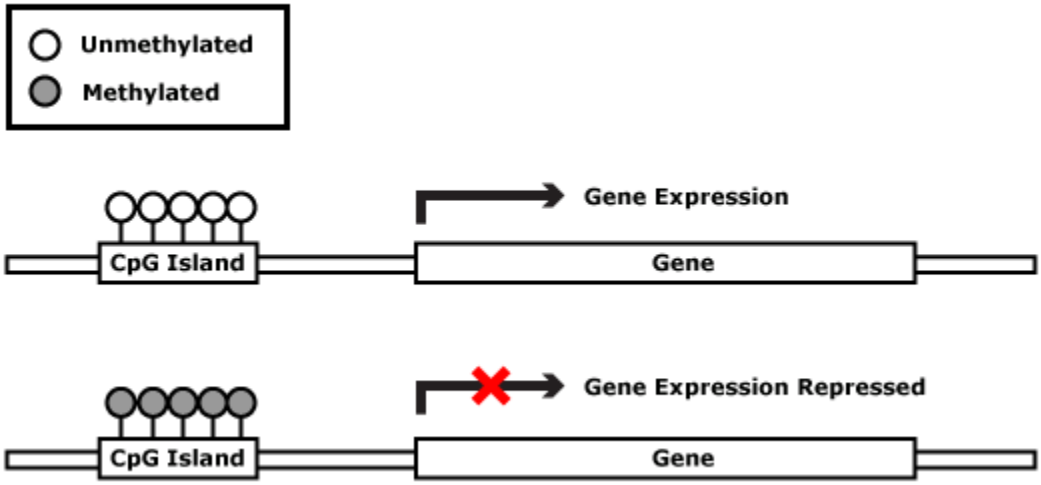
---

[12] I got this analogy from Nessa Carey, *The Epigenetics Revolution*, 55.

[13] It often attaches to the fifth carbon atom of a cytosine ring.

[14] Sally Robertson, "What is DNA Methylation?," accessed July 14, 2016, www.news-medical.net/life-sciences/What-is-DNA-Methylation.aspx and U.S. Department of Health and Human Services: National Institute on Alcohol Abuse and Alcoholism, "Epigenetics: A New Frontier For Alcohol Research," *Alcohol Alert* 86 (no date): 2; accessed August 4, 2016, http://pubs.niaaa.nih.gov/publications/AA86/AA86.pdf. What is "chromatin"? Packed inside the nucleus of every human cell is nearly 6 feet of DNA, which is subdivided into 46 individual molecules. Collecting all this material into a microscopic cell nucleus is an extraordinary feat of packaging. For DNA to function when necessary, it can't be haphazardly crammed into the nucleus or simply wound up like a ball of string. Consequently, during interphase (the period of time when the cell is *not* dividing), DNA is combined with proteins and organized into a precise, compact structure, a dense string-like fiber called chromatin, which condenses even further into chromosomes during cell division. Michael W. Davidson, "Chromatin and Chromosomes," Molecular Expressions: Cell Biology and Microscopy, last updated November 13, 2015, accessed August 4, 2016, https://micro.magnet.fsu.edu/cells/nucleus/chromatin.html.

gene not to code or to "be quiet." DNA methylation has important consequences and allows particular gene expression patterns to be stably transmitted to daughter cells.[15] By "turning off" other genes, the epigenetic mechanism of methylation makes sure that a particular cell only does the task assigned to it. Methylation usually results in silencing of a gene. The following diagram from the University of California, San Francisco demonstrates how methylation prevents a gene from expressing:



[16]

Another very important epigenetic mechanism is acetylation which regulates diverse cellular processes such as gene expression,

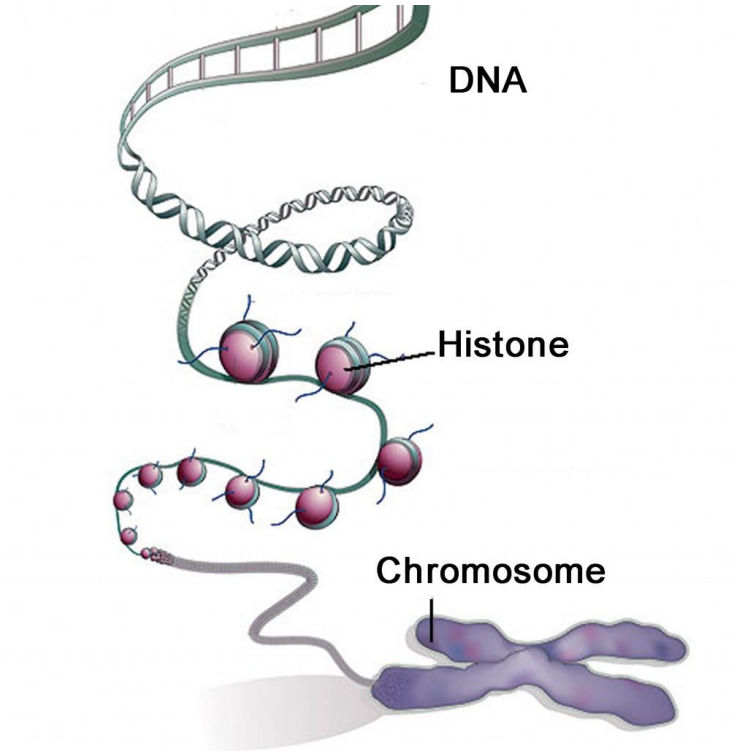
[15] Tom Strachan and Andrew Read, *Human Molecular Genetics*, 4th ed., 262.

[16] "CpG Islands" are short interspersed DNA sequences that deviate significantly from the average genomic pattern by being GC-rich, CpG-rich, and predominantly nonmethylated. Most, perhaps all, CGIs are sites of transcription initiation. A CpG island announces, "A gene is about to begin here." Aimee M. Deaton and Adrian Bird, "CpG Islands and the Regulation of Transcription," *Genes and Development*, 2011, accessed August 11, 2016, http://genesdev.cshlp.org/content/25/10/1010.full.

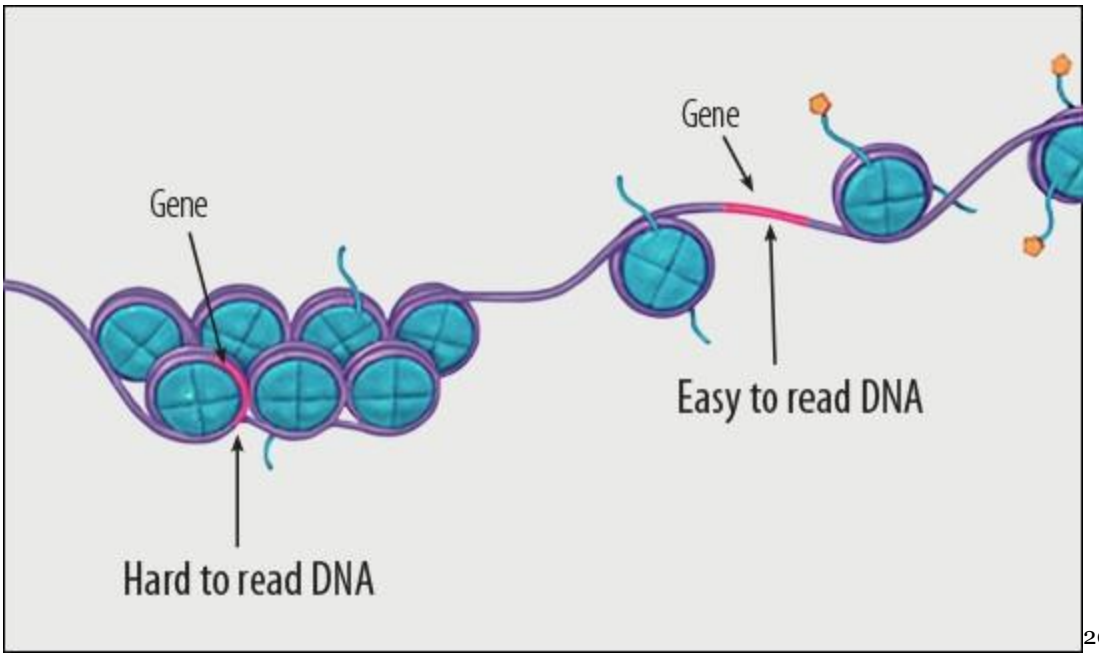
recombination, and DNA damage repair.[17] For some time the thought was that methylation turned genes "off" and acetylation turned genes "on." However, in many cases gene expression is more subtle than genes being either "on" or "off" like a toggle switch: it's much more like the volume dial on a radio with traits being amplified or muted.[18]

Histone modification is another important epigenetic mechanism. Histones serve as a kind of "spindle" around which DNA wraps itself. As was noted above, histones have chemical "tags" which affect the expression of genes. Histones can be modified in many ways, and these modifications can turn genes both on and off, depending on the situation.[19] The following diagram can help you visualize histones and how they function:

[17] Acetylation neutralizes the charge of lysine, which attenuates electrostatic interactions within and between nucleosomes, allowing other nuclear proteins to access the underlying DNA sequences. Yu Wang, Scott P. Kallgren, Bharat D. Reddy, Karen Kuntz, Luis López-Maury, James Thompson, Stephen Watt, Chun Ma, Haitong Hou , Yang Shi, John R. Yates III , Jürg Bähler, Matthew J. O'Connell , and Songtao Jia, "Histone H3 Lysine 14 Acetylation Is Required for Activation of a DNA Damage Checkpoint in Fission Yeast," *The Journal of Biological Chemistry* 287.6 (February, 2012): 4386.
[18] Carey, *The Epigenetics Revolution*, 68.
[19] U.S. Department of Health and Human Services: National Institute on Alcohol Abuse and Alcoholism, "Epigenetics: A New Frontier For Alcohol Research," *Alcohol Alert* 86 (no date): 4, accessed August 4, 2016, http://pubs.niaaa.nih.gov/publications/AA86/AA86.pdf.



Whether genes are expressed and “on” or unexpressed and “off” is related to how tightly they are wrapped around histones. Genes that are “loosely” wrapped are “on” and can be expressed meaning they can make proteins necessary for a specific function. Genes that are “tightly” wrapped are “off” and can’t be read by the cells. The following diagram from PBS gives an idea of how these processes work:



[20]

Epigenetics and The Dutch Hunger Winter

Within the field of epigenetics, some have strongly argued that stress during pregnancy can result in epigenetic changes which can be transmitted from mother to child and affect the child's health, with the Dutch "Hunger Winter" of 1944 – 1945 often being cited as evidence. In September, 1944, the Allies attempted to drive into Germany through the Netherlands in operation Market Garden. The operation failed and left the Netherlands in a desperate situation after the Germans imposed a total embargo on the Dutch population because of their support for the Allies. Food rations declined to extremely low levels between February and May

[20] Diagram from Nsikan Akpan, "Don't Blame Grandma yet, but your asthma may be her fault," *PBS Newshour* December 9, 2015, accessed November 7, 2016, http://www.pbs.org/newshour/updates/history-of-epigenetics-asthma/.

1945, resulting in an individual average daily official ration below 1000 calories.[21] Children conceived and born during the Dutch famine have served as a kind of "natural experiment" to study the effects of malnutrition during pregnancy on the health outcomes of the children being carried in utero during the famine. Numerous deleterious effects among this study group have been documented, including higher incidence of glucose intolerance for children exposed to famine during any stage of gestation.[22] Children exposed to the famine earlier in gestation were more prone to negative outcomes such as coronary heart disease, a more atherogenic lipid profile,[23] disturbed blood coagulation, and increased stress responsiveness. Women exposed to famine in early gestation also had an increased risk of breast cancer.[24]

One of the more surprising findings from the Dutch Hunger Winter has been the long-term effect on the weight of children born during this era. If a mother was well-fed around the time of conception and malnourished for the last few months of gestation, the baby was likely to be born small.

---

[21] Ernst J. Franzek, Niels Sprangers, A. Cecile J. W. Janssens, Cornelia M. Van Duijn, & Ben J. M. Van De Wetering, "Prenatal Exposure to the 1944–45 Dutch 'Hunger Winter' and Addiction Later in Life," *Addiction* 103 (2008):433.
[22] A. C. Ravelli, J.H. van der Meulen, R. P. Michels, C. Osmond, D. J. Barker, C.N. Hales, et al, "Glucose Tolerance in Adults after Prenatal Exposure to Famine," *Lancet* 351 (1998): 173–177.
[23] "Atherogenic Lipid Profile" means a higher susceptibility to formation of abnormal fatty masses in arterial walls.
[24] T. Roseboom, S. de Rooij, R. Paiinter, "The Dutch Famine and Its Long Term Consequences for Adult Health," *Early Human Development* 82.8 (August 2006): 485 – 491.

Furthermore, these children tended to remain small throughout adulthood and had much lower obesity rates than the general population. In contrast, if the baby was conceived during the height of the famine and the mother suffered malnutrition during the early months of conception, but was then well-fed, the baby was likely to have a normal birth weight. A somewhat unexpected finding is that people born to this second group – conceived in famine, but normal birth weight – had higher obesity rates than normal in adulthood.[25] Even more surprisingly, some of the negative traits associated with nutritional deprivation may be passed down to succeeding

[25] The description of these two groups is summarized from Nessa Carey, *The Epigenetics Revolution*, 3 – 4. The programming of adult obesity by intrauterine food restriction without accompanying changes in birth weight has been replicated in rodent and sheep animal models. Laura C. Schulz, "The Dutch Hunger Winter and the Developmental Origins of Disease," *Proceedings of the National Academy of Science* 107.39 (September 28, 2010): 16757. Research on children born during the Chinese famine of 1959 – 1961 found higher rates of obesity in adulthood for the women, but not men. Z. Yang, W. Zhao, X. Zhang, R. Mu, Y. Zhai, L. Kong, C. Chen, "Impact of famine during pregnancy and infancy on health in adulthood," *Obstetrics Review,* Supplement 1 (March, 2008): 95 – 99.

generations.[26] Prenatal exposure to the Dutch Hunger Winter is also associated with increased rates of schizophrenia[27] and drug addiction.[28]

Epigenetic mechanisms are largely credited with being the driving force behind the outcomes observed in children of the Hunger Winter. Their subsequent health was negatively affected by the crisis they endured in utero. While their DNA did not change, the manner in which it has been expressed appears to have been modified. A 2015 study of the Dutch Hunger Winter has suggested that the patterns of methylation were affected in utero in children of the famine. These authors emphasize the gestational timing of prenatal exposure to famine has a significant influence on the process of methylation, with early prenatal development being the most critical period for malnutrition to have epigenetic effects.[29] Of special

---

[26] T. J. Roseboom, R. C. Painter, A.F. van Abeelen, M.V. Veenendaal, S.R. de Rooij, "Hungry in the Womb: What are the Consequences? Lessons from the Dutch Famine," *Maturitas* 70.2 (October 2011): 141 – 145. Some readers may notice an echo of Lamarck's theory here: the inheritance of acquired traits. While remaining within the Neo-Darwinian synthesis, advocates of evolution are beginning to admit some traits acquired by parents may be passed down to succeeding generations. But these are in the minority and seen as a subset of the overall evolutionary process which focuses on changes in the genome itself as opposed to the epigenome. The implications of new discoveries in epigenetics on the theory of evolution are far beyond the scope of this paper.

[27] E. S. Susser and S. P. Lin, "Schizophrenia after Prenatal Exposure to the Dutch Hunger Winter of 1944–45," Archives of General Psychiatry 49 (1992): 983–988; E.S. Susser, R. Neugebauer, H.W. Hoek, A.S. Brown, S. Lin, D. Labovitz, et al. "Schizophrenia after prenatal famine. Further Evidence," Archives of General Psychiatry 53 (1996): 25 -31. Children exposed to the Chinese famine of 1959 – 1961 also demonstrated a higher incidence of schizophrenia. See David St. Clair, Mingqing Xu, Peng Wang, Yaqin Yu, Yourong Fang, Feng Zhang, Xiaoying Zheng, Niufan Gu, Guoyin Feng, Pak Sham, Lin He, "Rates of Adult Schizophrenia Following Prenatal Exposure to the Chinese Famine of 1959-1961," *Journal of the American Medical Association* 294.5 (August 3, 2005): 557 – 562.

[28] Ernst J. Franzek, Niels Sprangers, A. Cecile J. W. Janssens, Cornelia M. Van Duijn, & Ben J. M. Van De Wetering, "Prenatal Exposure to the 1944–45 Dutch 'Hunger Winter' and Addiction Later in Life," *Addiction* 103 (2008):433 – 438.

[29] Elmar W. Tobi, Jelle J. Goeman, et al, "DNA Methylation Signatures Link Prenatal Famine Exposure to Growth and Metabolism," *Nature Communications* 5 (July 7, 2015):

interest is that the researchers discovered genes associated with growth and development were affected by epigenetic changes which resulted in these genes being expressed differently, thus affecting the growth of the children.

The concept that maternal stress during early gestation affects children's epigenetics is central to new theories regarding the origin of homosexuality. Just as the children of the Dutch Hunger Winter give evidence of continuing epigenetic changes regarding the way their genes are expressed, some researchers are now suggesting that prenatal stress may influence the way genes related to sexual development in the brain are expressed, resulting in same-sex attraction and a homosexual orientation.

II. Homosexuality and Epigenetics

Suggestions that homosexuality may have an epigenetic origin are rather recent. The two most well-known statements of this argument to date are from an article by William R. Rice and colleagues in 2012 and a paper delivered Dr. Tuck Ngun of UCLA presented at the annual meeting of the American Society of Human Genetics in October, 2015.

Rice, Gavrilets, & Friberg, 2012

http://www.nature.com/ncomms/2014/141126/ncomms6592/full/ncomms6592.html. The researchers were associated with Leiden University Medical Center, Harvard University, and Columbia University's Mailman School of Public Health. The researchers compared the DNA of the Hunger Winter children at 1.2 million CpG methylation sites comparing them with same-sex siblings not exposed to famine.

In 2012, a team of researchers associated with the National Institute for Mathematical and Biological Synthesis and led by William Rice an evolutionary geneticist at the University of California, Santa Barbara, joined by Sergey Gavrilets, a mathematician at the University of Tennessee, and Urban Friberg, an evolutionary biologist at the University of Uppsala, suggested epigenetics may explain the heritability[30] of some forms of homosexuality. Published in December, 2012 in *The Quarterly Review of Biology*, they argued that epigenetic changes to the early embryo can affect the expression of genes related to androgen signaling which then influences later sexual orientation.

Two major theoretical premises undergird the claims of Rice and his colleagues. The first premise is that androgen levels drive sexual orientation in a manner similar to the way they drive the development of genitalia. The entire process of prenatal gender-specific growth is driven by the release of hormones at specific junctures. As children grow in the mother's womb, certain sex hormones are produced in quantity at specific times to help their tiny bodies grow in a gender-specific direction. Testosterone, an

[30] "Heritability" is a term used to describe the complex interaction between genes and environment which results in many traits we express. For example, someone may have a genetic predisposition to be taller than normal. However, if a child with this genetic trait is raised in a war-torn country in which his diet lacks essential nutrients, he will not grow as tall as he would have under better conditions. Both his genetics and his environment contribute to the final height he reaches in adulthood. Usually, heritable traits are those which demonstrate a lot of variation within the population as a whole. For example, someone who is two inches shorter than average is still within the normal deviation for height in a particular community. In contrast, a child born with three toes would be considered abnormal.

androgen, is especially important in this process. Both boys and girls produce testosterone, but testosterone production peaks in male babies at around 16 weeks of gestation, but after this declines to around the same level as in prenatal females. Sexual development in females is also driven by hormones, or more specifically the absence of male hormones. Since girls do not have testes, not enough testosterone is produced to masculinize genitalia and, thus, the external genitalia develop in a female manner.[31] In humans, the process of sex determination and forming of the external genitalia is virtually complete by the 13th week of gestation.[32] The theory of Rice, et al assumes that androgens are also central to the development of sexual orientation. Mainly, they argue that homosexuals received the correct hormones to guide their genitalia in proper development, but later in prenatal development – especially in the brain – they received the incorrect level of hormones or the wrong hormones, resulting in a homosexual orientation.[33]

---

[31] Since females do not have the SRY gene, the primitive gonads become ovaries and not testes. Female ovaries actually produce a small amount of testosterone. Both males and females produce testosterone and estrogen, but males produce far more testosterone and females produce far more estrogen.

[32] Margaret M. McCarthy, "Estradiol and the Developing Brain," *Physiological Review* 88.1 (January 2008): 91 – 124, accessed July 9, 2014, http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2754262/pdf/nihms117872.pdf, 7.

[33] The authors say, "The androgen signaling pathways differ among organs and tissues, the same inherited sexually antagonistic-epi mark can affect only a subset of sexually dimorphic traits, e.g., no effect on the genitalia, but a large effect on a sexually dimorphic region of the brain." William R. Rice, Urban Friberg, and Sergey Gavrilets, "Homosexuality As A Consequence of Epigenetically Canalized Sexual Development," *The Quarterly Review of Biology* 87.4 (December 2012): 358. This central premise – that androgens are in fact the driving factor in the development of sexual orientation – has not yet been proven. Eric Vilain, the lab supervisor for Tuck Ngun, agrees with the basic trajectory of Rice's model.

A second major premise is that a mother or father could pass down the wrong epigenetic marks to their children. Usually, epigenetic "tags" or "marks" develop very early soon after conception. The parents' epigenetic tags are erased and replaced by unique ones for the child. But if epigenetic marks that direct sexual development are not erased correctly, a mother could pass down epi-marks consistent with female development to her son, resulting in an attraction to men, and vice versa for a father and his daughters.[34] In other words, a young fetus inherits epigenetic marks that are not consistent with the baby's gender. They then hypothesize these sexually-antagonistic (opposed to the child's gender) epigenetic marks "influence androgen signaling in the part of the brain controlling sexual orientation, but not the genitalia nor the brain region(s) controlling gender identity."[35] In other words, the epigenetics cause a child to process the wrong sex hormones or sex hormones in the wrong amounts into the brain.

Vilain acknowledges that girls with Congenital Adrenal Hyperplasia were exposed to very high levels of androgens in utero and have masculinized genitalia and report a higher incidence of same-sex attraction. But he then adds, "It remains to be seen whether smaller variants of testosterone that do not result [in masculinized genitalia] also lead to attraction of same sex partners." Vilain does not agree with my moral stance regarding homosexuality. *See* Sabrina Richards, "Can Epigenetics Explain Homosexuality?," *The Scientist* January 1, 2013, accessed August 9, 2016, http://www.the-scientist.com/?articles.view/articleNo/33773/title/Can-Epigenetics-Explain-Homosexuality-/. Elsewhere, Vilain and Ngun concur with one of Rice's core assertions and say, "We believe it is very likely that sex-specific epigenetic marks are at least (partly) responsible for sexually dimorphic traits including sexual orientation." Tuck Ngun and Eric Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?," *The Epigenetic Shaping of Sociosexual Interactions: From Plants to Humans* 86 (2014): 175.

[34] Sabrina Richards, "Can Epigenetics Explain Homosexuality?"

[35] William R. Rice, Urban Friberg, and Sergey Gavrilets, "Homosexuality as a Consequence of Epigenetically Canalized Sexual Development," 358.

Thus, they hypothesize this causes the child to experience same-sex attraction as he or she matures. But determining whether or not these proposed epigenetic marks affecting sexual orientation exist has not been proven. Furthermore, to prove they have not been erased will be difficult to test because such marks, if they exist, will probably be in the brain.[36]

Rice, et al make a fascinating admission and say, "*Although we cannot provide definitive evidence that homosexuality has a strong epigenetic underpinning*, we do think that available evidence is fully consistent with this conclusion."[37] While they admit they cannot provide definitive evidence, they say in their conclusion, "If our model is wrong, it can be rapidly falsified and discarded."[38] The work by Rice and his team is a specific type of academic research called "meta-analysis, a quantitative, formal study design used to assess systematically previous research studies in order to derive conclusions about a particular body of research.[39] Such work is also called a *review* article, meaning it is an article that synthesizes other research already in print and suggests possible implications. Meta-analysis is often the first step in defining avenues for future research by

---

[36] This is Vilain's opinion. *See* Sabrina Richards, "Can Epigenetics Explain Homosexuality?" But again, Vilain finds a great deal of Rice's work compelling.
[37] William R. Rice, et al, "Homosexuality as a Consequence of Epigenetically Canalized Sexual Development," 357. Emphasis added.
[38] Ibid., 362.
[39] This definition is from A. B. Haidich, "Meta-Analysis in Medical Research," *Hippokratia* 14 (2010): 29.

summarizing what has been done, what conclusions have been reached, and providing suggestions for future research. Essentially, Rice, et al. are saying to other researchers, "Hey, you might look over here." In 2015, Tuck Ngun claimed to have discovered some findings quite consistent with the Rice model for epigenetics and homosexuality.

Tuck Ngun, 2015

On October 8, 2015, Tuck Ngun, a post-doctoral scholar in the Department of Genetics at UCLA's David Geffen School of Medicine, presented a paper titled "A Novel Predictive Model of Sexual Orientation Using Epigenetic Markers" at the annual meeting of The American Society of Human Genetics. The lead researcher was Eric Vilain (Ph.D., M.D.), associate professor and Chief of the Division of Medical Genetics at UCLA.[40]

Ngun and Vilain published a paper in 2014 in which they evaluated and critiqued the epigenetic model proposed by Rice and colleagues in 2012. Ngun and Vilain agreed with much of Rice's model, but disagreed that "sex-reversing sensitivity to androgen signaling via epigenetic markers

[40] "Eric Vilain, M.D., Ph.D.," David Geffen School of Medicine, accessed August 10, 2016, https://people.healthsciences.ucla.edu/institution/personnel?personnel_id=9435. Ngun presented his findings earlier on March 21, 2015 in Philadelphia at the meeting of the Society for Research in Child Development. Accessed October 10, 2016, https://gendercenter.genetics.ucla.edu/node/75.

will result in homosexuality in both sexes."[41] Ngun and Vilain reject this premise because they think the different biological and genetic factors affect homosexuality in men and women.[42] Essentially, they argue there are different types of homosexuality while Rice, et al appear to be striving at a model which is universally applicable to all homosexuals. Nonetheless, the suggestions of Rice, Friberg, and Gavrilets gave a trajectory for the research by Ngun and Vilain.[43]

Ngun claimed an algorithm his team developed can predict sexual orientation in males at a rate of 67% accuracy using epigenetic information from five to nine regions of the human genome.[44] The data was generated using a sample composed of DNA derived from the saliva of 37 pairs of identical twins who were discordant for sexual orientation (one was homosexual and one was not) along with a control group of 10 pairs of identical twins who were concordant for homosexuality (both were

---

[41] Tuck Ngun and Eric Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?," 175.

[42] Part of their reasoning is based on claims related to Xq28 and homosexuality in males while no genetic region whatsoever has been connected to homosexuality in females. I critiqued claims related to Xq28 in a 2015 paper delivered at ETS, "Xq28 and Homosexuality: An Update on Current Research."

[43] Ngun and Vilain said, "Rice, Friberg, and Gavrilets (2013) have proposed steps to test their epigenetic hypothesis. Our group is currently testing the hypothesis that discordance in sexual orientation between [monozygotic] twins is related to discordance in epigenetic traits." [43] Tuck Ngun and Eric Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?," 178.

[44] T. C. Ngun, W. Guo, N. M. Ghahramani, K. Purkayastha, D. Conn, F. J. Sanchez, S. Bocklandt, M. Zhang, C. M. Ramirez, M. Pellegrini, Eric Vilain, "Program Number 95: A novel predictive model of sexual orientation using epigenetic markers." A Paper Delivered October 8, 2015 at the Annual Meeting of The American Society of Human Genetics, accessed February 26, 2106, https://ep70.eventpilotadmin.com/web/page.php?page=IntHtml&project=ASHG15&id=150123267. One of the frustrating aspects of Ngun's research is that the paper has not been published. I personally wrote to Ngun via the USPS asking for a copy, but received no reply.

homosexual). Ngun and his colleagues looked for epigenetic modifications made to the genes of the 47 sets of male twins. Specifically, they analyzed 140,000 regions in the genomes of the twins and looked at 400,000 methylation marks, which can be thought of as "chemical Post-It notes" that dictate when and where genes are activated.[45] The team then used an algorithm they developed to search out gene regions in which methylation patterns differed significantly between the two groups. They found five sites of particular interest– three in regions of intergenic DNA, the role of which is unclear, and two in genes whose roles are relatively well established. One of the genes the Ngun team identified as having epigenetic changes is involved with the production of MHC II molecules which are important for a healthy immune system, but are also thought to affect sexual attraction by affecting response to odor.[46]

The Vilain-Ngun team then split their sample of 37 discordant twin pairs into two groups. Using the test results from 20 of these pairs, they developed a model to predict if a person in one of the seventeen remaining

---

[45] Ed Yong, "No, Scientists Have Not Found the 'Gay Gene,'" *The Atlantic* October 10, 2015, accessed July 15, 2016, http://www.theatlantic.com/science/archive/2015/10/no-scientists-have-not-found-the-gay-gene/410059/.

[46] Much of the information in this summary is found in Jessica Hamzelou, "Gay or Straight? Saliva Test Can Predict Sexual Orientation," *New Scientist* October 8, 2015, accessed August 10, 2016, https://www.newscientist.com/article/dn28307-gay-or-straight-saliva-test-can-predict-male-sexual-orientation/. *See also* Claus Wedekind and Dustin Penn, "MHC Genes, Body Odours, and Odour Preferences," *Nephrology, Dialysis, and Transplantation* 15.9 (2000): 1269 – 1271.

pairs is straight or gay based on the methylation patterns of their genes. When they tested their model on the remaining pairs of male twins using their algorithm, they claimed it correctly predicted sexual orientation 67 per cent of the time.

In their 2014 article, Ngun and Vilain suggested that slight variations in the uterine environment may explain why some twin pairs are discordant for homosexuality. For example they suggested the twins may receive different nutrients even though they share the same uterus, saying, "Although the nutrient bath in which both twins develop may be highly similar, there could be differences that could affect epigenetic markers on genes relevant to sexual orientation."[47]

The burgeoning field of epigenetics has provided a new avenue of research for people seeking a biological basis for homosexuality. Does Ngun's research into epigenetics provide compelling reasons to believe homosexuality is an innate trait caused by epigenetic modifications?

III. Evaluation of Arguments Regarding Epigenetics and Homosexuality

An evaluation of the data regarding homosexuality and epigenetics reveals some fascinating insights into the way we as humans function and

[47] Tuck Ngun and Eric Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?," 173.

the manner in which the human genome expresses particular traits. However, data to date does not substantiate the claim that prenatal epigenetic changes "hard-wire" someone for homosexuality. To demonstrate this claim, I will review some of the data about epigenetics in general, Ngun's research in particular, data regarding epigenetics and drug addictions, and then move to a Scriptural-theological evaluation of the data.

Epigenetics in General: Possible Insights

Christians must not hastily dismiss the evidence concerning epigenetic changes derived from the Dutch Hunger Winter: If the mother had a low caloric intake during pregnancy, the child often had problems with either being underweight or obese as an adult based on the period during the famine the child was in utero. This should not surprise us as many Christians are quite aware of Fetal Alcohol Syndrome and the negative outcomes in children associated with a mother who drinks while the child is in utero. What seems to be more surprising is the degree to which the *grandchildren* of the Dutch Hunger Winter also experience some of the same problems.[48] But at the same time, we must not adopt a

[48] One study using pigs claimed sire pigs who were exposed to inordinate amounts of stress experienced epigenetic changes on their sperm. These changes were passed on to offspring and led to alterations in the hypothalamic-pituitary-adrenal stress axis, affecting the offspring's response to stress. *See* Ali B. Rodgers, Christopher P. Morgan, N. Adrian Leu, and Tracy L. Bale, "Transgenerational Epigenetic

deterministic approach. The data indicates a higher occurrence and susceptibility to certain negative outcomes, but it does not point to an unalterable fixed destination in the lives of these people. Epigenetic factors can increase one's *susceptibility* to any number of problems, but human volition still plays a significant role in the progression of diseases associated with Dutch Hunger Winter children.

When considering epigenetics and homosexuality, it is at least possible that epigenetics play a role in some cases of same-sex attraction. But epigenetics are far from determinative. Perhaps our view should be influenced by the more robust model regarding prenatal epigenetics and disease. What we see is that prenatal epigenetics may contribute to an increased susceptibility to certain diseases such as cancer or heart disease, but these susceptibilities are affected by myriad of other factors related to the choices a person makes. Likewise, prenatal epigenetic changes may possibly increase a person's likelihood of being homosexual, but embracing a gay identity is fueled by many other factors related to human volition. In other words, a predisposition to increased likelihood of homosexuality does not mean one is *predetermined* to be a homosexual.

Programming Via Sperm microRNA Recapitulates Effects of Paternal Stress," *Proceedings of the National Academy of Science* 112.44 (November 3, 2015): 13699-13704.

Concerning the influence of epigenetics on inherited diseases, one standard source says, "There is currently little evidence for epigenetic changes as *primary* causes of human hereditary disease."[49] If this is true about diseases with a known etiology, it seems imprudent to make global pronouncements about a behavioral trait like homosexuality based on epigenetics. Epigenetic changes may play a contributing role in development of sexual orientation, but they hardly seem determinative.

One of the over-arching difficulties regarding arguments that maternal stress can affect the epigenetics of a child in utero is related to the influence of maternal care. It is well established in animal-research that different amounts of maternal care can have a profound, lasting effect on stress-related behavior in animal offspring. Thus, when studying the maternal transmission of traits, this makes it difficult to parse the effects of epigenetic mechanisms transferred in utero versus outcomes which are the result of maternal care or abuse.[50] In other words, it is difficult to know if some traits are the result of epigenetic changes inherited in utero or if the traits are the result of maternal care (or lack thereof) after the child is born. In regards to homosexuality, if epigenetic marks are discovered which are

[49] Strachan and Read, *Human Molecular Genetics*, 4th ed., 513
[50] Eric J. Nestler, "Transgenerational Epigenetic Contributions to Stress Hormones: Fact or Fiction?," *PLOS Biology* 14.3 (March 25, 2016):5.

conclusively associated with expression of the trait, it may be difficult to determine if the epi-marks were established prenatally or postnatally.

Analysis of Ngun's Data

The data presented by Ngun[51] in October, 2015 has received a fair amount of criticism from others in the research community. The fact that the report does not appear headed to publication is further confirmation of a rather lukewarm reception to his findings. The most glaring problem with the study is its size: the sample is tiny. Ed Yong of *The Atlantic* comments on this weakness in the Ngun paper and says, "The field of epigenetics is littered with the corpses of statistically underpowered studies like these, which simply lack the numbers to produce reliable, reproducible results."[52] Furthermore, remember that the team split their sample into two sets: One was a "training set" whose data they used to build their algorithm, and a "testing set" whose data they used to verify it. While this is standard practice in research, Ed Yong says the result here is to weaken further this

[51] Ngun is himself a homosexual. He received his PhD. In December, 2012, writing on the molecular mechanisms underlying sexual differentiation in the brain. Ngun claimed he was not afraid of critiques of his work and said, "Trust me, I've had to deal with a lot worse as someone who grew up gay and an outsider. Dealing with critiques about my work are nothing compared to dealing with people telling me I'm going to hell." October 9, 2015, accessed August 10, 2016, http://vizbang.tumblr.com/post/130817769270/a-brief-digression-from-pretty-pictures.

[52] Ed Yong, "No, Scientists Have Not Found the 'Gay Gene,'" *The Atlantic* October 10, 2015, accessed July 15, 2016, http://www.theatlantic.com/science/archive/2015/10/no-scientists-have-not-found-the-gay-gene/410059/.

underpowered study and says, "But splitting the sample means that the study goes from underpowered to *really* underpowered."[53]

Andrew Gelman, a statistician at Columbia University, claimed the Ngun study inaccurately presented results as statistically significant. Gelman roundly critiqued Ngun's methodology and said, "Now let me say right here that I think the whole training/test-set idea has serious limitations, especially when you're working with n=47."[54] Gelman also added, "In general it seems like you're asking for trouble when you start publicizing technical claims without supplying the accompanying evidence."[55] Ngun himself acknowledged that the study was underpowered in social media, but blamed his small sample on lack of funding and said, "Yes, we were underpowered. The reality is we had basically no funding. . . . the sample size was not what we wanted. But do I hold out for some impossible ideal or do I work with what I have? I chose the latter."[56] This seems like a bad case of special pleading. Essentially, Ngun is saying, "I know that in research it is important to have a good sample size. I couldn't afford that because I had no funding. But I still want you to take my

---

[53] Ibid. Emphasis in original. Sten Linnarsson, professor of Molecular Systems biology at the Karolinska Institute in Sweden (and no fan of conservatives!), tweeted about the Ngun study, "This is terrible science in so many ways I lost count." October 8, 2015, @slinnarsson.
[54] Andrew Gelman, "Gay Gene Tabloid Hype Update," Statistical Modeling, Causal Inference, and Social Science, October 10, 2015, http://andrewgelman.com/2015/10/10/gay-gene-tabloid-hype-update/.
[55] Ibid.
[56] Tuck Ngun, "A Brief Digression from Pretty Pictures," October 9, 2015, accessed February 26, 2016, http://vizbang.tumblr.com/post/130817769270/a-brief-digression-from-pretty-pictures.

research seriously because I'm sincere and genuine and doing the best I can with what I have." Earnestness and a strong desire to do research cannot compensate for an underpowered study.

John Greally of the Albert Einstein College of Medicine also noted that deriving the DNA sample from saliva could lead to misleading results for the type of research the Ngun team was doing. The epigenetic marks in the saliva could be quite different from those in the brain, which is the area of Ngun's focus. Greally also pointed out that the team developed a "new" algorithm to evaluate the data and asks, "Why use a new algorithm to identify these predictive markers, did current approaches not yield any results?"[57] Greally also says the authors tried to give their report an air of plausibility by noting specific roles played by the genes they identified, subtly suggesting they may influence sexual orientation. The problem with epigenetics research in general and the Ngun study in particular is that while it may be plausible that epi-marks on these genes affect someone's sexual orientation, it is also possible that sexual orientation affects the epi-marks. In other words, what Ngun demonstrated was a correlation in his

[57] John Greally, "Over-Interpreted Epigenetics Study of the Week," October 9, 2015, http://epgntxeinstein.tumblr.com/post/130812695958/over-interpreted-epigenetics-study-of-the-week-2.

data between sexual orientation and the epi-marks. His data does not demonstrate which direction, if any, causation is moving.

Other scientists have suggested the Ngun data may be an example of a "false positive." Johnjoe McFadden, a molecular geneticist at the University of Surrey, said, "Studies that associate biomarkers with particular traits are notoriously prone to false positive results due to the tendency of these studies to find spurious associations that are down to sheer chance."[58]

Of some interest is that a paper Eric Vilain co-authored in the Spring of 2016 did not mention the findings of his own research team. Vilain and Ngun's 2014 paper was cited, but not their findings delivered in the Fall of 2015. In fact, the only data cited in the paper Vilain co-authored in 2016 was from a 2011 study of 34 identical twin pairs which revealed no support for the hypothesis that epigenetics influences male sexual-orientation![59]

There seem to be contradictory claims about how many regions of interest were discovered in the epigenome. For example, Michael Balter in

---

[58] Jessica Hamzelou, "Gay or Straight? Saliva Test Can Predict Male Sexual Orientation," *New Scientist,* October 8, 2015, accessed August 10, 2016, https://www.newscientist.com/article/dn28307-gay-or-straight-saliva-test-can-predict-male-sexual-orientation/. A false positive is a result that indicates a given condition or attribute is present when it is not.

[59] *See* J. Michael Bailey, Paul L. Vasey, Lisa M. Diamond, S. Marc Breedlove, Eric Vilain, and Marc Epprecht, "Sexual Orientation, Controversy and Science," *Psychological Science in the Public Interest* 17.2 (April 25, 2016): 77. The authors cite S. Bocklandt, W. Lin, M.E. Sehl, F.j. Sanchez, J.S. Horvath, and Eric Vilain, "Epigenetic Predictor of Age," *PLoS One* 6.6 (2011): e14821 http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0014821.

Science said Ngun had found "five regions" while Ngun's abstract refers to nine regions of interest. I suspect he started with nine regions of interest, but narrowed it down to a subset of five.[60]

Ngun's summary of research regarding genetic and biological factors associated with increased rates of homosexuality is also misleading. For example, he states, "Male sexual orientation has been linked to several genomic loci, with Xq28 and 8p12 being the most replicated."[61] Ngun is referring to Dean Hamer's 1993 claim to have found co-inherited genetic information among homosexual brothers in the gene-dense Xq28 region. Actually, several attempts to replicate Hamer's findings have resulted in conflicting data. In 2015, Alan Sanders and Michael Bailey claimed to have replicated Hamer's findings concerning homosexuality and the Xq28 region in addition to discovering an area of interest at chromosome region 8q12. First, Ngun incorrectly identifies the region as *8p12***,** when Sanders and Bailey's research clearly says 8q12.[62] But more importantly, Ngun overstates the strength of the findings regarding each of these regions, with

---

[60] Michael Balter in *Science* said Ngun had found "five regions" while Ngun's abstract refers to nine regions of interest. *See* Michael Balter, "Can Epigenetics Explain Homosexuality Puzzle?" *Science* 350. 6257 (October 9, 2015): 148.

[61] Tuck C. Ngun, W. Guo, N.M. Ghahramani, K. Purkayastha, D. Conn, F.J. Sanchez, S. Bocklandt, M. Zhang, C.M. Ramirez, M. Pellegrini, and Eric Vilain, "Program Number 95: A Novel Predictive Model of Sexual Orientation Using Epigenetic Markers," Paper Presented at the American Society of Human Genetics 2015 Annual Meeting, Balitmore, MD, October 8, 2015, accessed February 26, 2016, https://ep70.eventpilotadmin.com/web/page.php?page=IntHtml&project=ASHG15&id=150123267.

[62] The centromere divides each chromosome into two major regionis: the smaller "P" region and the larger "Q" region.

all research demonstrating that the findings to date in these areas have a very weak predictive power.

Ngun also says "each male pregnancy a woman has increases the chance that her next son will be homosexual by 33% (the fraternal birth order effect)."[63] But this oft-repeated claim has many weaknesses, including the fact that around half of all homosexual males have *no* brothers, data from other sources which questions the existence of the fraternal birth order effect altogether, and the fact that the fraternal birth order effect (if it exists) can only account for homosexuality in one out of every seven homosexual men.[64] Finally, Ngun makes a passing reference to early life androgen exposure being associated with more homosexuality among women. Apparently, he has women with Congenital Adrenal Hyperplasia in mind, but doesn't mention that most of these women have a heterosexual identity. Ngun implies these findings – Xq28 and 8q12, the fraternal birth order effect, and prenatal androgen exposure in women – have a stronger influence than the data actually allows. In fact, findings in each of these areas only demonstrate a lower level of correlation between certain variables and a higher level of self-reported same-sex attraction. And there

[63] Ngun, et al, "A Novel Predictive Model of Sexual Orientation Using Epigenetic Markers."
[64] James M. Cantor, Ray Blanchard, Andrew D. Paterson, and Anthony Bogaert, "How Many Gay Men Owe Their Sexual Orientation to Fraternal Birth Order?" *Archives of Sexual Behavior* 31.1 (February 2002): 63 – 71.

is still possibility that the Xq28 and fraternal birth order claims may yet be disproved. Ngun simply over-states the data to make his own claim sound more plausible.

Ngun's own response to the data and critiques of it is a bit confusing. On one hand, he said that the researchers want to replicate the study in a different group of twins and also determine whether the same marks are more common in gay men than in straight men in a large and diverse population.[65] But Ngun told another source he had quit the lab at the Geffen School of Medicine out of fear of how the data they were generating might be used. He said, "I don't believe in the censoring of knowledge, but given the potential for misuse of the information, it just didn't sit well with me."[66] Ngun seemed concerned that his research could be used by evil people or governments to identify homosexuals for the purpose of persecuting them. Yet, the weak and flawed nature of his findings make this fear sound quite unreasonable.

Epigenetics and Drug Addiction

As was noted above, one problem with Ngun's data is that he assumes the epigenetic tags he identified *caused* homosexuality, when it may in fact

[65] Sara Reardon, "Epigenetic Tags Linked to Homosexuality," *Nature* October 12, 2015, accessed August 10, 2016, http://www.nature.com/news/epigenetic-tags-linked-to-homosexuality-in-men-1.18530.
[66] Jessica Hamzelou, "Gay or Straight? Saliva Test Can Predict Male Sexual Orientation."

be the case that homosexuality caused a difference in the epigenetic tags. Research into alcoholism indicates this is at least a plausible scenario.

A robust body of evidence strongly indicates that alcoholism can lead to epigenetic changes which actually strengthen the alcoholism itself. An emerging model suggests that some genetic factors may predispose some people to alcoholism. These genetic factors are accentuated because expression of certain genes can be modified by excessive alcohol consumption – epigenetic changes can be induced by alcohol which modifies gene expression. These changes encourage further alcohol use and ultimately contribute to addiction.[67] One source says, "Although researchers are still piecing together all the details, findings to date suggest that epigenetic changes in gene expression induced by alcohol consumption may be the source or contributing factor in the brain pathology and adaptations in brain functioning associated with alcohol abuse and alcohol dependence and may contribute to alcohol relapse and craving."[68]

One group of researchers in 2012 studied the brains of 17 alcoholics along with a control group of 15. In their small sample, alcohol abuse was

[67] For a summary of the findings on alcoholism and epigenetics, see Harish R. Krishnan, Amul J. Sakharkar, Tara L. Teppen, Tiffani D.M. Berkel and Subhash C. Pandey, "The Epigenetic Landscape of Alcholism," *International Review of Neurobiology* 115 (2014): 75 – 116.
[68] U.S. Department of Health and Human Services: National Institute on Alcohol Abuse and Alcoholism, "Epigenetics – A New Frontier for Alcohol Research," *Alcohol Alert* 86 (November 1, 2013): 4.

associated with widespread changes in brain gene expression.[69] In other words, consumption of alcohol was associated with a change in the epigenome which subsequently altered the manner in which genes were expressed in the brain, probably contributing to alcoholism. With some caution, I suggest that we should at least be open to the possibility that something similar may occur in homosexuality. People who engage in homosexual behavior may find that the behavior itself is reinforced by epigenetic changes brought on by the homosexual behavior. In this way, the behavior may become compulsive and feel quite "natural."

Such an epigenetic mechanism may also partially explain the higher rate of the experience of childhood sexual abuse experienced by homosexuals, a trend admitted by most pro-homosexual authors.[70] We know the age of sexual debut, the context in which it occurred, and the age and gender of the person with whom the sexual debut occurred have a strong organizing effect on later sexual identity. It is at least plausible that in some cases of child abuse, the abuse itself initiates a cascade of epigenetic changes which contribute to same-sex attraction in adulthood.

---

[69] Igor Ponomarev, Shi Wang, Lingling Zhang, R. Adron Harris, and R. Dayne Mayfield, "Gene Coexpression Networks in Human Brain Identify Epigenetic Modifications in Alcohol Dependence," *The Journal of Neuroscience* 32.5 (February 1, 2012): 1884 – 1897. I acknowledge this is a small sample.

[70] Because this is commonly admitted, I find it odd that Ngun and Vilain take issue with the idea that childhood abuse can contribute to a later homosexual identity, a claim they call "discredited." Ngun and Vilain, "The Biological Basis of Human Sexual Orientation: Is There a Place for Epigenetics?," 172.

Such a hypothesis has limited explanatory power since the majority of homosexuals do not report being abused as children.

It is important to remember that epigenetics is a somewhat new sub-discipline within genetics, so the exact mechanisms of epigenetic function are still being unraveled at a broad level, much less in the specific case of homosexuality. The degree to which sexual behavior affects the epigenetic signals within a person are speculative at present, but it is at least plausible that participation in homosexual behavior may alter one's epigenetics.

Biblical-Theological Critique

When evaluating possible relationships between epigenetics and homosexuality from a Biblical-theological grid, two central ideas emerge: A rejection of biological determinism and the dangerous and compulsive nature of sin.

Within arguments about an epigenetic cause for homosexuality, there is in the background a strong and unmistakable message of biological determinism. It is a worldview which insists humans are not morally accountable agents made in the image of God. Instead, humans are viewed as biochemical automatons merely responding to stimuli. Within this

worldview, sex has no intrinsic value other than its necessity as the device to pass on DNA and continue the process of human evolution.[71]

These deterministic arguments often point to homosexuality in animals as proof homosexuality is a stable and recurring characteristic like many others. For example, in a 2013, Rice, Friberg, and Gavrilets discussed George Murray Levick's (1876 – 1956) observations of homosexual behavior among penguins in Antarctica during 1912 - 1913. Rice, et al seem to take great delight in the fact Levick recorded these specific observations in Greek because he found them so offensive that he didn't want the average person to read about them.[72] The not-so-subtle message is that we can laugh at such things now because we live in a more enlightened era. The authors then move on to argue that homosexual behavior in animals is some sort of evidence in favor of modern, tolerant attitudes about homosexuality.

What Rice, et al do not report is that it was not mere homosexuality among the penguins that offended Levick. He also recorded necrophilia, abuse of weak penguins by "gangs" of stronger penguins, the abuse of

[71] From an evolutionary perspective, homosexuality is not a favorable trait. This at least partly explains why some are arguing for an epigenetic origin of homosexuality as opposed to a genetic origin. Others have suggested male homosexuality is retained as a recessive trait which provides certain advantages to females and increases female fecundity.

[72] William R. Rice, Urban Friberg, and Sergey Gavrilets, "Homosexuality Via Canalized Sexual Development: A Testing Protocol For A New Epigenetic Model," *Bioessays* 35 (2013): 764.

female penguins by the same gangs of males, and the devouring of chicks by other penguins.[73] Certainly Rice and his colleagues do not think these other actions are morally neutral merely because they commonly occur in the animal kingdom.

Integrating epigenetics into a Christian anthropology is part of our view of the human body. Christians do not believe the body is evil, but we confess that humans are "fearfully and wonderfully made" (Ps. 139:14). The greatest affirmation that the human body is good is the Christian hope of the resurrection, wherein believers will receive a new and glorified body (Rom. 6:5; 1 Cor. 15:42 – 44). Furthermore, humans are made in the image of God (Gen. 1:26 – 28) and humans alone have the responsibility as God's image bearers to exercise stewardship over creation. At the same time, Christians also believe humans have a soul, the immaterial aspect of a human that transcends nature.[74] We are not *just* a body; we are a body-soul unity, the body and soul being connected at all points.[75] While the condition of our body certainly affects the way we feel about ourselves, we

---

[73] Robin McKie, "Sexual Depravity of Penguins that Antarctic Scientist Dared Not Reveal," *The Guardian,* June 9, 2012, accessed August 10, 2016, https://www.theguardian.com/world/2012/jun/09/sex-depravity-penguins-scott-antarctic/.

[74] There are basically two ideas about the origin of the soul which have been advocated by orthodox Christians. 1) Creationist: God creates each individual soul at the moment he gives it a body. 2) Traducianist: Each soul is derived, along with the body, from the parents.

[75] The view I am advocating for the relationship of the soul to the body is perhaps best described by Millard Erickson as "conditional unity." Consistent with this view is rejection of ideas claiming the human body is inherently evil. *See* Millard Erickson, *Christian Theology*, 2nd ed. (Grand Rapids: Baker Books, 1998), 554 – 557.

are more than a complex biochemical machine. Because humans have a soul, we can exercise volitional control over our response to appetites, desires and temptations. At the same time, the human body and the human genome have been negatively affected by a historic space-time Fall. The deleterious effects of sin can be found in both the genome and the epigenome. It should not surprise us if we discover things in these areas of research which contribute to various human sins, including homosexuality.

One of the tragedies of sin is that while most people know how to begin a particular sin or sinful habit, very rarely do we realize the third and fourth order consequences of sin. One of the most painful results of sin is that it is habit-forming. As was noted earlier, alcohol and drug abuse can negatively affect the epigenetics of particular genes in the brain and reinforce addiction. This is probably also related to brain plasticity – the manner in which neural pathways form and become reinforced and stronger each time we engage in various sins. In this way, we begin to live out the consequences of Jesus' warning, "Everyone who commits sin is a slave of sin" (John 8:34). D.A. Carson comments on John 8:34 and says sin "actively enslaves" and that, for Jesus, sin is "vicious slavery to moral failure, to rebellion against the God who has made us."[76] The slavery Jesus

[76] D. A. Carson, *The Gospel According to John* (Grand Rapids: Eerdmans, 1991), 350. Leon Morris comments on Jesus' statement here and says, "Those who sin are slaves to their sin whether they realize it

describes here is an inward condition from which one cannot flee and which is rooted in a wrong relationship with God. As George Beasely-Murray said, "Such a slave needs a redeemer!"[77]

At present, we can only speculate as to the degree that human volitional sin affects epigenetics and the subsequent expression of certain genes. Yet what we do know about epigenetics is consistent with the idea that sin is indeed *slavish*. Participation in sexual sin actively enslaves one to further indulgence in the sin, an indulgence which may feel freeing, but is actually a deeper progression into bondage. At the same time, we must not rush to a hurried conclusion that says everyone experiencing same-sex attraction does so because of choices they have made which have altered their epigenome. While some people have certainly contributed to the strength of their same-sex attraction by their behavior, others experience same-sex attraction for reasons which elude us. The consistent Biblical witness is that regardless of the source of sexual temptation, the appropriate expression for sex is only within heterosexual and monogamous marriage.

---

or not. This means they cannot break away from their sin. For that they need a power greater than their own." Leon Morris, *The New International Commentary on the New Testament*: *The Gospel According to John*, rev. (Grand Rapids; Eerdmans, 1995), 407.

[77] George R. Beasley-Murray, *The Word Biblical Commentary*, vol. 36, *John* (Waco, TX: Word Books, 1987), 134.

Summary

Epigenetics is a burgeoning area of research with fascinating discoveries about how genes are expressed. Though touted by the media, Ngun's research is flawed with a small sample size, a confusing use of algorithms developed by the team itself, and an imprudent assumption that the epigenetic changes they observed caused homosexuality without considering the opposite hypothesis – homosexuality may have caused the changes. Furthermore, other research has not found the types of connections Ngun claims. Christians can expect other claims regarding homosexuality and epigenetics to emerge in the coming years. A rejection of biological determinism combined with a robust understanding of the manner in which sin actively enslaves will help interpret the data in a manner consistent with Christian ethics.

Bibliography

Bailey, J. Michael, Paul L. Vasey, Lisa M. Diamond, S. Marc Breedlove, Eric Vilain, and Marc Epprecht, "Sexual Orientation, Controversy and Science." *Psychological Science in the Public Interest* 17.2 (April 25, 2016): 45 – 101.

Beasley-Murray, George R. *The Word Biblical Commentary,* vol. 36, *John*. Waco, TX: Word Books, 1987.

Bianconi, Eva, Allison Piovesan, Federica Facchin, Alina Beraudi, Raffaella Casadei, Flavia Frabetti, Lorenza Vitale, Maria Chiara Pelleri, Simone Tassani, Francesco Piva, Soledad Perez-Amodio, Pierluigi Strippoli, & Silvia Canaider. "An Estimation of the Number of Cells in the Human Body," *Annals of Human Biology* 40.6 (July 2013): 463 – 471.

Bocklandt, S., W. Lin, M.E. Sehl, F.j. Sanchez, J.S. Horvath, and Eric Vilain. "Epigenetic Predictor of Age." *PLoS One* 6.6 (2011): e14821 http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0014821.

Cantor, James M., Ray Blanchard, Andrew D. Paterson, and Anthony Bogaert. "How Many Gay Men Owe Their Sexual Orientation to Fraternal Birth Order?" *Archives of Sexual Behavior* 31.1 (February 2002): 63 – 71.

Carey, Nessa. *The Epigenetics Revolution*: *How Modern Biology is Rewriting Our Understanding of Genetics, Disease, and Inheritance*. New York: Columbia University Press, 2012.

Carson, D. A. *The Gospel According to John*. Grand Rapids: Eerdmans, 1991.

Dobyns, William B., Susan L. Christian, and Soma Das. "Introduction to Genetics," in *Swaiman's Pediatric Neurology*, 5th ed., vol. 1, *Principles and Practice*. Kenneth Swaiman, Stephen Ashwal, Donna M. Ferriero, and Nina Schor, eds. New York: Elsevier, 2012.

Downer, Joanna. “Backgrounder: Epigenetics and Imprinted Genes.” Accessed April 24, 2014, http://www.hopkinsmedicine.org/press/2002/november/epigenetics.htm

Erickson, Millard. *Christian Theology*, 2nd ed. Grand Rapids: Baker Books, 1998.

Franzek, Ernst J., Niels Sprangers, A. Cecile J. W. Janssens, Cornelia M. Van Duijn, & Ben J. M. Van De Wetering. “Prenatal Exposure to the 1944–45 Dutch ‘Hunger Winter’ and Addiction Later in Life.” *Addiction* 103 (2008):433 438.

Krishnan, Harish R., Amul J. Sakharkar, Tara L. Teppen, Tiffani D.M. Berkel and Subhash C. Pandey. “The Epigenetic Landscape of Alcoholism.” *International Review of Neurobiology* 115 (2014): 75 – 116.

McCarthy, Margaret M. “Estradiol and the Developing Brain.” *Physiological Review* 88.1 (January 2008): 91 – 124.

Morris, Leon. *The New International Commentary on the New Testament*: *The Gospel According to John*, rev. Grand Rapids; Eerdmans, 1995.

Nestler, Eric J. “Transgenerational Epigenetic Contributions to Stress Hormones: Fact or Fiction?” *PLOS Biology* 14.3 (March 25, 2016):1 - 7.

Ngun, Tuck. “A Brief Digression from Pretty Pictures.” October 9, 2015, accessed February 26, 2016, http://vizbang.tumblr.com/post/130817769270/a-brief-digression-from-pretty-pictures.

Ngun, Tuck C., W. Guo, N. M. Ghahramani, K. Purkayastha, D. Conn, F. J. Sanchez, S. Bocklandt, M. Zhang, C. M. Ramirez, M. Pellegrini, Eric Vilain, "Program Number 95: A novel predictive model of sexual orientation using epigenetic markers." A Paper Delivered October 8, 2015 at the Annual Meeting of The American Society of Human Genetics, accessed February 26, 2016, https://ep70.eventpilotadmin.com/web/page.php?page=IntHtml&project=ASHG15&id=150123267.

Ngun, Tuck and Eric Vilain. "The Biological Basis of Human Sexual Orientation: Is There a Role for Epigenetics?" *The Epigenetic Shaping of Sociosexual Interactions: From Plants to Humans* 86 (2014):167 – 184.

Peters, Ted. *Playing God? Genetic Determinism and Human Freedom*. New York: Routledge, 1997.

Ponomarev, Igor, Shi Wang, Lingling Zhang, R. Adron Harris, and R. Dayne Mayfield. "Gene Coexpression Networks in Human Brain Identify Epigenetic Modifications in Alcohol Dependence." *The Journal of Neuroscience* 32.5 (February 1, 2012): 1884 – 1897.

Ravelli, A. C., J.H. van der Meulen, R. P. Michels, C. Osmond, D. J. Barker, C.N. Hales, et al. "Glucose Tolerance in Adults after Prenatal Exposure to Famine." *Lancet* 351 (1998): 173–77.

Rice, William R., Urban Friberg, and Sergey Gavrilets, "Homosexuality As A Consequence of Epigenetically Canalized Sexual Development." *The Quarterly Review of Biology* 87.4 (December 2012): 343 – 368.

Richards, Sabrina. "Can Epigenetics Explain Homosexuality?," *The Scientist* January 1, 2013; accessed August 9, 2016, http://www.the-scientist.com/?articles.view/articleNo/33773/title/Can-Epigenetics-Explain-Homosexuality-/.

Robertson, Sally. "What is DNA Methylation?," accessed July 14, 2016, www.news-medical.net/life-sciences/What-is-DNA-Methylation.aspx.

Rodgers, Ali B., Christopher P. Morgan, N. Adrian Leu, and Tracy L. Bale. "Transgenerational Epigenetic Programming Via Sperm microRNA Recapitulates Effects of Paternal Stress." *Proceedings of the National Academy of Science* 112.44 (November 3, 2015): 13699-13704.

Roseboom, T. J., R. C. Painter, A.F. van Abeelen, M.V. Veenendaal, S.R. deRooij. "Hungry in the Womb: What are the Consequences? Lessons from the Dutch Famine." *Maturitas* 70.2 (October 2011): 141 – 145.

Roseboom, T., S. de Rooij, R. Paiinter. "The Dutch Famine and Its Long Term Consequences for Adult Health." *Early Human Development* 82.8 (August 2006): 485 – 491.

Schulz, Laura C. "The Dutch Hunger Winter and the Developmental Origins of Disease." *Proceedings of the National Academy of Science* 107.39 (September 28, 2010): 16757 -- 16758.

St. Clair, David, Mingqing Xu, Peng Wang, Yaqin Yu, Yourong Fang, Feng Zhang, Xiaoying Zheng, Niufan Gu, Guoyin Feng, Pak Sham, Lin He. "Rates of Adult Schizophrenia Following Prenatal Exposure to the Chinese Famine of 1959-1961." *Journal of the American Medical Association* 294.5 (August 3, 2005): 557 – 562.

Strachan, Tom and Andrew Read, *Human Molecular Genetics*, 4th ed. New York: Garland Science, 2011.

Susser, E.S., R. Neugebauer, H.W. Hoek, A.S. Brown, S. Lin, D. Labovitz, et al. "Schizophrenia after prenatal famine. Further Evidence." *Archives of General Psychiatry* 53 (1996): 25 -31.

Susser, E. S. and S. P. Lin. "Schizophrenia after Prenatal Exposure to the Dutch Hunger Winter of 1944–45." *Archives of General Psychiatry* 49 (1992): 983–988.

U.S. Department of Health and Human Services: National Institute on Alcohol Abuse and Alcoholism. "Epigenetics – A New Frontier for Alcohol Research." *Alcohol Alert* 86 (November 1, 2013): 1 -7.

Wang, Yu, Scott P. Kallgren, Bharat D. Reddy, Karen Kuntz, Luis López Maury, James Thompson, Stephen Watt, Chun Ma, Haitong Hou , Yang Shi, John R. Yates III , Jürg Bähler, Matthew J. O'Connell , and Songtao Jia. "Histone H3 Lysine 14 Acetylation Is Required for Activation of a DNA Damage Checkpoint in Fission Yeast," *The Journal of Biological Chemistry* 287.6 (February, 2012): 4386 – 4393.

Wedekind, Claus and Dustin Penn, "MHC Genes, Body Odours, and Odour Preferences," *Nephrology, Dialysis, and Transplantation* 15.9 (2000): 1269 – 1271.

Yang, Z., W. Zhao, X. Zhang, R. Mu, Y. Zhai, L. Kong, C. Chen. "Impact of Famine During pregnancy and Infancy on Health in Adulthood." *Obstetrics Review* Supplement 1 (March, 2008): 95 – 99.

Yong, Ed. "No, Scientists Have Not Found the 'Gay Gene.'" *The Atlantic* October 10, 2015, accessed July 15, 2016, http://www.theatlantic.com/science/archive/2015/10/no-scientists-have-not-found-the-gay-gene/410059/.