

2-2012

Epigenetics: A Progressive View

Kimberly Gustafson

Johnson & Wales University - Providence, kag405@students.jwu.edu

Follow this and additional works at: https://scholarsarchive.jwu.edu/ac_symposium



Part of the [Arts and Humanities Commons](#)

Repository Citation

Gustafson, Kimberly, "Epigenetics: A Progressive View" (2012). *Academic Symposium of Undergraduate Scholarship*. 15.
https://scholarsarchive.jwu.edu/ac_symposium/15

This Research Paper is brought to you for free and open access by the College of Arts & Sciences at ScholarsArchive@JWU. It has been accepted for inclusion in Academic Symposium of Undergraduate Scholarship by an authorized administrator of ScholarsArchive@JWU. For more information, please contact jcastel@jwu.edu.

Project Abstract

This research report examines the field of epigenetics in three areas through secondary research. A progressive viewpoint of the field is taken. The historical perspective of epigenetics is examined, followed by the chemical and biochemical reactions involved, and finally an analysis of the field of epigenetics as it relates to Nutrigenomics and the health and well being of the population is conducted.

Epigenetics: A Progressive View

Introduction

Within the organism, all processes are controlled through genetic information. However, what makes genes “tick” has been an unanswered question for centuries. How does gene expression take place? Why are certain genes heritable, while others are not? Why is it that two siblings from the same parents look so different from each other? Is disease heritable? And how is it that what we eat can play such an important role in our own personal health and the health of our future offspring?

Conrad Waddington originally proposed the term “epigenetics”. From his original definition, the field of epigenetics took flight. Throughout history, our knowledge of what the true definition of epigenetics is has blossomed. Scientists have not only gained a true understanding of the term, but also of the chemical and biochemical reactions that it is wrapped around. Today, epigenetics is being used in the field of nutrition to provide a greater appreciation of how environmental stimuli interact in the human body.

Part I: The History of Epigenetics

The face of epigenetics today is deeply rooted in the work of original geneticists. In the mid nineteenth century, Gregor Mendel conducted his famous experiments on phenotypes of peas. His research revealed that certain phenotypic traits are inherited from parent genes, while others are silenced. Following his work, Edmund Beecher Wilson, a professor at Columbia University in the zoology department, researched the role of chromosomes in sex determination and heredity. His research paralleled the work of Mendel in regards to the display of specific phenotypes, but he concluded that this was chromosome-related (Babbitt 49). Thomas Hunt Morgan, a student of Wilson's, would later conduct a series of experiments on the *Drosophila Melanogaster*, a fruit fly that commonly has red eyes but is sometimes born with white eyes. He revealed evidence that genes are physical and located on chromosomes (Morgan 120). Morgan tied together the link between inheritance of traits and chromosomes by revealing the physical inheritance of chromosomes that produce a specific phenotype based on gene silencing, as observed by Mendel and Wilson. Up until this time, geneticists' focus on research was simply gene expression with regards to phenotype. They failed to answer the question of why these expressions were occurring.

Conrad Waddington considered these geneticists' discoveries to be naïve, as they did not focus on why these genetic variations were occurring, simply that they were (Jablonka and Lamb 83). Waddington is famous for coining the term "epigenetics," derived from the Greek stem words of *epi* meaning "upon" and *genetics* meaning "involving genes," or studying "beyond the gene" (Jablonka and Lamb 83). He defined the term as "the study of causal mechanisms by which the genes of the genotype bring about phenotypic effects" (Haig 1). He conceptualized the idea of the "genetic landscape," a landscape featuring various hills and pathways that could potentially sway the direction of cellular differentiation. The landscape depicts the ability of the phenotype to change based on an outside stimulus, as rolling hills direct a ball in a specific direction (Goldberg 635). He was the first to connect

genetics to canalization, or the end result of the developmental progress over a period of time based on natural selection (Jablonka and Lamb 85). His idea evolved into a “gene centered neo-Darwinian version of Darwinism,” shedding genetic light on the survival of the fittest and natural selection concepts of evolution that are discussed in the theory of Darwinism (Jablonka and Lamb 84). It was not previously noted that evolution might be related to heritable changes in the genotype. His focus was not only genetics, but also what the determinant of specific phenotypes was. Previously, genetics and developmental biology had been studied separately.

In his book, *Canalization of Development and the Inheritance of Acquired Characters*, Waddington explains his connection between genes and development by referencing two developmental biologists, Detlefsen and Duerden, both regarding the calluses on the feet of ostriches. Detlefsen hypothesizes that the calluses are due to the developmental response to an environmental stimulus, and Duerden concludes that the calluses appear in the embryo (Waddington). For the first time, a connection between development, the ability for genes to be permanently switched on and displayed due to certain favorable conditions, and the ability for said genes to be inherited in mammals was made (Jablonka and Lamb 85). Waddington’s theories and research explained why animals displayed certain phenotypes due to development that make them tolerable of their environment. However, he was naïve himself to think that the field of epigenetics could be so simply explained.

In 1958, scientist David L. Nanney introduced the term “epigenetic control systems,” theorizing that certain external stimuli are necessary to trigger a genetic response (Haig 2). He stressed the importance of the cytoplasm, specifically non-genetic material, in gene differentiation and inheritance. Apart from Waddington, he hypothesized that non-genetic material could also be inherited (Haig 2). The stimuli that change genes were his main area of study and he also hypothesized that genes have the ability to return to their original phenotype so there is no permanent loss of information (Haig 3). Nanney’s theories did not gain popularity in the years to come, and were almost left behind, as other

scientists focused less on the role of the cytoplasm and other external elements in gene expression and heredity. However, his ideas would be revisited in time (Haig 2).

From the late fifties to the early eighties, many ideas were considered in the development of the true definition of epigenetics and a definition that would encompass all aspects of the subject. In 1974, Løvtrup wrote *Epigenetics: a Treatise on Theoretical Biology*, focusing almost entirely on a developmental biology view of epigenetics (Jablonka and Lamb 87). A 1982 dictionary of biology defined epigenetics as “Pertaining to the interaction of genetic factors and the developmental processes through which the genotype is expressed with the phenotype” (Jablonka and Lamb 87). The problem with this definition was that it still insinuated that the phenotype was directly connected to genetic variation, an original idea of Waddington’s that scientists had since disproved. Scientists discredited Waddington’s original phenotypic approach because research deduced that phenotype and genetic variation were not directly related to each other. Genetic variation in the genotype was not necessarily linked to the emergence of one specific phenotype. Scientists determined that one genotype could produce various phenotypes (Jablonka and Lamb 85).

The most up to date definition of epigenetics was given by Keith D. Robertson in his 1996 book entitled *Epigenetic Mechanisms of Gene Regulation*. He writes that epigenetics is “the study of mitotically and/or meiotically heritable changes in gene function that cannot be explained by changes in the DNA sequence” (Jablonka and Lamb 88). His definition was developed because of new research regarding molecular mechanisms and heredity. The definition made it clear that non-genetic information was in fact heritable. It focuses more on the function of genes as well as non-genes and heredity and less on the phenotype. Current studies of epigenetics are focusing on DNA methylation and chromatin remodeling, alterations to the genes themselves but not to the DNA sequence (Jablonka and Lamb 88). Robin Holliday has conducted extensive research on cell memory and the role of heredity, such as the transfer of non-genetic material from parent to child, something he calls “nuclear” material

(Jablonka and Lamb 88). He has also created a new definition of epigenetics to include DNA interactions that control the activity of genes, DNA rearrangements in the immune system and “mitochondrial inheritance,” something that Nanney first brought to light (Jablonka and Lamb 89). The focus today is on non-genetic material that is inheritable and capable of influencing genotype, including chromatin remodeling and methylation patterns (Jablonka and Lamb 89).

The ever evolving field of epigenetics will change the way we look at evolution and heredity. It started as a mere connection between genotype, phenotype, and development and heredity. It evolved into the study of how genes interact with each other and their outside environment and the role of non-genetic influences on heredity. Currently, non-genetically heritable material is a field of intense study. Since it has recently been recognized as a true subject for research, it has the ability to gain even more importance in medicine, agriculture, and species conservation as we learn how different stimuli interact with genetic mechanisms (Jablonka and Lamb 88). It has come a long way since Waddington’s original definition, and today is much more complex than he originally defined, involving the study of specific chemical reactions that influence gene silencing and heredity.

Part II: Chemical Reactions

Waddington's original definition of epigenetics mainly focuses on phenotype, but not much research was done regarding specific genetic mechanisms. Today research has evolved and the specific understanding of deoxyribonucleic acid (DNA) synthesis and alterations to the DNA strand and DNA packaging are the main focus of the field of epigenetics. To conceptualize how epigenetics play a role in gene silencing, an elaborate understanding of DNA reproduction, or transcription and translation is necessary. In addition, Waddington originally noted that epigenetic phenotypic characteristics were heritable, as evidenced by the callosities on the feet of ostriches being translated to offspring. This can be further explained through epigenetic mechanisms.

The processes of transcription and translation result in protein synthesis for various uses throughout the organism. The first process, transcription, is "the process by which DNA directs the synthesis of hnRNA/mRNA molecules that carry the coded information needed for protein synthesis" (Stoker 749). The process of transcription is a multistep process, resulting in the formation of hnRNA. In the first step, the DNA double helix is partially unwound according to the required protein synthesis by the enzyme RNA polymerase. In the synthesis of ribonucleic acid (RNA), ribonucleotides are used. The difference between these and those nucleotides used for DNA synthesis is an oxygen molecule bonded to Carbon two of the ribose sugar. Free ribonucleotides align themselves within the unwound strand of DNA according to appropriate Watson-Crick base pairs. In RNA, the ribonucleotide uracil bonds with adenine rather than thymine, used in DNA synthesis. The enzyme RNA polymerase then assists to bind the free nucleotides from the three prime to five prime end. RNA polymerase is directed to "stop" transcribing once it reaches a specific signaling set of bases (Stoker 750). After the initial transcription phase occurs, what is called a "post-transcription process" can occur. In this process, mRNA (messenger RNA) is produced. In post-transcription, the "junk" RNA, or introns, are removed. A complex known as the spliceosome completes this process. The spliceosome is composed of snRNA molecules, or small

nuclear ribosomal molecules, and additional proteins. These smaller complexes are known as “snurps.” The “snurps” bond together to form the larger spliceosome molecule. The spliceosome is directed to recognize specific base sequences within the hnRNA and splice the intron from the exon. The exons, which code for genetic information, are then bonded together to create a strand of mRNA (Stoker 451-452). In order for the genetic message to be read properly, the introns must be removed (Stoker 453). Messenger RNA is the RNA that participates in the translation process.

Translation is “the process by which mRNA codons are deciphered and a particular protein molecule is synthesized” (Stoker 758). Protein synthesis occurs from the activity of the tRNA molecule as well as the ribosomal unit. The tRNA molecule is a RNA molecule that has been folded over upon itself due to intermolecular attraction, creating a “t” shape. It contains a hydroxyl group attached to the three prime end, which allows for an ester linkage with a highly energized amino acid. A covalent bond is formed, allowing the tRNA molecule to become “activated” (Stoker 759). The protein synthesis itself occurs within the ribosome, which is composed of rRNA as well as other proteins. They are either located on the rough endoplasmic reticulum just outside of the cell nucleus or free-floating in the cytoplasm (Stoker 758). The mRNA molecule enters the ribosome from the five prime to three prime direction in between the small and large subunits. Within the ribosome, the start codon, or group of three consecutive nucleotides, occupies the peptidyl, or P site. The start codon for protein synthesis containing the bases adenine, guanine, and uracil (AUG). At the P site, a tRNA molecule with the complementary anticodon attaches itself (Stoker 760). After this attachment has taken place, a second tRNA molecule attaches itself to the second codon set located in the A site. After the P site and A site have both been activated, the enzyme peptidyl transferase is activated and the two amino acids located on the tRNA molecules are linked to form a dipeptide (Stoker 761). A shift within the ribosome occurs because the tRNA molecule in the P site is stripped of its amino acid and the mRNA strand is allowed to move over. The second tRNA molecule with the started dipeptide chain shifts to the P site and another

anticodon from a separate tRNA molecule is attached at the now vacant A site. The process, known as translocation or an acyl transfer reaction, repeats itself to form a tripeptide, and then a polypeptide. The process is ended by stop codons UAA, UAG, or UGA (Stoker 762). Following the end of translation, a hydrolysis reaction occurs in order to cleave the polypeptide from the tRNA (Stoker 762). These processes of protein synthesis require genetic inheritance in order to determine the correct sequences and messages.

Mammalian genes are inherited through sex cells, which contain one copy of the father's chromosomes and one copy of the mother's. In other non-sex cells, one copy of the father's chromosomes is retained and one copy of the mother's to make up twenty-two pairs of chromosomes. Single gene inheritance follows the patterns identified by Mendel. In autosomal dominant inheritance, the gene is expressed on one of the sex chromosomes, either X or Y. Just one copy of the gene is required to be expressed, so the offspring have a fifty percent chance of inheriting the gene and being affected by it. In autosomal recessive inheritance, on the other hand, the offspring must inherit two of the genes in order to be affected. If they only inherit one of the genes, they are simply a carrier for the gene. In this case, the offspring has a twenty five percent chance of inheriting two of the genes. In X-linked inheritance, either dominant or recessive, the gene is only present on the X chromosome. In the case of X-linked recessive, generally only males are affected because they only have one X chromosome, and therefore do not have another to compensate for the gene, whereas women have more than one X chromosome. In inheritance, the combination of chromosomes from the female and male parents are combined to create a completely new non-sex cell with genes from both of the parents ("Inheritance Patterns"). However, certain inherited genes are capable of being turned on or off according to epigenetic mechanisms.

Epigenetic mechanisms include DNA methylation, histone modification, chromatin remodeling, and RNA interference (Champagne and Curly 1). These processes are able to affect whether a gene is able to be read, turned on, or not read, turned off. In the process of DNA methylation, a methyl group undergoes an addition reaction and is bonded to either the fifth Carbon molecule on the cytosine pyrimidine base or Nitrogen number six on the nucleic base adenine purine (Bird 7). This addition reaction is capable of altering gene expression patterns so there is no need to continuously send signals to remind a gene of its task, or lack thereof. This process can also be described as “gene silencing” (Bird 2). The main sites for methylation on the gene are CpG islands, which contain a large amount of CpG sites. A CpG site is a cytosine nucleotide next to a guanine nucleotide (Bird 9). There are two types of DNA methylation. Maintenance methylation occurs in order to preserve the methylation patterns after DNA is replicated. This process requires the use of the enzyme DNA methyltransferase DNMT1. DNMT1 will methylate the copied strands of DNA according to where the methylation occurs on the parent DNA strand. Therefore, different generations of cells will have the same methylation patterns (Bird 7-8). However, maintenance methylation is not practical in all applications. In the process of reproduction and development of an embryo, genes need to remain undifferentiated for developmental purposes. Genes become demethylated prior to implantation in order for non-specific cells to be generated (Bollati and Bacarelli 107). In de novo methylation, methylation of the DNA strand occurs in the embryo during development (Bird 8). De novo methylation requires the use of the enzyme methyltransferase DNMT3A and DNMT3B. This methylation process sheds light on how non-genetic material is inherited. Studies have been conducted in an attempt to provide evidence that the enzyme methyltransferase is not necessarily required to direct DNA methylation, but that RNA may be directing the process. However, these studies have not identified any conclusive information (Bird 9). Epigenetic mechanisms occur outside of the DNA molecule itself, as well.

The packaging of DNA itself can also undergo transformation. In histone remodeling, the histone proteins can either undergo acetylation, methylation, phosphorylation, glycosylation, or ADP-ribosylation (Bollati and Baccarelli 105). In the case of acetylation, an increased acetylation process will result in increased gene transcription, whereas less acetylation will result in a decreased rate of transcription. The acetylation process involves the lysine residues on the terminal end of Histone 3 (Bollati and Baccarelli 106). The histone and DNA molecule are tightly held together by opposite charge attraction. The tail of the histone is positively charged, whereas the DNA molecule has an overall negative charge, creating a dense coil. While the two are densely packed together, DNA cannot be transcribed. When an acetyl group is added, the tail of the histone becomes more negatively charged, causing the two similar charges to repel. In this case, the DNA becomes exposed and is able to be transcribed (Champagne and Curly 1). Therefore, increased acetylation results in increased gene expression because the DNA is exposed (Champagne and Curly 2). These processes can either be increased or decreased by the enzymes histone acetyl transferase and histone deacetylase, respectively (Vignalli, et al 1899). Another mechanism for histone modification is performed by an ATP-dependent complex, in which case ATP is used in a hydrolysis reaction to disrupt the bonds within the histone as well as the DNA. Histone modification can result in what is referred to as chromatin remodeling. Since the histones as well as the DNA are being changed due to either an addition reaction or hydrolysis, the chromatin itself is also being altered (Vignalli, et al 1899). These chemical reactions play a large role in whether or not DNA is accessible to copying, and therefore if the reactions do not take place, the gene is silenced (Champagne and Curly 1).

RNAi, or RNA interference, is the final epigenetic mechanism that plays a role in whether genes are displayed or silenced. In this process, the dsRNA molecule promotes gene silencing. DsRNA is made up of small RNA molecules, siRNA, or small interfering RNA, and miRNA, or microRNA. These two types of RNA are cleaved apart by the enzyme Dicer and the siRNA molecules are then able to be bound in the

RISC complex, or RNA-induced silencing complex (Hannon 245-6). These complexes bind to mRNA molecules to decrease protein synthesis. This can, in turn, increase or decrease activity in other pathways. This occurs when RISC identifies complimentary base pairing in mRNA and cleaves it according to a specific nucleotide sequence (Hannon 248). If the RISC complex decreases activity, the gene will not be able to assist in the synthesis of proteins and will become silenced. These chemical reactions are often influenced by environmental factors.

One particular genetic trait that has been studied as a potential result of environmentally influenced epigenetic mechanisms is asthma. Research has shown that the history of asthma within families is capable of changing, indicating that it may be the result of environmental factors. Many studies have been conducted regarding the results of prenatal exposure to environmental tobacco smoke (ETS) and respiratory problems. In those infants who were exposed to ETS, the airway structure is compromised resulting in an increased distance between alveolar attachments versus the normal airway structure of infants who were not prenatally exposed to ETS. This resulted in sudden infant death (Miller and Ho). In particular, the Columbia Center for Children's Environmental Health revealed evidence that at two years of age, children who were prenatally exposed to ETS and PAH, or atmospheric pollutants such as coal and tar or foods cooked by grilling, had an increased rate of breathing difficulty. These modifications result specifically in the Th gene and mRNA, resulting in histone modifications. Asthma is also a result of oxidation reactions that may increase activity of the enzyme acetyltransferase, causing an increased rate of acetyl group addition reactions to histones resulting in chromatin remodeling. This alteration occurs specifically on the "proinflammatory gene IL-1B promoter nucleosome," causing inflammation of the airway that is characteristic of those with asthma (Miller and Ho). Epigenetic mechanisms have also been used to prevent genetically inherited diseases that are a result of environmental stimulus.

One study was conducted using mice to determine if obesity is an inherited trait and, if so, if it is preventable. Studies revealed that children born to parents who underwent bariatric weight loss surgery were less likely to be obese. Also, maternal obesity has been proven to result in offspring with increased body weight and altered metabolism. This is due to the developmental environment of the mother during fetal development. In this particular study, obese rats were fed a methyl-supplementation during the course of pregnancy. The resulting offspring underwent hypermethylation resulting in genetic silencing of the obesity trait, and experienced normal body weight versus the offspring of those parents who were not supplied a methylated diet (Waterland, et al 1373-4). In this case, environmentally influenced epigenetic mechanisms were capable of being altered through another epigenetic chemical reaction.

The growing understanding of epigenetic mechanisms brings understanding to gene silencing. Environmental influences on epigenetic mechanisms are more clearly understood and more easily prevented. The field also has the ability to prevent and even cure certain diseases that are environmentally influenced, as seen in the obesity study. From the original research conducted by Conrad Waddington about environmental influences on the callused feet of ostriches to today, a greater understanding of the chemical and genetic processes has moved the field of epigenetics forward.

One of the most important aspects of environmental influences on epigenetic mechanisms is nutrition. Human ingestion of food, an environmental factor, has the ability to influence epigenetic mechanisms, and, in turn, is capable of improving the health of individuals or adding to existing or preexisting health concerns.

Part III: Nutrigenomics

The nutrients in food are capable of interacting with genes and altering the genetic environment. Just as environmental factors such as exposure to environmental tobacco smoke (ETS) can change gene transcription, in this case resulting in the onset of asthma in children, nutrients are also capable of silencing or activating genes (Mahan, Escott-Stamp, and Raymond 153). In the field of epigenetics, it is well known that environmental factors have an influence on epigenetic mechanisms. However, diet plays a key role in determining the development and inheritance of certain diet-related diseases that plague our society today such as obesity, diabetes, and metabolic disorders.

Nutrigenomics is defined as “the study of the influence of specific environmental factors on changes in the expression of particular genes” (Mahan, Escott-Stamp, and Raymond 148). But, more specifically, Krussmann, Krause, and Siffert define nutrigenomics as “how the body responds to diet at the genomic scale” (38). Food is capable of changing gene expression because of its bioactive components (Mahan, Escott-Stamp, and Raymond 154). Nuclear receptors are activated by the nutrients found in foods and are then responsible for an increase or decrease in gene transcription (Afman and Muller 572). After activation, these receptors are able to bind to specific patterns of nucleotides, resulting in altered gene expression, as explained by the epigenetic mechanisms (Mahan, Escott-Stamp, and Raymond 154). This process is key in organs where metabolism takes place, particularly the small intestine, adipocytes, and the liver. In addition to nucleotide bonding, nutrients also play a role in modifying chromatin, a very important epigenetic mechanism and a large component of transcription (Afman and Muller 572). It is evident that epigenetic mechanisms result in altered gene expression, however, it is important to develop a knowledge of how these processes are affected by environmental aspects such as nutrient intake. The field of nutrigenomics will be applied in concert with knowledge of epigenetic mechanisms particularly in medicine and treatment of disease.

Nutrigenomics can be used in the treatment of metabolic disorders, including obesity, which is a huge concern for the United States currently and a growing concern for the rest of the world (Afman and Muller 570). No two human bodies are alike, and therefore each individual requires different amounts of nutrients in order for the body and genetic mechanisms to function properly, specifically in metabolic reactions (Mahan, Escott-Stump, and Raymond 153). Personalized diets can be developed for the health of individuals based on genetic testing to reveal specific epigenetic markers (Afman and Muller 570). These personalized diets will result in a shift away from recommendations for nutrients that are currently being utilized by nutrition professionals based on age and gender. The shift will be toward recommendations built upon specific genetic make up (Mahan, Escott-Stump, and Raymond 153). Information obtained from genetic testing can be invaluable in the treatment of disease through a recommended diet containing specific nutrients. However, as with any scientific field, ethical issues are certainly a concern.

The revealing of such important health-related information through genetic testing has many individuals deeply concerned. There is a growing concern that people who are predisposed to certain diseases or disorders could lose health insurance and employment opportunities. Those with certain metabolic disorders, for example, could be considered “high risk” patients, and therefore not supported by many health insurance companies. Also, some could lose employment or not obtain employment based on lack of capabilities to complete certain jobs due to genetic make-up if employment agencies are enlightened to specific genetic markers. This, in general, brings up the concern of others having access to patients specific genetic information (Mahan, Escott-Stump, and Raymond 159). However, to protect patients against such discrimination and to quell fears that certain individuals may have, the Genetic Information Discrimination Act was passed in November of 2009 to make discrimination based on genetic information illegal in the United States (Mahan, Escott-Stump, and Raymond 159). One stipulation to this Act is that it does not necessarily cover epigenetic mechanisms, a subgroup of

genetics. It does not entirely prevent discrimination of individuals based on specific epigenetic markers that may reveal susceptibility to disease or disorder. In addition, genetic testing is an expensive process, and therefore there is a concern that only the wealthy will be able to afford to be tested and benefit. However, as it becomes more widely practiced, the cost could potentially go down (Mahan, Escott-Stump, and Raymond 159).

Nutritionists, Dieticians, and those studying in the field of nutrition today, need to be aware of the important role that diet has on genetics. They need to have the knowledge of how epigenetic mechanisms work and be able to read and interpret genetic information in order to treat people with proper bioactive foods for their specific genetic make up (Mahan, Escott-Stump, and Raymond 153). In the future, it is very possible that recommendations will be tailored to the individual, rather than just generalizing and estimating nutrient needs based on age and gender. The scientific fields are constantly growing and revealing new information, so it is essential that nutritional caretakers are able to keep up and understand the growing knowledge of the human body. Also, in some aspects, treatment with food and specific nutrients may become more beneficial than treatment through pharmacology. In certain instances nutrients can have numerous different roles in the body, whereas prescription drugs only target one issue (Afman and Muller 571). This information greatly increases the importance of the job of the Dietician and others involved in the nutrition field, so an understanding of epigenetic concepts and nutrient interactions in the body is imperative. A strong knowledge of epigenetic mechanisms, genetics, and nutrient roles in the human body can help to interrupt the heritability of obesity, reduce the occurrence of obesity-related diseases such as cardiovascular disease and diabetes, and decrease the instance of metabolic related disease that are such a huge concern in the United States today. The field of epigenetics is capable of building a more healthy society.

Conclusions

Epigenetics has traveled quite a way since Conrad Waddington originally defined the word. Waddington not only coined the word “epigenetics,” but he also first introduced other scientists to the phenomenon of gene expression and encouraged them to investigate why these occurrences were happening. Since then, the field has evolved dramatically. Scientists and health professionals have developed a deeper understanding of genetics through intensive research.

The understanding of various epigenetic mechanisms has helped to identify how and why gene silencing occurs. Understanding the chemistry behind gene expression will even further develop scientists’ understanding of epigenetic mechanisms. Most important in the field today is the growing knowledge of environmental influences which result in gene expression or silencing and have the ability to affect certain aspects of human health. With such substantial health concerns for today and future generations, a greater knowledge of how to treat disease is necessary. Epigenetic mechanisms provide this knowledge. Particularly in the field of nutrition, the role that food plays on genetic expression and DNA synthesis is crucial. Food, in conjunction with genetic testing for epigenetic markers, has the ability to build a healthier community, if not even develop a cure for the health concerns of the United States population.

Conrad Waddington originally proposed the idea of epigenetics based on observed physical characteristics in various animals and Darwinistic theories, but today many others are applying this knowledge and deeper understanding to various fields to improve the health and well being of the human race.

Works Cited

- Afman, Lydia and Michael Muller. "Nutrigenomics: From Molecular Nutrition to Prevention of Disease." *Journal of the American Dietetic Association*. (2006) 106:4. 569-75.
- Babbitt, Gregory A. "Chromatin Evolving." *American Scientist* (Jan/Feb 2011) 99.1: 48-55.
- Bird, Adrian. "DNA methylation patterns and epigenetic memory." *Genes and Development Cold Spring Harbor Laboratory Press* (2002) 16:6: 6-20.
- Bollati, V. and A. Baccarelli. "Environmental epigenetics" *Heredity* (2010) 105: 105-12.
- Champagne, Francis A. and James P. Curly. "Epigenetic mechanisms mediating the long-term effects of maternal care on development." *Neuroscience and Behavioral Reviews* (March 2007) 1-8.
- Goldberg, Aaron D., Allis, C. David, Bernstein, Emily. "Epigenetics: A Landscape Takes Shape." *Cell*. (23. Feb. 2007) 128: 635-638.
- Hannon, Gregory J. "RNA Interference." *Nature* (July 2002) 418: 224-51.
- "Inheritance Patterns." *University of Vermont Gene Primer*. <http://www.uvm.edu>
- Haig, D. "The (Dual) Origin of Epigenetics." *Cold Spring Harbor Symposia on Quantitative Biology*. (2004) LXIX: 1-3.
- Jablonka, Eva, and Lamb, Marion J. "The Changing Concept of Epigenetics." *Annals of the New York Academy of Sciences*. (2002) 981: 82-96. *Academic Search Complete*. Web. 6 Jan. 2011.
- Kusmann, Martin, Lutz Krause, and Winefried Siffert. "Nutrigenomics: where are we with genetic and epigenetic markers for disposition and susceptibility." *Nutrition Reviews*. (2010) 68:1. 38-47.
- Mahan, Kathleen L, Sylvia Escott-Stump, and Janice L Raymond. Krause's Food and Nutrition Care Process. Elsevier Saunders. St Louis, MO. 2012. "Clinical: Nutritional Genomics." 148-59.

Miller, Rachel L and Shuk-mei Ho. "Environmental Epigenetics and Asthma". *American Journal of Respiratory and Critical Care Medicine* (March 2008) 117:6: 567-73.

Morgan, T.H. "Sex Limited Inheritance in Drosophila." *Science* (1910) 32: 120-22.

Stoker, Stephen H. "General, Organic, and Biological Chemistry." (2012) *Brookes/Cole, Cengage Learning*: Belmont, CA. 748-762.

Vignalli, Marissa, Ahmed H. Hassand, Kristen E. Neely, and Jerry L. Workman. "ATP-Dependent Chromatin-Remodeling Complexes." *Molecular and Cellular Biology* (March 2000) 20:6: 1899-1910.

Waddington, C.H. *Canalization of Development and the Inheritance of Acquired Characters*. Zoological and Strangeways Laboratories. *Nature* (14 Nov 1942) 381: 563-64.