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Epigenetics a Decolonizing Science

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Abstract

Epigenetics is the study of gene expression that does not entail alterations to the actual DNA. Decolonization is a theoretical and political movement that seeks to deconstruct colonial institutions and ideologies and reconstruct new and balanced approaches that accept and respect Indigenous worldviews. This project studies the decolonizing potential of epigenetics. Using genealogy as the method, the study establishes a long history of reductionist and deterministic thought that shaped the study of genetic science. Particular instances like thrift gene theory are explored to highlight how genetic explanations have been detrimental to the health and wellbeing of Indigenous people and illustrate the need for decolonization. The conclusion encourages the use of epigenetics as a decolonizing science that can be applied to Indigenous community-based research projects.

Keywords

Epigenetics, Decolonization, Indigenous, Genetics, Thrift Gene, Two-Eyed Seeing.

Summary for Lay Audience

Epigenetics is an emergent and promising science that studies changes in gene expression. Epigenetics manages genes like turning on and off a light switch without altering the actual DNA makeup of the gene. Epigenetics is a mechanism like a volume dial that regulates our body in growth, amplifying or decreasing gene production. When that regulation is compromised, there can be very negative health effects.

Decolonization is a political and theoretical movement that tries to undo negative aspects of settler society that continue to affect Indigenous peoples. Decolonization is about centering Indigenous ideologies and worldviews and bringing them into a place of balance with Western knowledge systems. This project explores how epigenetics can be used as a tool for decolonization. I explore the history of genetic science, particularly its reductionistic and deterministic tendencies.

Through two case studies, I explore the potential of epigenetics as an applied decolonizing science. The first is the case of the Dutch famine, where I look at epigenetics and its promises to the application of health. The second case study examines thrift gene theory as a harmful and reductionist explanation for Indigenous diabetes. It looks at residential schools and positions social, environmental and multiple other factors, whereas the thrift gene offers a singular gene as a complete explanation. The conclusion compares these two case studies, emphasizing how epigenetics allows for a decolonized approach. It encourages the application of epigenetics to community-based research projects.

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To everyone named here,

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Preface

I am a Maliseet First Nations scholar. Like the majority of Canada's Indigenous people today, I grew up off-reserve. My father, who himself grew up on the St Marys' First Nations reserve in Fredericton, New Brunswick, made efforts to teach me about Indigenous values and histories, challenging the normative accounts that I learnt at school. It was important to my father that we knew about this aspect of our identities and for a long time, that was my only sense of Indigeneity. Early on, there weren't many avenues for exploring Indigeneity. I recall a particular instance about a desire to learn an Indigenous language instead of French in elementary school being met with shock and even disgust. The need for a student to learn French was prioritized over any recognition of an Indigenous student's attempt to explore that part of their identity. I always had a dichotomous sense of self, of being both deeply steeped and rooted from the Western tradition, but I also had this other sense of being an outsider. I am Maliseet and I yearned to have a deeper understanding and connection to those roots.

When I got to university, I had the opportunity to take Indigenous courses and meet other Indigenous students, and I seized those opportunities to engage closely with Indigenous ideologies and scholarship. In my last year of my undergraduate studies, I took a course taught by Dr Regna Darnell. I was quite linear in my thought process till that point. Dr Darnell challenged us to think rhizomatically, to embrace the chaos and acknowledge the complexity of contexts, trying to understand the same issue from a multiple set of perspectives instead of pinning it down to one explanation. The very significant aspect of thinking rhizomatically with Regna was her non-hierarchical approach that taught a respect for a diversity of opinions. Without that challenge to my thought processes, I doubt I could have ever arrived at the conclusions and ideas that form this thesis.

Regna's approach taught me to combine multiple worldviews and ideologies in a non-hierarchical manner. Mi'kmaw Elders Albert and Murdena Marshall together with Debbie Martin and Cheryl Bartlett, developed two-eye seeing as a decolonizing non-hierarchical

framework. “Two-eyed seeing is a theoretical framework that honours and accepts diverse ways of knowing” (Martin 2012:24).

With the Marshalls being from Eskasoni First Nation on Cape Breton island in Nova Scotia, the theory spoke very personally to me when I first encountered it. I was looking for a way to bridge my two worlds together when I found the theory. With my mother’s family being from Cape Breton island and a large component of them still living there, Indigenous theory about rectifying worldviews that had come from the place of my mother’s family, it felt much like the reflection of my own personal journey. Mi’kmaw also having proximate ideologies to Maliseet, mattered in this closeness I felt to the concept of two-eyed seeing.

Equipped with this framework, I set out to apply this approach to Indigenous health concerns. My attempt is to present epigenetics in the framework of two-eyed seeing to develop it as a decolonizing tool, both theoretically and in direct application. The work is intended for multiple audiences, as a theoretical contribution to academics and social theorists who are decolonizing scholars, while it hopes to support application through community use of epigenetic research as a means of decolonizing, deconstructing and rebalancing. The writing style may appear repetitive in places, which in itself is a decolonizing approach as Indigenous story telling traditions retell the same story, with each repetition presenting the story in a new way and providing new lessons to the listener. This thesis features a repetitive style that is reflective of that storytelling tradition and I would encourage readers to reflect on repetition as a moment of reflection, thinking about the new knowledge presented through each repetition.

Chapter 1 : Epigenetics a Decolonizing Science

1 Introduction

Epigenetics is a new and emergent science that studies changes in gene expression that do not involve alterations of the actual DNA sequence. Epigenetics is a mechanism by which chemical modifications alter the expression of a gene or genes, without altering molecular gene itself. Imagine genes are like turn dials, like that which controls the volume on a radio. Epigenetics is the mechanism that controls these dials. Chemical modifications change these dials either increasing or decreasing the expression of genes, higher volume meaning increased gene expression.

Decolonization is a multifaceted theoretical movement and political process that seeks to dismantle or undo the forces of colonialism. In one regard, decolonization involves deconstructing harmful and enduring colonial structures and ideologies. In another, decolonization involves the revitalization of Indigenous culture, knowledge systems, ceremonies and ideologies.

This thesis explores epigenetics, specifically highlighting how epigenetics represents a divergence from genetic science while simultaneously emphasizing the decolonizing potentiality of epigenetics. The following sections explore epigenetics and decolonization in much deeper detail.

1.1 What is Epigenetics?

1.1.1 Background

In 1903, Wilhelm Johannsen introduced the terms genotype and phenotype (Falk 2009), as an explanation of classical or Mendelian genetics. Genotype as term refers to the entirety of the genetic information that forms a gene. Phenotype describes the expressed or observable characteristic of the gene. A Punnett square is a tool used to model potential genotype combinations. Further they provide an excellent visual aid for explaining the difference between genotype and phenotype. Please refer to table 1 as an example of a punnet square.

Table 1: Punnet square used to illustrate possible genotype combinations when crossing two traits (B) and (g) (Source: Wade Paul).

	B	**g**
B	**BB**	**Bg**
g	**Bg**	**gg**

The genotype is comprised of two alleles that determine the phenotypic expression of the trait. In table 1 (B) and (g) represent individual alleles that correspond with a particular trait. For the purpose of this example, let (B) represent brown eyes and (g) green eyes. Additionally, brown eyes are the dominant trait and green eyes are what is known as a recessive trait. This means that if the genotype is heterozygous or comprised of two different alleles, the dominant trait is expressed. Recessive traits are only expressed when the genotype is homozygous, meaning when both alleles are the same. In the example above, the potential genotypes are (BB) (Bg) and (gg). In this example, the genotype (gg) would be expressed phenotypically as green eyes while (BB) and (Bg) would be expressed as brown eyes.

Epigenetics is the study of heritable changes in gene expression that do not involve alterations to the actual DNA sequence. In the context of genotype and phenotype, epigenetics is the study of the alternation of phenotypical expression of genes that do not alter the genotype. While this model is a wonderful explanatory model, it is important to note that it is also an overly simplified and reductionist explanation. This model is suggestive of a one-gene one-trait model. It indicates that phenotypical expressions such as eye colour are associated to a single gene. This is true of some characteristics which

are referred to as being Mendelian traits such as hair colour. However, most traits have been shown to be influenced by multiple genes.

The genotype-phenotype model was used to explain how genes were understood to function. In 1943, DNA (deoxyribonucleic acid) was identified as being the biochemical structure which carries the biological unit known as genes. This was cemented further by James D Watson and Francis Crick in 1953, when they identified the double helix structure of DNA (Keller 2000). With this, biologists were convinced that genes were nothing more than molecular sequences located in DNA.

1.1.2 Introduction to Epigenetics

Human beings are eukaryotes, which means the cells that constitute bodies have a membrane enclosed nucleus. The cells of our bodies have nuclei, or a separate portion isolated from the rest of the cell. Eukaryotic organisms such as humans protect their precious DNA by safeguarding it within the nucleus. All cells have the same DNA, however each cell holds its own individual copy of DNA within its nucleus. Epigenetics regulates the expression of the DNA. It is through this regulatory system that cells with identical DNA develop to serve a multitude of functions. The cells that comprise your fingers nails hold the exact same DNA as the cells in your heart. It is epigenetics regulation

Our DNA is structured as a double helix in which two identical strands running in opposite directions stack on top of each in what resembles a spiraling staircase (Blossey 2017). If stretched out, the length of the double helix in each cell is approximately two meters or six and half feet long (Piovesan et al. 2019). Our bodies are composed of trillions of cells. In every cell, the long strands of DNA must package or condense themselves in order to fit inside the nuclei of our cells.

To achieve this, DNA condenses itself into a fibre-like structure called chromatin. The process begins with the double helix of DNA wrapping itself around a group of proteins called histones forming a nucleosome. Spooled around groups of histones, DNA resembles beads threaded on a spring. The nucleosomes then bind together, causing the

DNA to fold on itself. As a rope is made from twisting or braiding together thinner fibers like string, the long strands of DNA are condensed into tightly compacted chromatin.

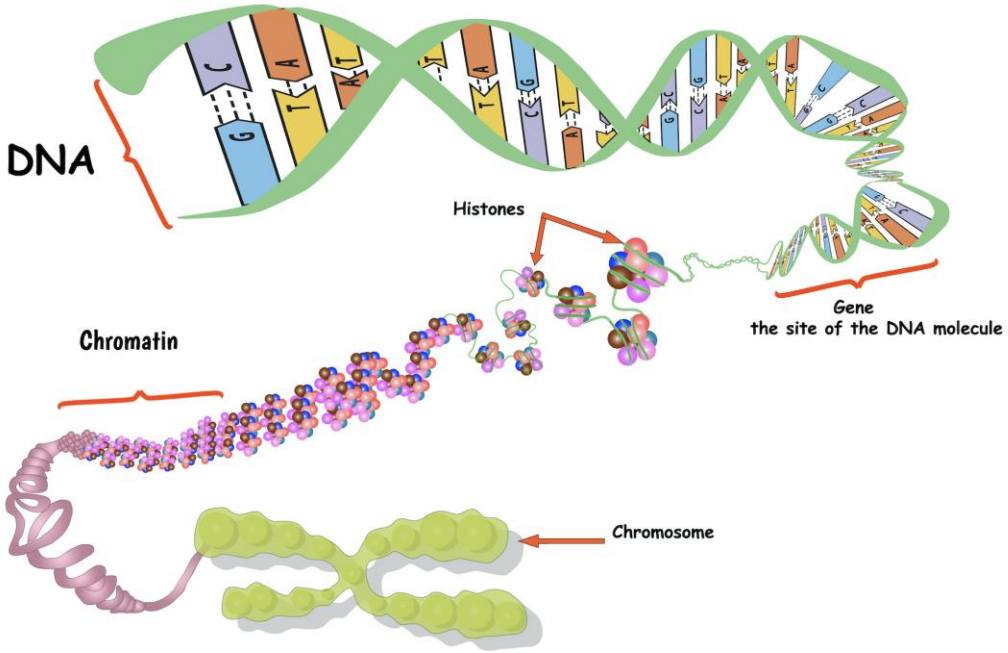

Figure 1: Diagram of DNA being packaged into chromatin (Source: From Zvitality [artist]. G*enome in the structure of DNA*. Shutterstock ID: 1162309045).

Through a variety of biochemical mechanisms such as methylation and histone modifications, epigenetic changes alter the structure of chromatin. Alterations to the structure of chromatin play a significant role in the regulation of gene expression. In the formation of nucleosomes, little histone tails remain exposed. These histone tails play an important role in DNA packaging as well as gene transcription and expression (Erler et al. 2014). Chemical signals bind to these exposed histone tails prompting chromatin to restructure itself. As chromatin undergoes a restructuring, particular segments become more or less accessible for transcription.

“In order to express genes, the double helix needs to be opened” (Blossey 2017:4). In order for a gene to be expressed, it must first be transcribed. DNA is transcribed involving a process known as RNA polymerase (Blossey 2017). In this process the

double helix is opened like the undoing of a zipper. The DNA is then read and made into RNA, an opposite of DNA similar to a negative of a photograph. Chromatin is structured in such a way that some segments of DNA are made readily available for transcription and are thus active. While other segments are too tightly compacted and not able to be transcribed therefore making these genes inactive.

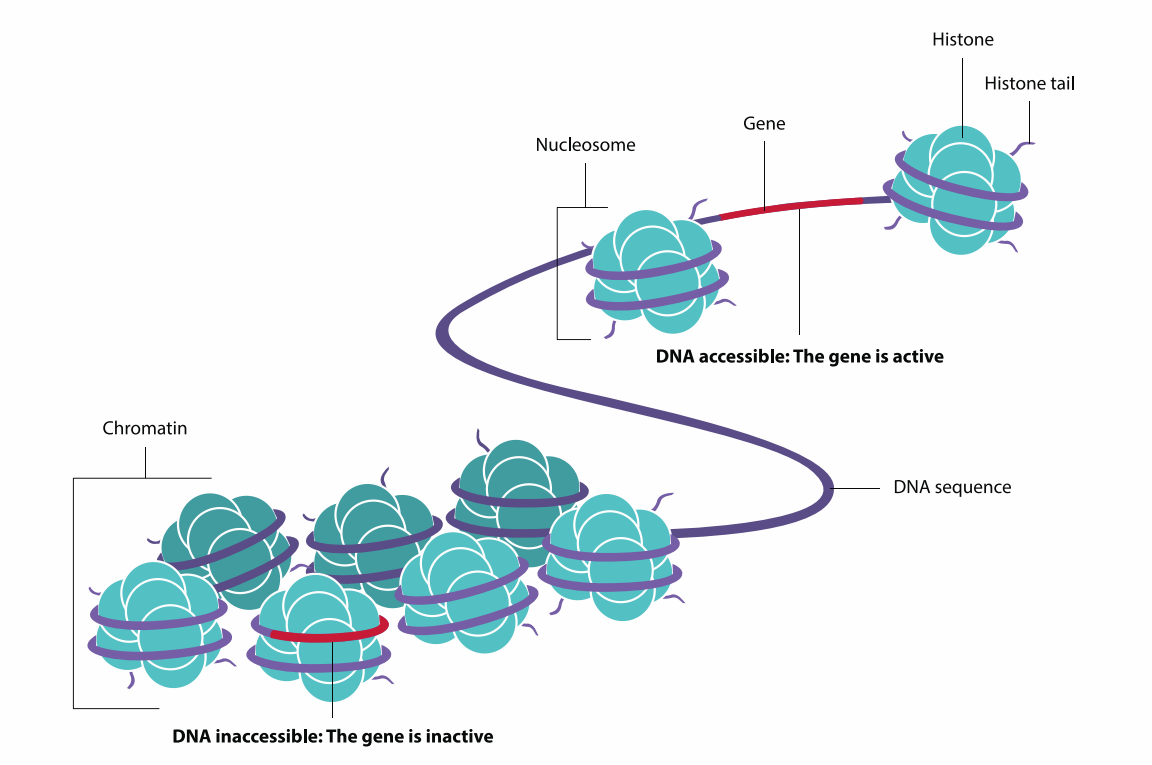

Figure 2: Diagram of chromatin displaying active and inactive gene segments (Source: From Mari-Leaf [artist]. Vector illustration of epigenetics. Shutterstock ID: 1232628649).

As a new and emergent field of study there is substantial optimism amongst researchers about potential applications of epigenetics. However, not everyone is convinced, and epigenetics does have its detractors. Currently the majority of epigenetics studies were conducted on animal test subjects (Chung et al. 2016) with limited data from human trails.

I was originally introduced to epigenetics by thesis advisor Gerald McKinley. Gerald is particularly fond of using studies in which researcher re-activate genes that have been

associated with tooth development in chickens, to illustrate the possibilities of epigenetics. In these studies, that activation of teeth in chickens causes their entire beak to develop completely differently. In turn the resulting heads of the altered chickens more closely resemble the head of an alligator (Harris et al. 2006; Bhullar et al. 2015). McKinley, particularly fond of epigenetic experiments, refers affectionately to the transformed chickens as 'evil chickens.'

One of the more well-known epigenetic studies by Weaver et al (2004), examined the epigenetic consequences of maternal grooming patterns in mice. Skepticism of epigenetics argue that "…rodent models translate poorly" (Chung et al. 2016:174). Lab based studies on rodents do not translate to animals in their own natural habitat that well. The lab allows for a level of control and isolation that cannot be mirrored in real world studies.

However, the optimism associated with epigenetics outweighs the detractors. Those who are optimistic think we should not wait for final results before exploring the consequences for human application. Their optimism is justified through epigenetics' explanatory power for the dynamics of human social order. Supporters like Evelyn Fox Keller have argued "there is little doubt that [epigenetics] discovery and its integration into mainstream genetics is indeed rocking the foundations of that science, and it is doing so in ways that have enormous implications for our conceptual framing of its core questions about heredity, development, and evolution" (2014:2423). Similarly, I also approach epigenetics with a comparable sense of optimism. I perceive epigenetics as having a revolutionary potential.

Epigenetic dysregulation, more particularly chromatin modifications through methylation has been associated with the development of cancer (Hessmann et al. 2017). "Disruption of the epigenetic machineries, either by mutation, deletion or the altered expression of any of their components, is known to provoke aberrant gene expression patterns that give rise to all typical cancer characteristics" (Rodríguez-Paredes and Esteller 2011:331). There is significant optimism that an understanding of the epigenetic mechanism that give rise to cancers also offers potential treatments options.

Epigenetic therapies have to potential to provide highly individualized or personalized medicines (Holder, Haque, and Skinner 2017). The development of cancerous tumors has been linked to epigenetic dysregulation. In the case of breast cancer hypermethylation of human tumor suppressor genes, effectively silencing them (Radpour et al. 2011). However, epigenetics does not entail alterations to the DNA and therefore to some extent is reversible. Utilizing this understanding of the epigenetic mechanism, researchers have suggested the ability to develop gene targeted treatments such as reverse methylate in the treatment for breast (Radpour et al. 2011) and pancreatic cancer (Hessmann et al. 2017). Epigenetic based therapies have the potential to revolutionize medicine by offering the potential to development personalized health care.

The notion of using epigenetics as a theoretical tool to connect distinct and sometimes opposing perspectives is not new. Epigenetic researchers Patrick McGowan and Moshe Szyf have advocated for use of epigenetics as theoretical tool to bridge diverse perspectives stating,

"Understanding the epigenetic consequences of social exposures stands not only to revolutionize medicine but also to transform social sciences and humanities as well. Epigenetics could serve as a bridge between the social sciences and the biological sciences, allowing a truly integrated understanding of human health and behavior" (McGowan and Szyf 2010: 71).

This is very similar to the position I argue throughout this thesis. However, I also argue that epigenetics as a decolonizing science has the potentiality to bridge Indigenous and Western scientific perspectives.

1.2 What is Decolonization?

Decolonization is a term that refers to on-going theoretical and political movements. Decolonization has been described as "…the intelligent, calculated, and active resistance to the forces of colonialism that perpetuate the subjugation and/or exploitation of our minds, bodies, and lands, and it is the ultimate purpose of overturning the colonial structure and realizing Indigenous liberation" (Linklater 2014:26). In this definition

decolonization is about intelligent, calculated resistance for the purpose of Indigenous liberation. Intelligent and calculated resistance recognizes the value of Western knowledge. As such decolonization does not represent a rejection of the entirety of Western knowledge.

While decolonization involves resistance is it is not a complete and total rejection. One of the foremost leading figures in the decolonization movement, Linda Tuhiwai Smith advocates for the decolonization without rejection of Western knowledge. In her pivotal *Decolonizing Methodologies* Smith states, "decolonization, however, does not mean and has not meant a total rejection of all theory or research or Western knowledge. Rather, it is about centering our concerns and world views and then coming to know and understand theory and research from our own perspectives and for our own purposes" (Smith, 1999:39). Smith promotes decolonization as actively approaching Western knowledge from an Indigenous perspective, and for Indigenous purposes.

Similarly, two-eyed seeing is a decolonizing theoretical framework developed by Mi'Kmaw elders Albert and Murdena Marshall. Likewise, the framework of two-eyed seeing advocates for decolonization over rejection. Albert and Murdena recognize that Indigenous communities can benefit from both Western and Indigenous knowledge systems. When we are able to seeing use both eyes "a new way of seeing the world has been created – one that respects the differences that each can offer" (Martin 2012:31). In this respect decolonization is about balancing or centering Indigenous perspectives. Two-eye seeing values the diversity of perspective the framework offers.

Beyond resisting colonial structures, decolonization entails the revitalization of Indigenous culture. In addition to resistance decolonization involves Indigenization. Conceptually, Indigenization represents a dismantling of knowledge systems that have been dominated by Western thought. Firstly, Indigenization envisions expanding the acceptance of systems of knowledge to include Indigenous ways of knowing. In this regard Indigenization describes a resurgence of Indigenous culture, practices and belief.

The process of Indigenization involves "constructing research programs that rebuild capacity for Indigenous intellectual institutions to determine their own intellectual

priorities and establishing local institutions to govern research projects in order to move beyond research collaboration with outsiders to community-led research projects" (Gaudry and Lorenz 2018:225). Indigenization attempts to rejuvenate and revitalize Indigenous concepts: Indigenous concepts such as those that conceive of humans as wholistic beings come to co-exist with Western traditions.

Additionally, Indigenization is a process of asserting self-control, and a reconstruction of power. Community-based research projects (CBRP) are emblematic of such instances in which Indigenous communities Indigenize. Smith, quoted earlier, advocated for the decolonization of Western knowledge. Rather than rejecting Western knowledge, Smith suggests that application and use of Western knowledge for Indigenous purposes is in itself decolonizing. In this respect CBRP are an example of Indigenizing and from the perspective of decolonization Western knowledge is put to use for Indigenous purposes.

In sum, decolonization describes an ongoing political moment and theoretical framework. It is the intelligent and calculated resistance to colonial structures. A decolonizing perspective does not seek to reject Western knowledge or supplant it with an Indigenous knowledge system. Rather it is a process of centering Indigenous perspectives and then coming to understand Western knowledge from a new Indigenized position. In arguing that epigenetics is a decolonizing science; I am claiming epigenetics has potentiality to resist the forces of colonialism and therefore that epigenetics Indigenizes Western knowledge.

1.3 Epigenetics Decolonizing Potentiality

Epigenetics flourished as science in the genomic era. Genomics is a branch of biology that is concerned with structure and function of the whole genome. This is in contrast with genetics which studies individual genes. As a branch of genomics, epigenetics is concerned with genome as whole. With genomics the gene is no longer isolated but a part of a much larger interconnected network. With Epigenetics this is furthered by the genome being placed within an environmental feedback loop. This environmental feedback loop is the epigenetic mechanism by which the environment interacts with DNA altering gene expression.

Epigenetics is decolonizing as it is resistant to colonial structures. Genetic thought and notions of the gene have long been associated with reductionist and deterministic styles of thought. *The Mismeasure of Man* is Stephen Jay Gould's seminal work and critique of biological determinism in which he links, "…biological determinism to some of the oldest issues and errors of our philosophical traditions – including reductionism" (1996:27). Throughout this work Gould explores the uses of bodily measures to rank and order individuals.

Gould attempts to deconstruct the reification of intelligence into a single number indicative of comparative rank such as IQ. Reification is "the propensity to convert an abstract concept (like intelligence) into a hard entity" (Gould 1996:27). Gould explores the deterministic and reductionist thought that became associated with intelligence through fields like craniology that attempted to reduce intelligence to its correlation with anthropometric measures of the head.

Further, Gould argues that, "…the use of these numbers to rank people in a single series of worthiness, invariably to find that oppressed and disadvantaged groups – races, classes or sexes – are innately inferior and deserve their status" (1996:57). This is illustrative of how colonial structures that promote, and advance reductionist and deterministic thought become harmful. From the standpoint of this thesis it is indicative of need to decolonize genetic thought.

Epigenetics provides active resistance to such colonial styles of thought. As an alternative to the colonial reductionism of an isolated gene, epigenetics establishes the gene as a social actor. This challenges the fundamental conception of the gene. Dislodging colonial conceptions of genetics and with it reorients aspects of Indigenous experience such as health and well-being.

Further, epigenetics holds a decolonizing potentiality in its ability to Indigenize. Epigenetics can bridge Western and Indigenous knowledge systems. As a decolonizing science, epigenetics has the potentially to see with both eyes. By this I mean epigenetics is more compatible with Indigenous perspectives of health and well-being.

Indigenous conceptions of health and well-being emphasise our wholistic nature. Indigenous conceptions hold that “We are physical, emotional, spiritual, mental beings interacting in multifaceted environments” (Hart, Straka, and Rowe 2017:335). Epigenetics implicates social stimuli and exposures as educing epigenetic alterations. As such epigenetics resists colonial explanation of health and well-being, alternatively suggesting the need for a wholistic approach.

Similarly, epigenetics allows for the centering of Indigenous knowledge systems such as the Haudenosaunee concept of seven generations of stewardship. The principle encourages individual to think about the consequences of their actions seven generations from now (O’Sullivan 2011). This concept pertains primarily to stewardship of mother earth and the responsibility to maintain her for seven generations. However, it also connects the actions of individuals today with the health and well-being of seven generations later. Epigenetic alterations have also been shown to persist through successive generations (Skinner 2015). As such epigenetics is decolonizing as it works to center Indigenous ways of knowing within a Western knowledge.

1.4 Methodology

Decolonization as theoretical framework is a tool to critique the Western world from an Indigenous perspective. Further, decolonization seeks to underscore and draw attention to pervasive power imbalances and ongoing colonial forces. As such, Michel Foucault’s concept of genealogy offers methodological underpinning for my exploration of epigenetics as a decolonizing science. Genealogy has been described as, “… a historical lens, but not of the conventional kind. It does not seek to identify causes and effects, to find an inherent truth about the past, or to expose ideas as smokescreens for what is really happening” (Murray 2018:355). As described genealogy is perhaps the perfect decolonizing methodology here.

Genealogy is the active resistance of the accepted accounts of history. Genealogy seeks to unearth truths that deconstruct the official accounts of history. In this sense genealogy represents a decolonizing approach to a historical study of genetics. As part of this thesis I trace a genealogy of genetic thought. I establish the lineage of deterministic and

reductionist thought that has persisted through various iterations of Western knowledge that produced genetic science. In illustrating epigenetics as a decolonizing science, I intend to highlight the ways epigenetics actively resist colonial styles of reductionist and deterministic thought.

Chapter 2, the first part of my genealogy, I explore styles of thought that pre-date and were direct predecessors of genetics. Exploring concepts such as generation, preformation and reproduction, I attempt to trace the lineage of thought that reduced human-ness to collection of isolated genes.

In the second part of genealogy, Chapter 3 I explore genetic thought from rediscovery of Mendel and the onset of classical genetics to the development of epigenetics. In this section I pay close attention to the language and metaphorical representation of the gene and genetic thought.

I follow-up my genealogy of genetic thought with two case studies in chapters 4 and 5. The first case study is one the Dutch Famine which occurred at the end of the Second World War. In this case study I explore extensive collection of research conducted on individuals exposed to the famine. I present epigenetic evidence of prolonged and varying negative health outcomes that are the result of epigenetics alterations caused by famine exposure.

Next is my second case study, this time exploring thrift gene theory. Thrift gene theory is colonial expiation for disproportionate rates of types two diabetes amongst Canada's Indigenous population. In this case study I use epigenetics to deconstruct thrift and decolonizing or dismantle harmful colonial structures such as thrift.

1.5 Conclusion

What is anthropological epigenetics and how does a study of its' decolonizing potential fit in the field of anthropology. Franz Boas is recognized as the founder of the Americanist Anthropological tradition. One of Boas' most impactful works his, *Changes in Bodily Form of Descendants of Immigrants*, in which he considers the effect environment has on the phenotypical expression of head forms through a comparative

analysis of the offspring of immigrants within family lines. Boas compared anthropometric measurements to demonstrate the significant role the environment plays in the process of development and the impact that environment change can manifest on physical form within as little as one generation. Boas described the rapid ability for these morphological features to change in response to environmental change such as immigrating from Europe to the United State – plasticity.

Working with the United State Census, Boas was able to collect anthropometric data including head width, height and weight of immigrants that he categorized as Bohemians, Slovaks and Hungarians, Poles, Hebrews, Sicilians, Neapolitans and Scotts. Utilizing comparative analysis Boas compared traits of offspring that were born in their "home" country with that off offspring born in America. As part of his work for the United State Census Boas collected anthropometric data including head width, height and weight of immigrants that he categorized as Bohemians, Slovaks and Hungarians, Poles, Hebrews, Sicilians, Neapolitans and Scotts. Boas applies a comparative statistical analysis to traits of offspring that were born in their "home" country with that off offspring born in America. One of Boas' more well-known findings was the change in head shape of immigrant offspring of Italian and Jewish immigrants. Indicative of epigenetic changes, Boas noted that the long head of the Sicilians became rounder in offspring. Alternatively, the round head of Hebrew's became longer with offspring.

With his work on immigrants arriving in New York, Boas established a direct connection between gene expression and the environment. This was not surprising as the previous year Boas had published his paradigm shifting *The Mind of Primitive Man,* in which he "underscored the importance of considering the effects of culture, environment, and history alongside racial or biological determinants." (Darnell 2018: 6). Boas was decades ahead of his contemporaries and it would take decades for an explanatory model such as epigenetics to develop. Boas had unique and advanced perception of the body and development as being highly plastic and subject to changes.

Boas advocated for the consideration of environmental effects alongside biological determinants. Decades later epigenetics provides the mechanism to explain the changes in

gene expressions observed by Boas. Boas established the four-field subdivision in anthropology as part of his wholistic approach. Epigenetics as science explores human behavior and development that requires a wholistic approach as advocated for by Boas. Far in advance of his contemporaries, Boas established a basic research foundation that has come to be mirrored by epigenetic styles of thought and approaches to human development. As such I view epigenetics as discussed in this thesis as representative of an anthropological pursuit in accordance with Boas' foundations of discipline.

Chapter 2 : Genealogy Part 1

2 Introduction

In *The Politics of Life Itself,* Nikolas Rose defined a style of thought as "a particular way of thinking, seeing and practicing" and further stating, "a style of thought is not just about a certain form of explanation, about what it is to explain, it is also about what there is to explain" (2007:12). In this sense, a style of thought is a way of seeing and knowing that is situated within a particular understanding that establishes the criteria of knowledge for both its creation and for evaluating subsequent claims such a style of thinking asserts as valid.

This chapter uses genealogy to explore what I describe as a genetics style of thought. My genealogical analysis traces the emergence of key theoretical concepts from the seventeenth to the present day that have been fundamental in shaping a genetic style of thought. I will present, analyze and challenge foundational concepts such as generation, taxonomy, species, genes, genomics and DNA. Through this analysis I aim to unsettle and destabilize accepted facts. In challenging these accepted 'norms', I hope readers are able to come to see them in a new light. I use this to reframe our understanding and perspective of current science and its practices. In particular, this genealogy explores a lineage of reductionistic and deterministic thought trends in genetics style of thought.

This chapter explores genetics as a style of thought that is reductionistic and deterministic and how a genealogy of genetics can be decolonizing.

Genetics is a style of thought in the sense that it offers a forum of explanation, but it also defines what there is to explain, the 'gene'. However, genetic styles of thought have been hampered by a pervasive legacy of deterministic and reductionist lines of thought. With my genealogy I intend to show that the gene as a single determining unit was the only thing a genetic style of thought was able to discover. Styles of thought of course do not emerge in a vacuum devoid of a history, but rather are the continuation of an ongoing history. Therefore, in order to critique a style of thought it is necessary to understand and appreciate that it is situated within a particular history of thought.

In this chapter, I attempt to establish a genealogy of genetic science as a style of thought. I aim to trace lineage of thought that has been rooted in reductionistic and deterministic styles of thought. In so doing I wish to highlight these concepts not a new or unique to genetic science, rather my intention is to illustrate the manner in which genetic science represents a persistent and ongoing process of colonization.

2.1 Decolonizing Genealogy

Genealogy is a historical research method originally developed in the works of Friedrich Nietzsche and then later popularized by Michel Foucault. Genealogical analysis is a historical investigative technique that challenges what is accepted, in so doing intrinsically offering a critique of the present status of knowledge. Borrowing the words of Deborah Cook, "genealogies seek 'to uncover the battles that gave birth to the world that we accept as natural, to make it questionable again, and to make it possible to fight over it once more'… unearthing the historical conditions that made possible phenomena that have become so familiar that we simply take them for granted, genealogy aims to defamiliarize them and distance us from them" (Cook 2018:9). Genealogies encourage the re-assessment and re-evaluation of discourses of knowledge, challenging and questioning official accounts, and how in modern society they effectively work to limit and subject people.

Decolonization is about resistance; genealogy is decolonizing because it exemplifies the type of resistance advocated for in decolonizing theory. Genealogy can be used for decolonization as it provides a framework for the critical analyses of the relationship between knowledge and power in modern society. Only by unearthing, exposing and revealing the structures that maintain the status quo (colonial forces), do we make it possible to challenge and overthrow them.

Genealogies encourage the re-assessment and re-evaluation of discourses of knowledge, challenging and questioning official accounts, and how in modern society they effectively work to limit and subject people. Genealogy displaces the primacy of the subject found in conventional history and targets discourse, reason, rationality and certainty.

Genealogical analyses aims to illuminate the contingency of what we take for granted, to disrupt and challenge the official account. Through the examination of histories, genealogies seek to uncover and reveal the contingency of what we take for granted. Genealogies seek to challenge, disrupt and displace what has come to be accepted as normal. In doing so, genealogical analyses encourage the active re-assessment and re-evaluation of the discourses of knowledge and power. Genealogy seeks not to eliminate history, rather it is rooted in re-establishing history. Genealogy does not seek to destroy, rather it seeks to uncover or make visible the conditions that made certain phenomena possible. In doing so, genealogy seeks to recast history and display bodies and structures as completely and totally imprinted by history.

Karen Bridget Murray describes the method of genealogical analyses, “the aim is to trace out lineages of knowledge claims, comprised of both language and practices, silences and audibility, and to ascertain the forms of power they produce, such as political subjectivities and territorial boundaries naturalized through them” (2018:355). The following sections, through a genealogy of genetic thought, seek to uncover the deterministic and reductionistic roots of genetics. This genealogy attempts to re-evaluate genetics as a discourse of knowledge.

2.2 A Genealogy of Genetic Thought

A style of thought is a particular way of thinking and reasoning. In this sense as style defines a specific method of understanding the world and explaining phenomena. Not only defining what is accepted as valid form of explanation, styles of thought also establish the boundaries of what there is to be explained. Genetic science blossomed from deterministic and reductionistic styles of thought.

The science of genetics and many of its cornerstone concepts such as the gene, seem so firmly established to us today. However, genetics science in our modern interpretation is a relatively recent science. Many consider the 1900 rediscovery of Gregor Mendel’s rules of inheritance as starting what we now regard as classical genetics. The history of genetics styles of thought often indicates this as the beginning. Although Mendel is now heralded as the father of genetics, he received little acclaim or acknowledgement for his

work during his lifetime. Mendel's famed study of the characteristic of plea plants that educed a paradigm shift, never actually achieved such magnitude with Mendel in 1865. Rather its major impact came three and a half decades later when it was spontaneously re-discovered by Hugo de Vries, Carl Correns and Erich von Tschermak. Even then it was not the three rediscovers that would use Mendel to launch genetics but rather William Bateson.

Gregor Mendel is a microcosm that illustrates the true complexity of the history of genetic styles of thought. In the words of François Jacob, "Mendel's case represents a good example of the impossibility of tracing a linear history of ideas, of finding the succession of stages that logic would have deliberately followed" (1974:208). This would require the endpoint being known in advance and it cannot be so in the process of discovery.

Genetics as a scientific discipline is relatively recent, only emerging as its own distinct discipline at the beginning of the twentieth century. The twentieth has aptly been dubbed that century of the gene by Evelyn Fox Keller, due to the central and impactful role the discipline played over that century. In 1909 Wilhelm Johannsen introduced the term 'gene' as in the words of Keller he, "wanted a new word so it that it might be free of the taint of preformationism" (2000:1–2). The gene has since established itself as one of genetics' cornerstone concepts. As a genealogy this section seeks to uncover the taint of preformation, to trace strands of deterministic and reductionist styles of thought as they percolate and permeate into genetic styles of thought and shape it in numerous ways.

2.3 Generation and Preformation

The foundations of genetics are rooted in its predecessors as far back as the seventeenth century, which perhaps explains its reductionist and deterministic tendencies. Prior to the seventeenth century, the most pervasive and dominant figure in scholarship was Aristotle (Fara 2009). Sixteenth century beliefs held that, "each mundane object, each plant and each animal can always be described as particular combination of matter and form." And with matter being comprised of the same four elements, "an object is thus characterized by form alone" (Jacob 1974:20). Aristotle's model of generation held that the mother's

menstrual blood provided the matter and the father provided the form with the sperm (Cobb 2006). In this model primacy is granted to the sperm that provides form to the matter therefore giving it its shape.

In 1651, William Harvey's *De Generation Animalium* questioned and attempted to explore the origins of life. In which he wrote '*Omnia vivum ex ovo*' meaning all life comes from an egg (Jacob 1974). In so doing Harvey presented a model of generation that opposed that of Aristotle. Harvey's model shifted primacy in generation away from the form providing sperm and to the egg. However, with both Aristotle and Harvey, the essence was understood to be unitary, and not collaborative. This was a long way off from thinking of the egg *and* the sperm as being co-contributors.

Further, until the seventeenth century, "spontaneous generation had been just as easy to explain as generation by seed, since it required the direct action of divine forces on matter" (Jacob 1974:53). Generation was only understood to be possible by divine intervention, as a spontaneous act of God. In other words, the creation of life required a divine force to implement the essence of life (soul) into matter to grant life.

In 1668, Francesco Redi sought to test if rotting flesh engendered flies. Redi's experiment was inspired by a reading of Homer's *Iliad.* Curiosity was sparked in Redi by Achilles' efforts to protect Patroclus from insects (Jacob 1974). In his experiment Redi observed that maggots do not spontaneously form on meat, rather they are born from eggs laid on the meat. This provided the necessary evidence prove that the generation of life was not spontaneous but rather life is engendered through the egg such as the generation of a plant from a seed. Which is to say, life was not spontaneously created, rather life is created or given by the egg. With this Redi reaffirmed Harvey's claim that all life come from the egg.

Styles of thought at this time were characterized by analogous or comparative systems. In the seventeenth century the distinction between beings and things was less well defined. These styles of thought allowed for analogies between the living beings and machines (Morgentaler 2000). In his 1628 *De motu cordis* (*Movement of the heart and blood in animals),* Harvey established the heart as a pump which singularly circulates blood

throughout the body (Fara 2009). However, Jacob argues that this is “an inversion of the orders of events” (1974:34). In a way that is illustrative of the need for further deconstruction, Jacob suggests that in actuality it is because the heart is a pump and as such abides by all the same laws of hydraulics, allowing for the analogous comparison.

As such preformation was introduced in 1669 by Dutch Naturalist Jan Swammerdam. Preformation theory believed that bodies existed as miniature versions of themselves fully formed adult size (Morgentaler 2000). In this sense children represented little more than miniatures of their fully formed adult self. Recall, that in Aristotle’s model of generation, primacy was granted to male semen for providing form. Thus, with preformation, the essence providing form for Aristotle exist pre-formed. Establishing the roots of deterministic models of thought and therefore possible explanations has continued to persist into genetics in the twentieth-first century.

Preformation brought a reaffirmed belief in the fixity of species that would persist into the eighteenth century. The great chain of being or, *Scala Naturae* was the belief that the living world was comprised of long continuous web of beings. This hierarchical notion suggests God, “…filled the world with a continuous, progressive presence of life, from the lowest to the highest, all distinct yet each adjacent to its neighbor” (Falk 2009:14). In establishing a hierarchical order of distinct life forms the great chain of being also establishes the foundations of the concept of species.

As the seventeenth century neared its conclusion, styles of thought began to reduce limits of validity to that which could be observed. As Jacob explains, “only what is visible enabled the universe to be understood, for although one sees a star, one does not touch it, or taste it, or hear it” (Jacob 1974:44). In this structure all that the senses are able to recognize are not considered equal, as greater validity is attached to what can be observed. Of which it was readily observed that animals developed a universal character or appearance that was consistently preserved the through succeeding generations.

Preformation enhanced and strengthened the notion of the fixity of species.

Through preformation, species were fixed and each generation resembled every other. Furthermore, “the permanence of species through succeeding generations ensured that the living world as it now appears was indeed an accurate reflection of what was laid down at the origin” (Jacob 1974:52). Consequently, it was no longer possible to conceive of generation as a lone isolated event. With this arose the concept of species and the continuity of form across generations

2.4 Reproduction

The term reproduction was originally used to denote the phenomena of animal regeneration, e.g., referring to animals like salamanders that can regrow a lost tail. The term gained a wider meaning in eighteenth century. In 1748 French biologist Georges Louis Leclerc de Buffon published his *Natural History of Animals*, in which he used the term to include animal generation (Jacob 1974). In this wider use, “…reproduction denoted repetition of conserved qualities in the process of embryogenesis” (Falk 2009:15). In this sense reproduction came to represent the mechanism by which species is preserved: the continuity of species existing in the continuity of form.

With the concept of species having been established, it became necessary to establish structures of organization. In 1735, Swedish botanist Carl Linnaeus published the first edition of his *Systema naturae*. Over ten subsequent editions Linnaeus would establish the foundations of taxonomy that have remained to today. Taxonomy and the concept of species arose at the end of the seventeenth and turn of the eighteenth century out of the need of naturalists to classify and order the world. For Linnaeus, to study an organism it was first necessary to observe and describe it. This meant, “…reducing a living being to its visible aspects and translating its shape, size, colour and movement into words” (Jacob 1974:45).

Linnaeus was originally a strong proponent of the fixity of species, believing, “there are as many species as there were different forms created by the Infinite Being at the beginning,” (Falk 2009:19). In this conception there are no new species and anything otherwise undocumented represents the discovery of a previously unknown species not the emergence of new one.

Linnaeus' search for God's divine order, established over the six-day Creation period, made the fixity of species most appealing to him. Buffon, however, was drawn to the conceptualization of a universe that afforded a level of flexibility and change. In 1759, an essay submitted to the Academy of Sciences at St. Petersburg shows a modification of Linnaeus' earlier position that was staunchly rooted in the fixity of species, stating that it was possible for the generation of a new species through the process of hybridization (Falk 2009). In dislodging the concept of the fixity of species, the essay paves way for the theories of Lamarck and Darwin that explored more flexibility and change.

Hybridization came to be used as a research tool to clarify taxonomic relations. For instance, Carl Friedrich von Gartner performed almost 13,000 hybridizations on various fruit plants (Falk 2009). The hybridist research tradition focused on the hereditary transmission of desired characteristics. Carl Friedrich von Gartner's experiments focused on specific traits rather than the organism as a whole. The hybridist research tradition focused on the transmission of hereditary characters, and Mendel's work, which was rooted in this tradition, provided the mathematical organization for the study of heredity.

This section ends the pre-Mendel genealogy of genetics, and the next chapter, which continues this genealogy, looks at the discourse of gene action with a focus on the gene as a unit, briefly examining the human genome project. The chapter's focus lies in how epigenetics is a decolonizing science with a dynamic quality that contrasts to the paradigm.

Chapter 3 : Genealogy Part 2

3 Introduction

This chapter continues the genealogy of genetic thought from the rediscovery of Mendel's work, tracing the onset of classical genetics till the development of epigenetics. The study of the gene in this chapter pays close attention to the language and metaphorical representation of the gene and genetic thought. Such an analysis will illustrate the reductionistic and deterministic roots of genetics and illuminate the decolonizing potential of epigenetics.

3.1 The Gene

Darwin's theory of evolution suggests that change occurs by the accumulation of little steps in a series of successive generations. Pangenesis was the mechanism by which Darwin explained this. According to pangenesis "each fragment of the body, each cell, produced a little germ of itself, or 'gemmule', that was sent to the germ cells and commissioned to reproduce the same fragment in the next generation"(Jacob 1974:206). Reproduction in successive generation relied on this notion of a hereditable unit that would get passed on.

In his 1892, *Das Keimplasma* or The Germ Plasm, August Weismann presented the germ plasm theory, which was a mechanism of inheritance. According to Weismann, Germ Plasm was a substance of a particular molecular composition and by its nature had the properties to reproduce traits in new generations. Weismann "assumed the existence of particulate, self-reproducing elements that determine the properties of an organism"(Keller 2000:16) . Fittingly, Weismann referred to these particulates as 'determinants'.

This model of reproduction speculated on the existence of a germ cell which was different in structure and function from regular or somatic cells (Jacob 1974). In this model, the hereditary trait or unit is carried exclusively in the germ cell. In doing so, the model isolates the hereditary material and mechanism in germ plasm, which is

emblematic of the deterministic mode of thought that made the emergence of a science of genetics possible.

In 1889, Hugo de Vries presented *Intracelluläre Pangenesis* as a theory of reproduction and generation. Hugo de Vries attempted to rectify evolution in germ plasm theory by proposing an alternative to the Darwinian hypothesis of gradual and persistent change (Falk 2009). Weismann observed that species were not gradually changing, rather remaining consistent for generations before sudden and distinct changes occurred. "Unlike fluctuations and gradual imperceptible changes, mutations are accessible to observation and experiment" (Jacob 1974:221).

Similar to Weismann, de Vries offered a model that assumed the existence of a particulate. For de Vries, that particulate was the "pangene". Pangenes were described by de Vries thus: "...the carriers of heredity were particles of a specific order: the characters of species are intendent of each other and their transmission calls for highly independent hereditary particles, the *pangenes*" (Falk 2009:45). In both instances, Weismann's "determinants" and de Vries's "pangenes" are clear precursors to the gene, evidence of a lineage of reductionism.

Looking for support to strengthen his theory, de Vries was involved in the 'rediscovery' of Mendel. With the rediscovery of Mendel, he revived his method for isolating and modeling statistical models of character traits. The experimental model set by Mendel allowed for the search of easily distinguishable individual traits. Mendel's method, focused on the isolation and prediction of individual traits, established a statistical and mathematical model for doing so.

Introduced to the rediscovery of Mendel through de Vries, William Bateson saw immediate potential in Mendel's model, particularly in its isolation of specific traits as individual hereditary units (Falk 2009). Bateson urged a widespread acceptance of Mendelism. The school of thought that synthesized Darwin's evolution and Mendel's mathematical model of heredity would be named genetics.

The gene has quickly come to be considered as the central point and locus of control for human development. Evelyn Fox Keller has described this style of thought as the '*discourse of gene action'*. Early genetic thought ascribed to the gene a position of ontological and causal priority as the gene was cast "as the basis of life" (Keller 1995). First generation geneticists spearheaded by T.H. Morgan, viewed the gene as having direct causal relations with traits. These first generation geneticists tasked themselves with tracking the patterns of transmission of genes (Keller 2014). In this early conception, it was believed that genes produced traits, and individuals were comprised of an assemblage of traits. It then followed that if traits made the individual, and genes made traits, therefore genes made the individual.

Early prioritizing of the gene inflated the role the gene plays in human development in a manner which promotes genetic determinism. In establishing ontological superiority from the onset, deterministic rhetoric has become so entrenched it is still common and visible in contemporary genetic discourse. In *Refiguring Life* (1995), Evelyn Fox Keller argues that through the discourse of gene action, foundational metaphors describing the gene as the book of life guided thought in genetics. These conceptual metaphors were pervasive throughout the 20th century. The metaphors of gene action were particularly resilient; though not the only conceptual tools, they became fundamental core tenets of genetics that were intrinsically resistant to conceptual change. With that said, it is important to remember that any proper consideration or understanding of a complex system requires a starting point. That is what the gene became to genetics, a launching point.

While the gene has proven itself to be an exceedingly fruitful launching place, understanding the complexity of human development requires expansion beyond the gene. The discourse of gene action, however, continued to maintain the ontological and causal priority of the gene, a position that would be further buttressed by the second generation of geneticists. The gene became firmly cemented as the basis of life. It logically follows then that to understand the gene meant to understand life. In 1932, the International Congress of Genetics put forth the core question of the discipline, 'how do genes produce their effect?' (Keller 1995: 14). In response, genetics researchers focused on the functional mechanisms of the gene. This question would set the direction and

primary objective for genetic research endeavours for years to follow. Here the gene has already been reduced into the determinative cause and effect category.

In 1940, George Beadle and Arthur Tatum, with their study of *Neurospora,* were able to put forth the explanation describing the *'chain of reaction'* through which genes produced their effects. This explanation came to be known as the one gene-one enzyme hypothesis (Keller 1995). The chain of reaction of the one gene-one enzyme maintained the core tenant of 'gene action', that held the gene as the '*biological blueprint'* and as the '*book of life'* (Landecker and Panofsky 2013). The hypothesis did not alter the preconception of the gene's role in development. Instead, it further secured the gene's position of developmental control. With the one gene-one enzyme hypothesis, each individual trait was thought to be the result of a single individual gene. Under this hypothesis, genes behaved independently of one another and the effects they produced were isolated to that gene.

The biochemical chain that explained how genes produced their effects was furthered in 1953, when J.D. Watson and Francis Crick identified DNA (deoxyribonucleic acid) as the fundamental genetic material (Keller 1995). The location of the gene within the human body could now be known. Genes existed as segments along the sequence of DNA. Coupled with the one gene-one enzyme hypothesis, it was widely accepted that the gene had been located and the chain of reactions through which genes produce their effects was understood. The combination of these two revelations established the central dogma that: "DNA makes RNA, RNA makes proteins, and proteins make us" (Keller 1995: 18). This model of gene function where DNA is the carrier of genes which is the genetic code that is transcribed into proteins which produce an effect (a phenotype), this model persisted as the dominant explanation throughout the remainder of the 20th century (Landecker and Panofsky 2013). This marked a shift in the discourse of 'gene action' to a molecular level. DNA was compromised of genes, and genes were carriers of molecular codes that produced an effect by making proteins (Keller 2014). The collection of genes in this model is what became to be known as the genome.

This transition marked the first major shift in genetic discourse, moving from the isolated and individualized conception of the gene into a new era of genomics. Genomics although commonly mis-used synonymously or interchangeably with genetics, masks a significant difference. Both examine genes (sections of DNA) studying their molecular composition and functionality. With genetics the approach is to consider single genes whereas genomics approaches the task considering multiple genes and their interrelated connections. The consequence of the transition from genetics to genomics was that the genes were no longer isolated and independent, but their traits or effects influenced other genes. It was no longer one gene responsible for a single trait but rather an interconnected assemblage of traits working as part of a complex network.

3.2 Genomics

'Gene action' reached an apex at the tail end of the 20th century with the Human Genome Project (HGP). Then director of the National Human Genome Research Institute (NHGRI) Francis Collins described the project as "a research program that would characterize in ultimate detail the complete set of genetic instructions of the human being" (Collins 1999: 28). The primary objective of the HGP was to sequence or map the cumulative collection of genes in the human genome.

Of course, I do wish to highlight that this endeavor to map the human genome is one that conceives of the genome as static or fixed. Although this project emerged long after Darwin and his theory of evolution, the HGP viewed the genome of each individual as fixed. To clarify that statement, from this perspective, each individual's genome is constant and unchanging through their life course. Through epigenetic data it is clear that this is not the case, the genome of each and every individual is responsive and adaptive and will alter over a life course. Perhaps hampered and constrained by the concept of generation, the mutations that drive Darwinian evolution were thought to occur across generations.

Ontological priority at the onset of the HGP was still granted to the gene for as Collins described, "genes contain the basic information about how a human body carries out its duties from conception until death" (Collins 1999: 28). This is illustrative of the

deterministic and reductionist thought that characterized the gene paradigm. Prior to the HGP the gene was only conceived as producing or enacting an effect. That is to say the gene was always thought as the causal or determining actor. A one directional flow in which the gene produces an effect.

The HGP promised to answer many questions about human development with one of the foremost being, "what makes us uniquely human" (Lock and Nguyen 2010: 308). Researchers of the HGP were anticipating sequencing somewhere between 80,000 and 100,000 unique (protein coding) genes (Collins 1999; Landecker and Panofsky 2013). However, before the HGP even began, some biologists such as Robert Weinberg voiced concerns over the viability of the project. Regulatory genes are protein coding genes, which regulate the rate of protein synthesis and structure of other genes (Keller 2014). Comprised of regulatory genes, upwards of 95% of the human genome was cast aside in the early portion of the HGP as junk DNA (Keller 2005). What resulted was disappointment as only around 20,000 genes were sequenced and it became clear that there was significantly more DNA in our genome that was considered junk than that which is specifiable to specific genes (Keller 2014; Landecker and Panofsky 2013).

Junk DNA otherwise known as non-coding DNA, are segments of the DNA that do not code for a protein. In not coding for a protein these segments do not carrier the genetic material that comprises a 'gene'. These segments of DNA have been referred to as junk as it was originally believed to be non-functional. Through epigenetic research however, it is becoming clear that these segments serve regulatory functions.

It has been suggested by Fox Keller that this disappointment was what eventually dislodged the discourse of 'gene action' (2005). With less than 5% of the genome actually being comprised of genic information, questions about the purpose of 'junk' DNA shortly arose (Landecker and Panofsky 2013). It was increasingly clear that the genome sequence was not going to be able to deliver on its many lofty promises. Further, it was becoming apparent that reductionist perspectives that isolated that gene were no longer compliable with the genomic era (Keller 2005). The result was a shift such that what was thought to originally be 'junk' played a significant regulatory role that

promised to lead to meaningful discoveries (Keller 2014). It was becoming increasing clear that what had been placed aside as junk DNA, plays a highly influential role in gene expression. Thus, sequencing the human genome, which was expected to elucidate the molecular functionality of the gene, alternatively shook the paradigm of gene action and created a space for the establishment of epigenetics.

The sequencing infrastructure established through the HGP provided the technical foundation for epigenetics, and research attention shifted towards non-genic DNA's role in regulating gene transcription (Landecker and Panofsky 2013). The gene was not displaced as the unit of genetic information, that has very much persisted in epigenetics. Rather, what has changed is the perception of the genome. Once sequenced it became clear that genome was responsive, and its casual chains of response functioned not in altering genic material but in regulating gene expression. The HGP promised to tell us what makes us human and while it did not fulfill this promise as originally expected (through genetic reductionism, i.e., explaining how genes make us who we are), it was able to introduce further complexity [and accuracy to the real world functioning of the genes] to the question. For some time, we have understood "that organisms interact with their environments, that interactions between genetics and environment, between biology and culture, are crucial to making us what we are" (Keller 2014: 2428).

In many instances such as the potential for personalized medicines, epigenetics has assumed the mantle of next best hope in medical and other various research fields. There is a great deal of optimism that epigenetics will be able to deliver some of the unfulfilled promises of the HGP. Already, epigenetic drugs have been put in use in treating various cancers, and it is anticipated that this will increase in the future where we will see epigenetic drugs employed in the treatment of additional health conditions (McGowan and Szyf 2010). However, one of the biggest long-term benefits made possible through epigenetics is a reconceptualization of genetics. It offers to lessen if not completely displace genetic or gene determinism. By accepting increased complexity, epigenetics has the potential to put to rest the nature-nurture dichotomy. The HGP revealed that the genome does not operate devoid of environmental context, and epigenetics allows researchers to engage critically with the biochemical interaction whereby the

environment is in a continuous negotiation with the genome resulting in epigenetic changes that regulate gene expression (Meaney 2010).

Epigenetics further drives the conceptual shift that began with the transition to genomics from genetics. Epigenetics offers to expand its explanatory scope beyond the gene. Epigenetics widens the complex network of gene interaction to now include the environment. Through the discourse of gene action, the gene and its effects were conceived as individual and isolated. The genomic eclipse of gene action altered this as genes became interconnected and influenced one another. Now with epigenetics it has become evident that genes interact with the environment which mediates gene expression. The gene has gone from individualized and isolated to an actor as part of an interconnected network that includes other genes as well as the environment. It is precisely this call to expand beyond the gene about which I am most optimistic

3.3 Epigenetics: Gene x Environment Interaction

A major revelation to emerge from the HGP was, that non-coding portions of DNA played a significantly more important role in the regulation of gene expression than originally believed. With the genome being comprised of significantly fewer genes than estimated, coupled with the knowledge that our genome was composed largely of non-genic or non-coding segments, questions quickly arose as to the purpose of this dark or junk DNA. Interest had shifted from the gene to understanding what all this extra DNA was for. The disappointment of the HGP as discussed above is often credited as giving rise to epigenetics (Landecker and Panofsky 2013). One thing the HGP project was able to make abundantly clear was that the gene no longer fit the definition of an ultimate determining factor. Instead, the gene's strong hold of both temporal and ontological superiority has since been under ever mounting scrutiny.

In the wake of the HGP's letdown it became evident that genetic/genomic research had to move beyond the gene. Epigenetics provided the conceptual space to consider the gene as being acted upon. Consider the etymology of epigenetics for a moment: we see how

the gene was dislodged for its podium. The prefix ‘epi’ comes from Greek and mean upon, on or above. Epigenetics means on, upon or above genetics. In this epigenetics managed to eclipse the ‘discourse of gene action’. Shifting from genetic determinism wherein the locus of control for biological development was solely held by individual genes. Epigenetics established a new paradigm and in doing so replaced the conceptual metaphors through which genetics was understood. Out was the old dogmatic metaphor of chain reaction in which DNA made proteins (Keller 1995), now to be replaced with genes as switches that can be turned on and off (Chung et al. 2016). This change is so fundamental it completely alters the questions researchers must ask. Instead of seeking to understand the ‘genetic code’ and identifying what gene code for certain proteins, researchers should now seek to understand what activates gene’s switches.

In these emerging metaphors the locus of control for biological development and function has been moved from level of the gene and in effect is creating a new level that is above or acts upon the gene. In doing so epigenetics has uprooted the gene and revealed genetic determinism to be entirely fallacious. Significantly, epigenetics perhaps offers a definitive end to the long-standing debate of nature (gene) vs nurture (environment). This debate has endured because it is predicated on a notion that the individual or more specifically their DNA is separate from the environment. Rather, “the individual is best considered as the emergent property of a constant interplay between the genome and its environment” (Meaney 2010: 45). What epigenetics is making clear is that gene expression is situated in complex socio-environmental entanglement.

In the epigenetic context I speak of here, the environment “is the relation people [and their genes] have with their dynamic biophysical surroundings” (Ramutsindela 2018:102). Environmental stimuli such as the experience of poverty enter into a direct dialogue with genes through epigenetic regulation of gene expression. The continuous dialogue between gene and environment resituates the gene as a social actor. Gene expression is intrinsically tied to the spatial and temporal context of the individual. Thus, to consider the functionality of genes in a manner that ignores the influence of specific social experiences will always be incomplete.

it is critical that as epigenetics develops further it does not replace genetic determinism with environmental determinism. By this I mean, epigenetic researchers must avoid reducing environmental experiences to a biochemical level as doing so reestablishes a linear causal chain that epigenetics has been able to disrupt. Rather, the focus should be placed on understanding the biological effects of specific social experiences. Epigenetics as a human science should have the objective of tracing the connection between social experience and biological expression. Policy and action plans should be developed to prioritize social rather than biological interventions by developing targeted specialized biological responses to negate the biologic impact of particular social experiences as opposed to addressing the social condition as a band-aid and not in a true sustainable solution.

3.4 Transgenerational Epigenetics

Humans are eukaryotes which means the cells that makeup the human body have a nucleus. The nucleus is a membrane enclosed portion of the eukaryotic cells wherein DNA is stored and protected. Our DNA exist as in extremely long strands in the famed double helix, that familiar long spiraling ladder. However, DNA is so long it must condense itself in order to fit inside the nucleus. This is achieved by DNA binding histone grounds which behave like a DNA spoil. Similar to how string is wound to a spoil, DNA winds itself to histones forming nucleosomes which resembles beads on a string. Nucleosomes then stack on each other folding and further condensing DNA forming chromatin. During the cell division and replication (meiosis and mitosis) chromatin will become further condensed to form chromosomes. However, outside moments of meiosis and mitosis DNA inside the nucleus is condensed only to the level of chromatin.

Partial segments of the DNA are copied that are known as RNA. These RNA segments are able to pass through the membrane of the nucleus. Outside of the nucleus, RNA is able to use the cellular machinery to code or make proteins. Proteins in turn build cells, cells form tissues, tissues make organs and organs form humans. This is the line of progression that forms the central dogma of genetics, that DNA makes RNA, RNA makes proteins and proteins make us. However, epigenetics has revealed a wrinkle in

genetics liner system. Exposed tails of the histone groups that form nucleosomes are subjected to enzyme-catalyzed modifications (Ueberheide and Mollah 2007). Enzymes can bind to the histone tails causing alterations to chromatin structure (Ueberheide and Mollah 2007; Meaney 2010). These structural modifications of the chromatin can increase or decrease the availability of segments of DNA to be transcribed into RNA. These modifications occur at the first part of the genetic chain and therefore alter the entirety of the chain.

These alterations do not actual modify DNA itself, rather what is modified in how it has been structured into chromatin. Enzyme-catalyzed modifications are brought about as response to environmental exposures and experiences, effectively establishing the biological pathways through which epigenetics is able to trace the how social/environmental exposures become imprinted on the genome and regulate gene expression. It is important to note that influence of genetic and environmental influences cannot be separated but rather must understood as in constant interaction.

The Moshe Szyf and Michael Meaney laboratory out of McGill University have been at forefront of epigenetics research connecting social experience to with genetic modifications. In a wide cited 2004 paper Weaver et al. established connections between maternal care style and the stress phenotypes of adult rats. The study compared the effects of maternal grooming habits of high-licking and low-licking maternal grooming styles. The study found that low-licking resulted in epigenetic changes to the pup's glucocorticoid receptors (GR) in the hippocampus resulting in higher levels of anxiety in pups. The Szyf-Meaney lab has conducted studies on human as well. A separate study, (McGowan et al. 2009) compared the postmortem hippocampal tissue of suicide victims. They discovered that there were higher levels of methylation of GR or decreased GR expression amongst suicide victims with experiences of child abuse. Tissues were compared to both suicide victims and non-suicide tissue samples from individuals who did not have a history of child abuse. These studies have demonstrated that environmental exposures become epigenetically imprinted on the genome.

Further it has also been shown that these epigenetic imprints are stable enough to be passed to future generations. Epigenetic markers that result in chromatin modification are reproduced during cell proliferation (Skinner 2011). This includes meiosis or the development of sex (germ-line) cells. This of course means that epigenetic markers are molecularly stable to the point that they are reproduced into germ cells and passed from parent to off-spring in what is called epigenetic transgenerational inheritance. Through this process the biological implications of a given environmental exposure are able to affect future generations even without direct exposure. When an environmental/social experience is able to enact its affect upon multiple generations this has been termed multigenerational exposure (Skinner 2014). It is important to note that multigenerational exposure does not require direct exposure by all generations. Rather, it refers to the exposure of effects induced and imprinted upon the genome by the original experience. Multigenerational exposure is the exposure of effects opposed to the actual experience. Here exposure and experience become distinct and separate terms.

It then becomes possible to point to a single environmental/event as an experience and trace the number of generations that have been exposed to it even without direct experience. Using this framework, it becomes possible to trace the multigenerational exposure of holocaust in Jewish families. Although, recent generations did not have direct experience they may be exposed to its effects still. This similarly can be applied to Indigenous families and the experience of residential schools. The Truth and Reconciliation Commission (TRC) sought to explore the effects of residential schools. However, the focus of the TRC was predominantly cast on individuals with direct experience. Further, instances in which the TRC consider the intergenerational effects of residential schools often pertained to the social and cultural ramifications described as intergenerational trauma. The extent to which the TRC considered generations without direct experience is largely confided to culture lost. Yet when considered from an epigenetic perspective it seems almost obvious that effects of residential schools are also an instance of multigenerational exposure. Indigenous groups should be able to utilize epigenetics to point to residential schools as having epigenetic impacts that have persisted for generations. Presumably these effects are cumulative and mutually reinforcing.

3.5 Conclusion

As a decolonizing method, two-eyed seeing is a method that embraces the contributions of both Western and Indigenous ways of knowing. Mi'kmaw elders Albert and Murdena Marshall developed two-eyes seeing as a decolonizing methodology for bringing together two distinct worldviews (Martin 2012). Epigenetics is decolonizing in the sense that it allows for the possibility of two-eyed seeing. Two-eyed seeing is not about rejecting or displacing Western science, rather it represents a balancing and re-centering of Indigenous perspectives. In that sense, epigenetics can be a decolonizing science when utilized for Indigenous objectives. The remainder of this project consists of application through a comparison of two case studies that explore the decolonizing potential of epigenetics.

Chapter 4 : The Dutch Famine Case Study

4 Introduction

This chapter consists of a case study of the Dutch Honger Winter or Dutch famine. The famine is one of the most well-researched events with mass epidemiological and epigenetic data events historically. It is an exceptional case due to the extensive and in-depth birth records maintained in Holland.

Utilizing the Dutch famine as an example, I intend to highlight how epigenetics may provide valuable insight into the connection between health and environment. I will explore the health outcomes that have been associated with famine exposure during the critical developmental window of prenatal development. Epigenetics will be used to understand and explain the biochemical alterations established in response to the famine and increased disease susceptibility. Highlighting epigenetics clarifies the effects of environment alongside biology to produce alterations to human bodily form and functions that display the plasticity of human development. The science behind this case study will be applied to an Indigenous context of food conditions in residential schools to present epigenetics as a decolonizing way of thought.

4.1 Historical Background

Following the successful beach invasions on D-Day, Allied forces were able to push back the Nazi defensive lines. The advancing Allied forces had liberated much of France, Luxembourg and Belgium and by mid-September 1944 had progressed to the Netherlands. Given the speed at which Allied forces had advanced to this position, it was believed by many commanders that the Germans would be forced to surrender within a matter of days (Roseboom, de Rooij, and Painter 2006). But the Allied advance was brought to a halt with the failure of Operation Market Garden, whose primary objective was to secure key bridges in the Netherlands to help establish a foothold on the other side of the Rhine; this resulted in a military standstill. The consequence of Market Garden’s failure, apart from their military strategy, was that it left the Netherlands divided with one

part remaining under German occupation while the other parts of the country already had been liberated at the time of the standstill.

The Dutch government in both a display of support of the Allied effort as well as to reassert its authority over the nation advocated for a railway strike. Already heavily weakened, the Germans responded to the strike by placing a ban on all food transports to the western part of the Netherlands. Food supplies in the Netherland's urban west were quickly depleted (Scholte, van den Berg, and Lindeboom 2010; Roseboom, de Rooij, and Painter 2006). Early in November 1944 the German forces lifted the embargo on food transports; however, an unusually early and cold winter had already set in. By that time critical waterways and canals that were frozen, making food transports by water which the Dutch relied upon greatly no longer viable. As a result of the embargo coupled with the early and uncharacteristically cold winter "the western part of the country was closed-off from any imports of food, fuel, medication, etc." (Scholte, van den Berg, and Lindeboom 2010: 19). This caused famine in urban centers in the west of the Netherlands that would persist until the European end of WWII in May 1945.

Particularly interesting with the Dutch Famine is the establishment of a definitive beginning and end of the period of famine. This is the first of three reasons for which I refer to the Dutch Famine as being neat and tidy. Famine frequently follows war and drought (and other major ecological events), however, this combination frequently works to blur the lines of demarcation surrounding famines. Generally speaking, most other famines do not have clearly defined temporal boundaries. The Great Chinese Famine has been cited as occurring between 1959 to 1961 by some sources while other sources date the famine as taking place between 1958-1962. Furthermore, discrepancies amongst sources indicate a death toll ranging from 36-45 million (Gustafsson 2019). While with other famines, such as with the Chinese, temporal limits are not as easily defined, but the Dutch Famine has very clean and near universally accepted period of famine.

Additionally, the Dutch Famine began suddenly and rapidly and concluded as suddenly as it had started. This aided in the development of a clear period of famine as discussed above. This is important because both prior to and after the famine the Dutch population

in affected areas of the Netherlands were poorly nourished. Leading up to the famine Dutch war-time rations for adults had been slowly declining from an average of 1800 calories/day in December 1943 to an average of 1400 calories/day in October 1944 just prior to the onset of famine (Roseboom et al. 2011). While not the 2000 calories recommended, the Dutch population could be considered well-feed and properly nourished for war-time Europe. At its peak, adults in exposed areas had daily rations that had been reduced to between 400-800 calories/day (Roseboom et al. 2011). Once liberated, rations immediately rose to above 2000 calories/day (Scholte, van den Berg, and Lindeboom 2010). Daily rations both before and after the famine provided a sufficient source of nourishment. In this way the Dutch Famine is not only clearly defined temporally, but also has a sharply defined period of actual food shortage.

A second major benefit the Dutch Famine has provided for researchers is a built-in control group. As previously discussed, the Allied forces had liberated a portion of the southern part of the Netherlands; however, failure of operation market garden refocused efforts eastward towards Germany. Consequently, this left a large portion of the western Netherlands under German occupation while the majority of country had been liberated. This meant major urban centers of the west such as Amsterdam, Utrecht and Rotterdam were exposed to effects of famine. Meanwhile, other major urban centers that had been liberated such as Eindhoven and Tilburg were not exposed to the effects of the famine. As with the period, the famine has well-defined spatial boundaries. The spatial and temporal boundaries that define the famine effectively established two different control groups for this naturally produced experiment. Firstly, the more obvious of the two is the spatial control group; non-exposed individuals born outside the spatial boundaries during the time of famine. The second more specifically refers to those who were born in the area prior to the onset or conceived after the famine (Scholte, van den Berg, and Lindeboom 2010). Or more plainly those individuals born within the boundaries of the famine around the time of the effect without being exposed in utero.

A nearly perfectly designed experiment, the naturally built-in control groups of this birth cohort presented a truly unique opportunity for the study of the effects of famine. Substantial scholarship has been dedicated to the study of long-term effects of this

famine. The temporal control has been particularly valuable in producing same-sex siblings for comparison (Heijmans et al. 2008). This of course is only made possible by the third and final exceptional circumstance of the Dutch Famine, that being the remarkably detailed records kept by the Dutch.

Even throughout the period of famine medical professionals including doctors and midwives continued to provide professional services (Roseboom, de Rooij, and Painter 2006). They continued to maintain birth records during the famine. This is significant because the Dutch have a notorious reputation for record keeping. The Dutch birth record tracks mother's name, address, age, occupation, religion, last menstrual period, as well as bodily measurements, while for the child records are kept on sex, length (head-to-crown), birth weight and head circumference (Lumey et al. 2007). This rich data set has proven to be immensely valuable to future researchers. Providing a rich and complete data set on individuals for long-term studies is another instance of how the Dutch Famine is neat and tidy.

A population well-fed and nourished, a sudden and rapid onset of famine that ended immediately upon liberation, sharp, well-defined period of famine, naturally occurring experiment, complete with control group in tow. An exceptional and exemplary instance of record keeping providing a detailed data set.

I have highlighted how the Dutch Famine is a neat and tidy case study from an experimental perspective. However, one particular instance in which the Dutch famine is compact, and tidy is in something I call 'the blame narrative'. The Dutch Honger Winter or Dutch famine occurred during World War II, and as such has inherited non-controversial bad guy(s) to place the blame on. There are multiple ways in which the events and conditions of the Dutch famine can be blamed on the Nazis. This is something that adds to the exceptionalism of the Dutch famine as a research topic. It has evolved from a mass multinational conflict to become a non-politicized research asset.

This is a critical and for the most part unique distinction of the Dutch famine. Epigenetics as a study of the interaction of genes and environment is likely to uncover information that could easily become politicized. The first instinct may to use epigenetics as a means

of pointing the finger and casting blame on institutions and individuals. The objective of epigenetics is not to uncover horrible deeds in order to assign blame. Epigenetics does not need to assign blame, but rather to acknowledge the social experience as the catalysts of epigenetic alterations. Blame entails a need for justice or that some retribution must be paid by the offending party. Acknowledgment does not lessen the causal nature of the experience but also does not seek justice. However, epigenetics has a decolonizing potential as we see in chapter five on residential schools and Indigenous health.

It is important to remain mindful so that Dutch famine is not burdened by the blame narrative. As such researchers should prioritize establishing a foundational knowledge of the epigenetic alterations induced by famine. There is a clear acknowledgement of a perpetrator in the case of the Dutch famine, whereas trends in biogenetics undercut the habitual Canadian narrative to blaming the victim. The TRC's impetus and narrative implicates institutions and forces in a collective manner that exempts individual agency or ethics, thus exempting powerful perpetrators from vilification. Rather than implicate the colonial institutions like residential school, the Canadian context cast blame on the victims manifesting in commonly expressed sentiments like the 'drunken Indian'. Such attributions blame the victim for the symptoms, while avoiding any acknowledgement of the perpetrators.

4.2 Developmental Origins of Health and Disease

Near immediate distribution of food provisions by Allied troops following German surrender providing the famine stricken western Netherlands with much needed relief. Additional Allied support prioritized medical aid. Medical professionals from the United States and United Kingdom were sent to provide medical services. Amongst the first of these doctors to observe the effects famine had on the Dutch population's health was Clement Smith of Harvard Medical School (Roseboom et al. 2011). Straightaway, Smith was able to identify the valuable opportunity that had presented itself, an opportunity to observe and study how adverse maternal nutrition influenced the offspring. Utilizing the comprehensive birth records from famine-stricken Rotterdam and The Hague, Smith was able to analyze the impact of prenatal exposure to famine. In 1947 Smith published, *the*

effect of famine on pregnancy and its product, detailing his observation that those born during the famine hereto exposed in late gestation were born roughly 200 g lighter.

Initial observations made by Smith focused on the immediate impact of famine by analyzing the anthropometric measurements taken immediately upon birth or closely thereafter. Early studies sought to use anthropometrics from the time of birth such as weight, length and head circumference, to make analytical comparisons of effects across differing periods of prenatal exposure (Stein and Susser 1975a). From the onset, researchers of the Dutch Famine not only categorized individuals of the birth cohort into unexposed and exposed, they further subcategorized exposed individuals according to the stage of pregnancy during exposure. The neatness of the Dutch Famine made the categorization of the unexposed simple as famine was geographically contained. Exposed individuals were separated according to the stage of gestation at the time of famine which was commonly inferred using date of birth.

Introductory studies relied predominately on anthropometric measures focused on effects of the immediate product of the pregnancy. Or more plainly, interpreted measurements of new-borns. Preliminary finds were limited in their effectiveness by only being able to make unequivocal inferences with respect to those individuals exposed during the third trimester of pregnancy. Persons conceived prior to the famine but born during the famine would have had such late gestational exposure and were found to be lighter, slightly shorter and have smaller head circumferences (Stein and Susser 1975a). Measures such as weight of offspring were markedly impacted by famine, while body length displayed less famine induced variation, possibly suggesting particular tissues like fat (weight) and muscle are more susceptible to environmental influences like nutrition than tissue like bones (body length) (Stein and Susser 1975b). Analyzing such trends researchers were able to infer the role of nutrition during intrauterine exposure.

The focus of these early research projects was centered around the impact of famine on prenatal development. Measures like birth weight and length provide insight into the immediate impacts and consequences of famine. The long-term impact on the exposed birth cohort was first seriously investigated by Mervyn Susser and Zena Stien, in their

study of Dutch military conscripts in the early seventies (Roseboom et al. 2011). Their study was propelled by an increased awareness that adverse nutritional conditions early in life may result in permanent damage to the brain. It can be safely concluded that this was only possible after some time had elapsed. Eventually, they concluded there was no detectable effect of famine exposure and mental performance amongst the conscripts (Stein et al. 1972). But they established a recalibrated approach to studying the effects of famine, moving past the immediate effects, instead questioning how the prenatal environment effects individuals into and throughout postnatal life.

While investigating the effects famine exposure has on mental performance, they were able to make supplementary observations that would guide their subsequent work. Follow-up studies highlighted their observation that, men exposed to famine early in gestation were more commonly obese, while men exposed in late gestation were less commonly obese (Ravelli, Stein, and Susser 1976). Divergence from prior studies including Smith's on birth weight only detected effects contributable to famine in individuals exposed to famine late-gestationally (during the third trimester) (Stein and Susser 1975a). Implicit in these findings is the suggestion that effects of prenatal famine exposure may lay dormant, not producing an immediate effect at the time of birth.

Awareness increased of the significance of intrauterine environment and the implications and impact it has on later adult health. In the 1980's this concept was formulized into theory by British epidemiologist David Barker's 'fetal origins hypothesis' (also referred to as Barker's hypothesis) (Roseboom et al. 2011). Mapping and cross comparing infant mortality between 1921-25 with ischaemic heart disease mortality between 1968-78 throughout England and Wales, Barker and Osmond found a strong geographical relation between the two (Barker and Osmond 1986). Although ischaemic heart disease rates had been increasing in Britain at the time of study, the duo noticed that paradoxically the least prosperous regions experienced the highest frequencies (Schulz 2010). After establishing an association between heart disease and infant mortality, further examination into the causes of infant mortality pointed to malnutrition during prenatal and early postnatal life as the cause (Barker and Osmond 1986). The fetal origins hypothesis suggested that

adverse prenatal and early postnatal nutrition increases susceptibility to heart disease in adult life.

Barker further developed his hypothesis examining the effects of prenatal undernutrition at different developmental stages of the pregnancy (Wadhwa et al. 2009). Distinct phenotypic characteristics, each with associated hormonal alterations producing metabolic irregularities in later adult life were linked to the gestational stage during undernutrition (Barker et al. 1993). Barker's fetal origins hypothesis was thus proposing that prenatal undernutrition reprograms and permanently alters the body and metabolic profile such that it increases an affected individual's risk of heart disease in later adult life. The Dutch Famine was invaluable as a model to test Barker's fetal origins hypothesis with human subjects in the early nineties (Schulz 2010; Roseboom et al. 2011). Studies of the famine's birth cohort have provided evidence of impaired glucose tolerance, obesity, increase blood pressure and raised cholesterol levels, which contribute to heart disease, originate in-utero (Roseboom et al. 2001), supporting the hypothesis that disease is the result of fetal adaptations made in response to an environment of undernutrition.

Barker's hypothesis attracted global attention and built upon the concept that the fetus responds to environmental cues developing predictable adaptions which could increase the risk of diseases in adult life. The Fetal Origins of Adult Disease (FOAD) was the focal topic for two world congresses that were assembled, the first in Mumbai (India) in 2001, the second in Brighton (United Kingdom) in 2003. Following the second FOAD congress meeting, the International Society for the Developmental Origins of Health and Disease (DOHaD) was established. The transition from FOAD to DOHaD intended to "recognize the broader scope of developmental cues, extending from the oocyte to the infant and beyond, and the concept that the early life environment has widespread consequences for later health" (Gillman et al. 2007:625). Expanding the critical window well beyond the period of fetal development, the DOHaD begins consideration from the oocyte (female germ cell or egg) to infant life.

The Society for DOHaD defines the paradigm as " a multidisciplinary field that examines how environmental factors acting during the phase of developmental plasticity interact with genotypic variation to change the capacity of the organism to cope with its environment in later life" (Heindel et al. 2015:19). This definition aligns with epigenetics conceptually as applied in my project. The paradigm proposes that environmental stimuli during critical periods of developmental plasticity modify the development trajectory in response, which can lead to increased disease susceptibility. Additionally, it is currently believed that other windows of developmental sensitivity occur throughout the lifespan. These likely include phases of significant hormonal changes and growth such as puberty and peri-menopause amongst others (Heindel and Vandenberg 2015). At present it is not clear how influential each of these critical transitionary windows are as the bulk of the research has been contained to in utero fetal development. And we must assume that they do not operate discretely?

While a proficient understanding is yet to have been fully developed, DOHaD framework acknowledges that biochemical pathways mediated by environmental stimuli reengage with the biological composition throughout the life span. The DOHaD approach has been described as "a new developmental synthesis is evolving where the weights of genetics, development, and the ancestral, intergenerational, and current environments in disease causation are more balanced than previously thought" (Hochberg et al. 2011:162). Dutch famine survivors have been used to research connections between genetics and development to the environmental stimuli of prenatal malnutrition. In the following section Dutch famine survivors have provided a unique retrospective preceptive to DOHaD. More specifically, they highlight the epigenetic modification of the growth gene Insulin line Growth Factor 2 and its larger impact on later adult life including increased risk of diabetes and coronary heart disease. The objective of this example is to present an epigenetic profile that can be catalogued and compared.

4.3 Insulin Growth Factor 2

Human growth and development is mediated by a multi-variable complex of programmed interactions of key non-linear growth pathways. To a certain degree epigenetics has been shown to play a key role in the regulation of these developmental and growth pathways.

One of the most well-known epigenetically regulated growth pathways is the gene Insulin like Growth Factor 2 (IGF2). IGF2 is an important growth gene that is epigenetically regulated by the nearby H19 gene that is located 90kb downstream (Hochberg et al. 2011). In this interaction the H19 gene imprints an insulator which prevents interaction between IGF2 and downstream enhancers effectively inhibiting IGF2 expression. Essentially what is this means is that H19 methylation acts like a barrier that prevents the expression of the growth gene IGF2. It is through understanding the mechanisms of epigenetic regulation such as IGF2 that we are able to gain valuable insight into developmental plasticity by understanding the molecular response of genes to the environment.

In understanding how epigenetics regulated the expression of genes essential to growth and development it becomes possible to uncover instances of epigenetic dysregulation of gene expression. Epigenetic dysregulation of gene expression potentially could uncover causal links between changes in gene expression and various pathologies. IGF2 is one such epigenetically regulated growth gene which provides an excellent example to illustrate my point.

As mentioned IGF2 is one of the most well understood epigenetically regulated growth pathways. In examining the IGF2 patterns in individuals exposed to the Dutch Famine an interesting pattern makes itself visible. Individuals exposed to the famine during early gestation displayed lower levels of IGF2 methylation compared with a control of unexposed same sex siblings (Heijmans et al. 2008; Heijmans et al. 2009). This is interesting as it would suggest that exposure to traumatic experience of famine early in gestational development alters the epigenome's regulatory programming to methylate the IGF2 gene. The environmental experience of famine during the critical developmental window of early gestation induces the molecular response of genes as epigenetics does not restrict the expression of the IGF2 gene resulting in dysregulation of the growth pathway.

Now with an understanding of the epigenetic dysregulation of individuals with famine exposure early in gestation we can re-evaluate some already mentioned health outcomes.

Early gestational exposure has been linked to adverse mental and health related outcomes (Lumey, Stein, and Susser 2011). The previously discussed Dutch conscript studies found an increased prevalence of obesity amongst individuals exposed to the famine in early gestation. Furthermore, studies have connected early gestational exposure to the Dutch famine with a more atherogenic lipid profile (promotes fatty plaques in the arteries) and an increased risk of coronary heart disease (Roseboom et al. 2011; Lumey, Stein, and Susser 2011). It does not require too much extrapolation to see the underlying connection here.

IGF2 is a human developmental and growth pathway that is epigenetically regulated. Exposure to an environmental stimulus (famine) during a critical period of development (early gestation) resulted in the molecular response of the genes that caused epigenetic regulatory methylation of the IGF2 gene to be prevented. Consequently, the IGF2 growth pathway remains unmethylated or uninhabited allowing for its continuous expression. The loss of methylation in turns means exposed individuals continue to promote growth which leads to increased levels of obesity. Increased obesity levels as well as an atherogenic lipid profile are known contributors to coronary heart disease. Therefore, it seems reasonable to infer that the exposure to a stimulus during a critical period of development produced a genetic response that limits the epigenetic regulation of the IGF2 growth pathway, if unregulated the IGF2 growth pathway increases risk of obesity, atherogenic lipid profiles and risk of coronary heart disease.

Dutch Famine survivors that were exposed to the famine during early gestation have been the focal point into the leading research on the epigenetic regulation. A complete genomic analysis of such individuals reported associations between five phenotypes and identified six key differentially methylated genomic regions (Tobi et al. 2014). Additionally similar health outcomes linked to epigenetic regulation of metabolic tissue have been for reported for prenatal exposure to gestational diabetes (Bouchard et al. 2010) and maternal antidepressant use during pregnancy (Soubry et al. 2011). These various molecular alterations of genes caused various environmental experiences can be catalogued and profiled as epigenetic signatures for cross comparison. With this approach it is crucial that we appreciate the environmental stimuli that produced an epigenetic

response in this equation is itself a variable. This equation requires an in-depth knowledge of the epigenetic mechanisms as well as the environmental experience.

Survivors of the Dutch famine have provided the foundation of the for the epigenetic profile of prenatal malnutrition. An epigenetic profile can be reduced to a clean linear line of experience at a critical window altering genes towards an adverse health outcome. Here it is, an adverse nutritional environment during early gestation reduces IGF2 methylation leading to increased risk of obesity and coronary heart disease. The variables in this equation are environment and time to promote epigenetics alterations to genes that consequently can increase susceptibility to various adverse health outcomes. Epigenetic profiles can become a method to categorize and understand the interaction between genes and the environment.

A potential roadmap to future research into the interaction between health, genes and environment may be found in fingerprints. Fingerprints offer retrospective insight into the critical developmental window of early gestation. Fingerprint ridges which not only have significant genetic factor, have been shown to reflect the nongenetic environment. They are permanently formed prior to the mid-point of gestation (Kahn et al. 2008). Exposure to the Dutch famine early in gestation has been linked to the non-expression of an environmentally induced fingerprint marker, which has been further associated to diabetes diagnoses past 50+ (Kahn et al. 2009). Identifying connections between phenotypic expression of fingerprint markers with health outcomes provides an inexpensive method to establish potential research populations in order to expand past the exceptional case of the Dutch famine. In doing so it is possible to use the Dutch famine as the cornerstone in which an extensive catalogue of epigenetic profiles is mapped and analyzed, expanding and deepening our understanding of the mechanism behind the gene-environment interaction regulated through epigenetics.

This has the potential to expand the DOHaD paradigm to systems that can describe the mechanics of developmental plasticity. A comprehensive understanding of the influence of our environment is required in order to understanding the developmental plasticity of humans. In understanding the developmental and functional origin of disease it becomes

possible to develop case specific medical and social programs to address the environmental cause and the medical consequence.

4.4 Conclusion

Epigenetics provides in-depth insight into the genetic modifications that occur during critical windows of development and throughout the life course of an individual. In the exceptionally well-documented case of the Dutch Hunger Winter or Dutch Famine, individuals display phenotypic and health related outcomes directly associated to famine exposure in utero. Individuals exposed to the famine during early gestation lack a distinct fingerprint marker, while the marker is expressed on non-exposed individuals. The lack of this fingerprint marker has been further connected to late adult diabetes diagnoses. Negative health outcomes like diabetes and coronary heart disease present at an increase rate amongst individuals with early gestational exposure to famine. These health outcomes have been connected to an epigenetic profile in which the growth pathway of the IGF2 gene is less methylated as a response to early gestational famine exposure.

Epigenetics applied to the case of the Dutch Famine has provided an immense amount of insight into developmental plasticity. This has been the cause for great optimism amongst many health professionals. I believe the evidence connecting fingerprint patterns suggests fingerprints may provide valuable retrospective insights into the early gestational environment. Collection and analyzing fingerprint patterns amongst a population with a shared environmental exposure or of a population diagnosed with a particular disease, provides a manageable and inexpensive means to identify populations for more advanced epigenetic profiling. Developing an extensive catalogue of epigenetic profiles should be the objective proceeding forward. That is, growing the catalogue of environmental stimuli and the epigenetic changes induced that result in risk of a particular health outcome

Chapter 5 : Thrift Gene Case Study

5 Introduction

Canada's Indigenous population experiences a significantly greater burden of poor health than the general population. Negative health outcomes such as chronic disease like diabetes (more particularly Type 2 Diabetes mellitus), disproportionately impact Indigenous peoples in Canada. Most recent data available exposes the age-standardized prevalence rates of diabetes for on-reserve First Nations to be 17.2%, off-reserve First Nations at 10.3%, and 5.0% for the non-Aboriginal population (Public Health Agency of Canada 2011). Intriguingly though, prior to the 1950s, diabetes was extremely rare amongst Canada's Indigenous population, being virtually non-existent (Young et al. 2002). However, less than half a century later in 1997, Stewart Harris et al. reported that Sandy Lake, an Oji-Cree of North Western Ontario, saw the prevalence rates reach heights of 26.1% among all adults (Harris et al. 1997). It is painfully evident that during that time, instances of diabetes amongst Indigenous peoples and communities increased exponentially.

Thrift gene theory is one of the more frequently used in the Canadian health and scientific discourse to explain these figures. Thrift gene theory conveys a simplistic explanation, implicating the evolution of a 'thrifty' gene amongst hunter-gather populations. The theory suggests that an evolutionary trait developed amongst hunter-gather populations that helped endure sustained periods of food scarcity. Further, the theory suggests that this genotype has passed its evolutionary purpose in modern society which is not subjected to the cyclical ebbs and flows of food availability of a hunter-gather lifestyle. Thrift gene theory exhibits the characteristic reductionism and determinism that has been so firmly entrenched in genetic styles of thinking. Moreover, it exemplifies the need for genetic styles of thought to be decolonized.

Thrift gene theory rooted in genetic styles of thought provides an explanation from a 'western' perspective. Throughout this thesis, I have argued that epigenetics is a decolonizing way of thought. This chapter will continue that argument with a case study

of thrift gene theory. While this chapter is a continuation, it also represents a direct application of the decolonizing potentiality of epigenetics.

This chapter is a case study of the cultural construction of thrift gene theory. The first-hand survivor accounts and archival material used here establish a history of malnutrition and systemic underfeeding at residential schools. These survivor accounts coupled with historical accounts and government reports are the sources for my analytical claims in this chapter.

My first case study was on the Dutch famine and the ensuing plethora of epigenetic research that followed it. That case study indicates epigenetic links between famine exposure and a number of negative health outcomes including diabetes. My case study of the Dutch famine provides the scientific backbone to arguments I make in my conclusion as I contrast my two case studies

5.1 Thrift Gene Theory a Colonial Explanation

Proposed by American geneticist James Neel in 1962, the thrifty gene theory offered a simplistic explanation for the disproportionate instances of non-insulin dependent diabetes mellitus (type 2 diabetes) among Indigenous peoples.

In his explanation of the genetic basis for diabetes, Neel states: “For the population geneticist, diabetes mellitus has long presented an enigma. Here is a relatively frequent disease, often interfering with reproduction by virtue of an onset during the reproductive or even pre-reproductive years, with a well-defined genetic basis, perhaps as simple in many families as a single recessive or incompletely recessive gene. If the considerable frequency of the disease is of relatively long duration in the history of our species, how can this be accounted for in the face of the obvious and strong genetic selection against the condition? If, on the other hand, this frequency is a relatively recent phenomenon, what changes in the environment are responsible for the increase? Current developments in the study of this disease suggest an explanation with important biological ramifications” (Neel 1999:694).

As mentioned earlier, there is documented evidence to suggest that the high prevalence of diabetes amongst Canadian-Indigenous people is a relatively recent phenomenon. Therefore, by Neel's own logic, it is evident that changes in the environment are responsible for this increase.

Paradoxically, however, Neels claims that the cause of the increase itself is evolution, reverting to the gene for an explanation. The theory contradicts itself in this manner. He argues, instead, that the thrifty gene phenotype evolved amongst hunter-gatherer populations as an advantageous method to deal with food scarcity.

The theory suggests the evolution of a single trait which Neels describes by employing, in his own words, "a somewhat colloquial but expressive term, a "thrifty" genotype, in the sense of being exceptionally efficient in the intake and/or utilization of food." (Neel 1999:695) Neel is thus explaining that the thrifty genotype enables those who have it to accumulate fat at a much more rapid pace than others. This genotype, in this sense, is being presented as an advantageous trait for hunter-gatherer communities, having developed in response to a need. The ability to rapidly accumulate fat would better equip individuals to sustain themselves in prolonged periods of scarcity.

Neel's theory is based on the notion that the thrifty genotype represents a mismatch in modern societies. Neel (1962) suggested that thrift gene evolved to mediate food scarcity, however, life in a modern society (which assumes an abundance of food) prepares the individual for a famine that never comes. This assumption of an abundance of food can be considered something of an overgeneralization, compounded by the fact of Indigenous poverty which often perpetuates food insecurity. In making this assumption, the theory suggest that Indigenous peoples' high prevalence of diabetes can be justified as the result of the genetic predisposition of the thrift gene.

In the Canadian context, the processes of contact, settlement, and colonization resulted in an irreversible and indelible impact on Indigenous peoples' lives. Neel suggests that part of these changes include an introduction of a Westernized diet and styles of food procurement. This in turn, according to Neel, eliminates the food scarcity experienced by hunter-gatherers. In this sense, Neel's theory suggests a mismatch between the

evolutionary trait and the lifestyle, which now consists of food abundance, leading to increased rates of diabetes and obesity amongst Indigenous peoples globally. Therefore, he offers a reductionist explanation by implicating the theorized thrifty genotype in an overdetermined universal manner that excludes environmental explanations completely. This implies that a trait which was previously advantageous, is now displaced in the context of colonization and becomes a harmful trait.

Neel proposes that people's bodies are in preparation of a famine that is never coming. He goes as far as to indicate that the association is flipped, responding to the research that links obesity to diabetes, calling the thrifty gene a diabetic gene, implying thereby that diabetes causes obesity. Neel ascribes causality inversely, by placing the responsibility in the locus of the gene.

The thrifty gene evolved as a biological mechanism to mediate/ survive the conditions of feast and famine. An individual who possessed this thrift genotype would accumulate fat quickly when food was abundant, thus enabling them to endure sustained periods of food scarcity. The gene becomes problematic because its evolutionary purpose is in conflict with the realities of modern lifestyle.

This position can be juxtaposed with that of anthropologist Marshall Sahlins, who has argued that hunter-gatherer peoples were *The Original Affluent Society.* As opposed to thrift gene theory, Sahlins suggests that the hunter-gatherer life was a good one, in which, "all the people's material wants usually can be easily satisfied" (Sahlins and Graeber 2017:35). Sahlins deconstructs the misconception of the dominant narrative surrounding the hunter-gatherer lifestyle – of the hunter-gatherer's constant search for food to prevent starvation. Rather Sahlins presents the hunter-gatherer lifestyle in a positive light as one that leaves time for leisure and creativity.

The next section explores food and the narrative around hunger, while examining how it is not reducible to one gene but needs to be understood wholistically and requires an epigenetic and decolonized approach. Thrift gene is a colonial explanation and looking at it epigenetically presents a decolonizing approach. Exploring residential schools and

hunger in this section, the attempt is to deconstruct the thrift gene narrative, by approaching the issue from an Indigenous perspective.

5.2 Food Conditions at Residential Schools

Research on residential schools has largely been focused on trauma and its psychological effects. In 2001, Charles Brasfield proposed a residential school syndrome that was modeled after post-traumatic stress disorder. The residential school syndrome diagnosis differs from PTSD in some respects. The significant cultural impact is one, along with an increased propensity for alcohol and drug abuse in survivors of residential schools (Brasfield 2001). The syndrome is not central to my analysis, as it is another example of a colonial impulse to explain Indigenous experience. However, it acknowledges the trauma of the residential school system.

The issue of Indigenous diabetes therefore requires a decolonized approach. As a way of thought epigenetics is decolonizing as it approaches the issue from a wholistic perspective that is more in line with Indigenous conceptions of health and wellbeing. An example of such a conception is captured in Naomi Adelson's *Being Alive Well*, an ethnography on Cree conceptions of health and well-being. Adelson describes the Cree conception of health thus: "… it is less determined by bodily functions than by the practices of daily living and by the balance of human relationships intrinsic to Cree lifestyles. 'Being alive well' means that one is able to hunt, to pursue traditional activities, to eat the right foods and to keep warm" (Adelson 2000:15). Health is not limited to the physicality of the body and is defined as a wholistic well-being that comprises of all the various aspects of life, thus not dependent on any singular explanation like a gene.

Much of the recent research on residential schools has focused on intergenerational effects. The context is intergenerational trauma. Intergenerational trauma stems from a historical trauma and its impact on successive generations. A past historical trauma continues to pose challenges for the wellbeing of the group today. Further, persistence of the impact of a historical trauma across generations has been demonstrated by Bombay,

Matheson, & Anisman (2014), which I believe is indicative of an epigenetic signature being passed on through successive generations.

The study of food conditions in residential schools in the following sections will illustrate the negative food conditions of caloric deprivation and malnutrition experienced by the children. Drawing upon firsthand survivor testimonies as well as archival sources, I intend to establish that hunger and malnutrition were a constant and systemic issue of residential schools. The Dutch Famine has shown that childhood malnutrition can have profound consequences on development. Studies on children exposed to the Dutch Famine from 1944-1945 report increased prevalence of obesity, diabetes, coronary heart disease (Roseboom et al. 2011; Heijmans et al. 2008; Lumey, Stein, and Susser 2011), in addition to poor mental health and increased risk of schizophrenia (Susser et al. 1996; Stein and Susser 1975a).

This section explores food conditions in an effort to establish a connection between the experience of residential schools and famine. In order to deconstruct inadequate colonial explanations of thrift, this analysis proposes a decolonized epigenetic explanation in which diabetes and its prevalence can be explained through a multiplicity of factors, in particular the negative food conditions including malnutrition and food deprivation. Here intergenerational trauma stemming from residential school food experiences, contributes to the increased prevalence of diabetes. I argue that experience of residential schools had mirrored similarities to the experience of famine at a vulnerable developmental age. Further, these comparable experiences resulted in similar epigenetic alterations across schools and generations of students. More particularly, I argue that this line of decolonized thinking discredits colonial explanations of Indigenous health and well-being such as thrift gene theory.

The experience of hunger at residential schools was one of the most consistent testimonies by survivors who spoke to the Truth and Reconciliation Commission of Canada (TRC). Survivor Andrew Paul, who attended residential school in Aklavik, Northwest Territories testified to his experiences before the TRC saying, “We cried to have something good to eat before we sleep. A lot of the times the food we had was

rancid, full of maggots, stink" (The Survivors Speak 2015:71). Andrew Paul's statement illustrates themes of scarcity and a substandard of quality. Scarcity meant children would go to bed with an unrelenting hunger and a substandard of quality of the food that was available, being either rotten or spoiled.

Other survivors attest to inadequate food being supplied in addition to direct accounts of being hunger survivors. They attest to the inadequate food being supplied by speaking of efforts to secretly obtain food. For some like survivors Ken A. Littledeer and Don Willie, they stole food; both made statements describing midnight raids of the kitchen. Others searched the school grounds for food, giving accounts of eating frozen apples found on school grounds, sneaking off grounds to find food in dumpsters, or trying to get food from nearby families (The Survivors Speak 2015; Howard 2014).

In 2014, Heather Howard explored food conditions in residential schools in an ethnographic account of an enduring legacy of negative food relations amongst survivors. Howard presents survivor testimonies of a regimented dining atmosphere associated with punishment, that leaves an enduring legacy of negative food relations. Survivors describe a disciplinary relationship with food that connects food to power as withholding it becomes a form of punishment (Howard 2014). Survivors describe meals being withheld as a form of punishment. Additionally, mealtime itself was used to humiliate children; withholding food was part of a general attempt meant to humiliate. Children who wet the bed would have their tea cup turned over at meals (Howard 2014:536). This functioned to not only deprive the children as punishment but also works to humiliate the child by marking them a bed wetter.

On the other hand, survivor testimonies also have described the use of food and treats by school staff with immoral intentions. Kamsack school in Saskatchewan survivor Elaine Durocher states: "there was a little canteen in the church, and the priest would sell us candies. Well, after they got to know us, they started making us touch their penis for candy. So not only were we going to church to pray, and go to catechism, but we were also going to church 'cause they were giving us candy for touching them. We didn't have money." Another account by John B. Cluster, describes staff giving food for sex:

"…would give us little gifts, like bananas and oranges, and I had no choice but take them, because we were always hungry" (The Survivors Speak 2015:158). The stress associated with food was amplified by the conditions that used food as punishment and 'reward', creating psychologically traumatic associations with food.

Archival records support survivor testimonies

In addition to the survivor testimonies in the previous section, research has unearthed archival documents that provide evidence to support survivors' testimonies. John Milloy's work suggests that there was a long and sustained hunger experienced in residential schools prior to the experiments that were conducted, thus making the malnourished bodies pliable to the research experiments that were to follow. The malnourished and underfed children presented an opportunity when research interests coincided with their particular conditions, though presumably this was an incidental convenience rather than source of the food deprivation.

Survivor testimonies of conditions of hunger and malnutrition corroborated with numerous official records. John S. Milloy's *A National Crime* is an extensive history of the residential school system, which originated as part of the 1996 report of the Royal Commission on Aboriginal Peoples (RCAP). While Milloy explores the residential school system as a whole, his chapter on food and clothing from 1879-1946 confirms that hunger and malnutrition were continual and systemic. Food historian Ian Mosby brought renewed attention to decades-old nutritional studies from 1942-1952. Residential schools became the research subject as decades of neglect made schools ready-made populations of human subjects for researching malnutrition. These projects not only provide evidence of consistent malnutrition throughout its duration but provide further support of the systemic and persistent hunger as well as malnutrition.

Due to Milloy's research originating as part of the RCAP, he was granted unprecedented access to the departmental files of Indian Affairs. Using this unique access Milloy calls on official reports from local Indian agents, school staff and inspectors as clearly valid evidence. Milloy establishes a genealogy that traces the birth, development and many failures of the residential school system. Milloy presents candid reports from school

officials themselves and reports with attested validity from local officials. While Milloy explores the residential school system as a whole, I am particularly interested in his section on food and clothing from 1879-1946.

One such instance highlighted is the report of Squamish Indian agent, F. Ball, “it is difficult to keep a close check on the food supply as officials are courteously but none the less effectively prevented from close investigation and one is naturally desirous of avoiding any unpleasantness with the reverend principal who has been in charge so long. The only meal that I have actually seen was one at mid-day which consisted of a piece of bread and a raw carrot. It may have been a fast day, and I have not since been successful in actually seeing a meal on the table” (Milloy 1999:115). In this account, agent Ball not only clearly indicates that his ability to inspect the food supply was obstructed, he further explains that he did not press the issue as to maintain his relationship with the principal. Further, agent Ball indicates suspicion that the children were under-fed as he attempts to reason away the inadequate meal he did witness as being on a fast day.

Dr. A. B. Simes, Medical Superintended of the Qu’Appelle Indian Hospital in Saskatchewan, compiled a report for the department of Indian Affairs in 1944 on nearby Elkhorn school in Manitoba. The report was ordered in response to complaints made by The Pas and Halfbreed association about the conditions at the school. Comparing the food served to a copy of the menu he had obtained, Simes notes that in reality there were more omissions than substitutions. Simes reported that at the Elkhorn school 28% of the girls and 69% of the boys were under weight. He perceived this as evidence that menus were exaggerated and not a true representation of school meals. Simes concludes his reports stating, “nothing favourable can be said for the recent administration at this institution”(Milloy 1999:114).

Similar accounts by Indian agents Ivor Foughner at Kitimat and John Smith at Kamloops suggest similar efforts to elevate the appearance of food conditions. Both suggest that children were directly ordered by school staff to mislead inspectors. This suggests that reports do reflect an accurate depiction of the full extent of hunger and malnutrition. In other instances, such as Reverend P. Bosquet, who was principal of the Fort Frances

School in Ontario, efforts were made by staff to obstruct and prevent an accurate assessment. Reverend Bosquet opposed an inspection by the Chief and Council of Kootchicing Reserve. Advocating against the assessment, the Reverend argued that the Chief and Council were 'Soviets' who would rile up the children to pretend and make false complaints to their parents.

Amongst the archival accounts that Milloy draws upon, one consists of an employee of the auditor general who reported that children appeared neither well-fed nor well-clothed while delivering a contract to the Carcross school in 1911. A protest event in Kitimat in 1922 required a public meeting between parents, the principal, Indian agents and two RCMP constables. Amid the complaints of poor treatment was how children were not properly fed (1999:114,123). Government officials who were outside the system, by being neither members of the school staff or employees of the Department of Indian Affairs, had no personal stakes in their documentation and thus have added credibility.

Milloy provides ample evidence to support survivors' claims that hunger was a continual and enduring part of the residential school experience. Milloy ultimately concludes, "…the operation of the system did not guarantee that all children would be fed properly, and moreover, that hunger was a continual and systemic problem: that the food given to boys and girls was too often too meagre" (Milloy 1999:115). Milloy extensively documented that hunger was a persistent and ongoing problem which resulted in the continued malnourishment of children.

Although, Indigenous parents had made efforts to establish awareness about the pervasive hunger in schools for decades, it wasn't until after 'official' investigations such as the Simes Report that Indian Affairs acknowledged the widespread instances of malnutrition. However, the department of Indian Affairs acknowledged the malnutrition by allowing the children to be subjected to a number of nutritional experiments that were performed in residential schools across the country.

In 2013, historian Ian Mosby brought renewed attention to a series of nutritional experiments that were conducted on Indigenous Residential School children across the country between 1942 and 1952. In his article *Administering Colonial Science*, Mosby

describes a number of nutritional experiments conducted on Indigenous communities. More particularly, Mosby explores multiple long-term nutrition experiments conducted on residential school children. In experiments conducted on the behalf of the Department of Indian Affairs, Mosby highlights the roles of Dr Frederick Tisdall, former president of the Canadian Paediatric Society and co-inventor of Pablum, Director of the Nutrition Services Division Dr. Lionel Pett, and Dr. Percy Moore, Indian Affairs Superintendent of Medical Services.

In 1947, Dr. Pett estimated that the per capita food grant provided for food was half of what was required. For Dr. Pett, that meant that without any sudden or unforeseen changes to the per capita funding, the already malnourished population would remain as such (Mosby 2013). For Dr. Pett, this represented an unrivalled opportunity as residential schools supplied hungry and malnourished children for experimentation.

In the 1940s, when nutrition was a relatively recent area of scientific inquiry, and much of the expertise of the time was based on animal studies; many of Canada's top nutritional experts saw that the widespread malnutrition in residential schools provided an unprecedent research opportunity. This provided the opportunity to perform control-based malnutrition experiments on human subjects. Malnourished Indigenous children at residential schools came to be viewed as ready to use research bodies. Government officials including nutritional scientists and medical doctors performed a number of nutrition experiments on malnourished residential school children. A variety of experiments investigated the effectiveness of fortified and vitamin enriched foods on malnutrition. Researchers investigated the effectiveness of dietary interventions through enriched foods, using existing levels of malnutrition for baseline comparison. Throughout these experiments, test subjects (malnourished children) were deprived of sufficient nutrition.

In one experiment at St. Mary's Residential School in Kenora, Ontario, children were given the Newfoundland Flour Mix as treatment. The flour mix, which could only be sold in Newfoundland, contained added thiamine, riboflavin, niacin and bone meal (Mosby 2013). This experiment did not provide a nutritional improvement, as rather than

mitigating the problem, this particular research group saw increased levels of anemia with the flour treatment. Another experiment consisted of increased milk consumption at the Alberni Residential School in Port Alberni, British Columbia. The initial assessment found, "…that the diets of the children were lacking in vitamins A, B, and C and iodine because they were not being provided with enough foods like milk, fruit, vegetables, eggs, cheese, and iodized salt" (Mosby 2013:161). To mediate this, Dr. Pett tripled the amount of milk provided to children from 8 to 24 ounces of milk per day.

These examples provide further evidence that children at residential school were under-fed and malnourished. In these studies, quantifiable evidence indicates that children at residential schools were malnourished and that their diets lacked key essential vitamins and nutrients.

While these experiments were going on, Canada was a part of the development of the Nuremberg code of research ethics. In these experiments, subjects were neither informed nor did they provide consent (Mosby 2013). Mosby indicates the irony of this situation, in pointing out how experiments were being conducted on children at residential schools without any informed consent, while a code of research ethics that encompassed Canada's inputs was being put in place.

Survivor testimonies, which have been substantiated by archival records, provide credible evidence that residential school survivors were subjected to a sustained period of malnutrition. When compared to the intergenerational and epigenetic effects of the Dutch famine, the evidence suggests that high prevalence of diabetes among Indigenous peoples in Canada is associated with nutritional deprivations experienced by children in residential schools.

5.3 Cultural Medicine

In *Decolonizing Methodologies,* Linda Tuhiwai Smith defines Indigenous peoples as a population that shares the experience of being subjected to colonization. Smith states, "they share experiences as peoples who have been subjected to the colonization of their lands and cultures, and the denial of their sovereignty, by a colonizing society that has

come to dominate and determine the shape and quality of their lives, even after it has formally pulled out" (1999:7). Thrift gene logic represents an ongoing colonization of Indigenous peoples.

Historian Patrick Wolfe has described colonization as possessing a force that is both destructive and creative. Destructive in that it strives to dominate peoples and resources through the dissolution of Indigenous societies. Creative, in the sense that it seeks to erect a new settler society in its place. In the words of Wolfe, "…settler colonizers come to stay: invasion is a structure not an event" (2006:388). Thrift gene as an explanation for increased prevalence of diabetes amongst Canadian Indigenous peoples is representative of ongoing colonization. By which I mean thrift gene is being rooted in a settler perspective of genetics and is a Western or settler explanation. Using epigenetics to introduce Indigenous concepts of health and wellbeing, is a decolonizing methodology because it is the application of western knowledge for Indigenous purposes. Epigenetics not only displaces genetics' determinism and reductionism rhetoric, it does so in a way that is more in line with Indigenous conceptions of health and wellbeing.

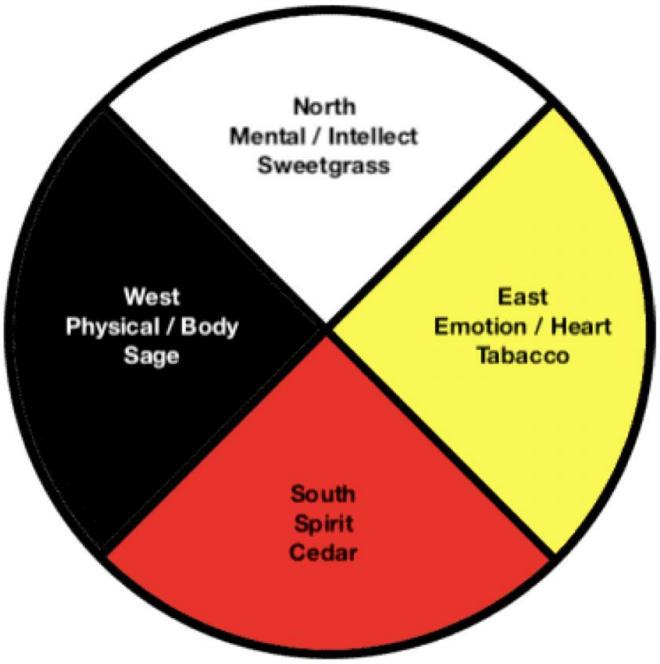

Figure 3: Example of a medicine wheel (Source: Wade Paul).

Considering Indigenous conceptions of wellness takes us to the use of medicine wheels: "Medicine Wheels are contemporary teaching tools that are used to explain concepts, philosophies and traditional teachings" (Linklater 2014:85). The medicine wheel, which is a traditional teaching tool, is used to illustrate the wholistic nature of health, and is comparable to the multi-faceted approach of epigenetics to health. While thrift gene has been used as a colonial explanation, the school programs studied in the next two sections are examples of culturally appropriate interventions.

Kahnawake School Diabetes Prevention Project

Kahnawake are a Haudenosaunee (Mohawk) community situated along the Saint Lawrence River across from Montreal. In 1985, it was observed that the rate of type 2 diabetes amongst adults of the community of Kahnawake was double that of the general public at the time (Tremblay et al. 2018). Community leaders quickly mobilized to develop and integrate a diabetes prevention program. The community invited experts and academic researchers to help develop the Kahnawake Schools Diabetes Prevention

Project (KSDPP); KSDPP is a community-based research project with high degree of participation and community ownership.

The project developed a health education program taught to children in grades one through six. The curriculum focused on healthy lifestyles including lessons on nutrition and physical fitness as well as diabetes (Paradis et al. 2005). KSDPP curriculum is ground in traditional Kanien'kehá:ka values that promote a wholistic approach to health and well-being that integrate physical, mental, emotional, and spiritual aspects of health (Tremblay et al. 2018). The project also included significant involvement in community events and the construction of a walking path (Paradis et al. 2005).

Sandy Lake School

Sandy Lake First Nations is a remote community in Northwestern Ontario. In the early 1990s, the Chief and Council of Sandy Lake First Nations consulted Dr. Stewart Harris, who at that time was the medical director of Sioux Lookout Zone, voicing their alarm concerning the growing prevalence of diabetes amongst the community. The joint partnership between the community and Dr. Harris resulted in the establishment of a community-based research project named the Sandy Lake Health and Diabetes Project (SLHDP).

The SLHDP makes frequent use of the radio station having a weekly community show as well as a 'Diabetes Kids' show. SLHDP adapted traditional teaching techniques to create a school based educational program. Storytelling is used to introduce and explain new and intricate concepts. Student learn alongside characters, Missy and Buddy Daaybway, while their activities teach them of the importance of a living a healthy lifestyle and preventing diabetes (Saksvig et al. 2005). In an effort to encourage increased physical activity, the SLHDP worked to develop a community wide walking path. Chief Sugar Daddy the SLHDP's well known mascot attends community events such as the Sand Lake's Muddy Water Music Festival to promote diabetes awareness and encourage healthy lifestyle choices (Kakekagumick et al. 2013).

Both the KSDPP and the SLHDP projects have faced challenges but are widely considered successful in mitigating the risk of diabetes in the respective communities. With regard to the KSDPP, follow up studies on the prevalence of diabetes have indicated a strong decline from 1986 to 1994. The rates of diabetes at Kahnawake are between the national and Indigenous averages, with both incidence rates and gender ratios being closer to the national average (Horn et al. 2007). The SLHDP is used as the standard template for developing community-based projects aimed at addressing Indigenous health concerns.

5.4 Conclusion

Canada's Indigenous population experiences a disproportionate burden of indicates of diabetes. In some demographics Indigenous diabetes rates are four times that of the national average. Colonial thought suggests that this is the result of a genetic predisposition by explaining it through the singular mechanisms of the thrift gene theory.

It is difficult to accurately measure the nutritional consumption of residential school children. However, there are ample survivor accounts that consistently testify to a diet that was inadequate in both quality and quantity and was severely limited. Archival documents have provided evidence to support survivors' claims. This chapter has studied how conditions in residential schools were comparable to the conditions of a prolonged famine.

Childhood malnutrition has been connected to a number of negative health outcomes. Amongst the many profound effects, research has linked malnutrition and increased risk of diabetes amongst survivors of the Dutch Famine. Research has suggested that exposed individuals share an epigenetic signature that increases the risk of developing diabetes.

It is abundantly clear that being exposed to sustained durations of caloric restrictions, like those experienced at residential schools, produce biological changes that increase the likelihood of poor health. Poor health is not a gene but an epigenetic profile. The high prevalence of diabetes in Indigenous people is not the result of a mismatch between a

thrifty gene and its current environment, but rather, indicative of epigenetic alterations that have become intergenerational, leading to negative health outcomes.

Chapter 6 : Conclusion

6 Introduction

I began this thesis by introducing epigenetics a new science that has received significant praise and accolades for its potential to be a transformative science. In the opening chapter I shared the following statement by McGowan and Szyf, “Epigenetics could serve as a bridge between the social sciences and the biological sciences, allowing a truly integrated understanding of human health and behavior” (2010:71). Similar optimism and praise for the evolving science’s revolutionary inter disciplinary potential has been echoed by other advocates such as Landecker and Panofsky who claim, “…with the opening up of various aspects of life and biomedical science to a consideration of the social and cultural world, there is a sense of new potential for collaboration and crossover, perhaps constituting a door in the sometimes obdurate wall between the life and social sciences” (2013:334). Statements like these highlight the intersectional nature of epigenetics, wherein increased dialogue between the natural and social sciences is not only encouraged, it is necessary.

My thesis fully embraces the transformative potential of epigenetics and further expands upon its revolutionary potential as I present the specific argument that epigenetics is a decolonizing science. I extend the same transformative potential to cross disciplinary lines, that has been the cause of so much optimism surrounding epigenetics, explicitly claiming that the same is possible cross-culturally.

I present decolonization as the intelligent, calculated and active resistance of the forces of colonialism. My approach to decolonize draws upon key figures such as Linda Tuhiwai Smith, who encourage resistance but not total rejection. In developing my understanding of decolonization, I lean upon Two-eyed seeing as the foundation of my theoretical construction of decolonization. At its core, two-eyed seeing promotes multiplicity in thought by embracing an approach that views the world from a variety of perspectives. As a decolonizing approach two-eyed seeing integrates Indigenous and Western knowledges and perspectives.

Through the five-chapters of my thesis I provide a study of the relevance of epigenetics across disciplines, including anthropology, Indigenous studies, history of science, and science and technology studies. The key theoretical constructs in this thesis include epigenetics, Two-Eyed seeing, and decolonizing science. Methodologies central to the thesis include secondary historical analysis and genealogy *as* decolonization.

The initial chapters of my thesis focus on a genealogical analysis of the concepts of genes, genomics and the emergence of epigenetics. My genealogical analysis seeks to actively demonstrate the resistance and unsettling process that is central to the methodologies of decolonization. By tracing relevant scientific conclusions from the seventeenth through the twentieth centuries with the application of Two-Eyed seeing, my research presents previous scientific conclusions in a new light and repositions a new understanding of the current science of epigenetics. The final two chapters provide case studies which allow a reframing of epidemiological understandings of health beyond simplistic notions of genetic vulnerability. Although not explicitly expressed, my choice of case studies were carefully considered to highlight the racialized biases of colonial attitudes towards Indigenous peoples.

I have shown how epigenetics is a way of understanding not just the health experiences of vulnerable populations, but is a particularly useful construct in providing a decolonizing explanation for the prevalence of certain health conditions (such as diabetes) in Canada's Indigenous communities.

6.1 Future Directions

In the following sections, I turn towards future implications and uses of this work as I look to highlight and illustrate how epigenetics can be used as an active force in order to develop a decolonized approach to health and well-being. I decided to separate this section into segments, making the designation between epigenetics the science and epigenetics as a decolonizing science.

6.1.1 Epigenetics as an Unsettling Science

As a science, in my view epigenetics is primed to progress towards the integration of human test subjects. Practical applications of epigenetics as a science include:

1) moving towards community-specific and -driven health interventions
2) sampling and the development of biobanks
3) targeted and specialized gene-based medical therapies

In moving to a model that involves human test subjects, the most immediate need is to grow and develop rich data sets. This is an endeavor that needs to be undertaken on a large scale, with centralized data collection at the national level. These data sets can be used to form and develop epigenetics-based health interventions and therapies.

In designing community-specific interventions, however, there is an ethical responsibility to actively work *with* communities rather than *for* them. Interventions must be designed only in collaboration with communities. The need for caution must also recognize the associated risks and pitfalls of the proximity for misuse and appropriation towards eugenics. Our current global trend towards hyper-surveillance is indicative of the looming danger of eugenics in the form of bio-surveillance. Possession of certain genes, for instance, though indicators of potential negative health outcomes, are not hard-lined diagnoses.

Through this thesis I have presented epigenetics as a decolonizing science. However, the truth of the matter is epigenetics is an unsettling science. Decolonization is only one example of the potential applications of epigenetics' unsettling and destabilizing potential. The perspective of epigenetics I presented is one that challenges standard narratives and seeks to establish new insights. This unsettling potential extends beyond the context of decolonization. The same potential to unsettle established understandings and recreate more inclusivity and diversity into understanding is true with other forms of study that resist a standard narrative, such as disability studies.

A central question that arose during my defense was about facts – I was asked by one of the examiners if I believed in there being such a thing as a scientific fact. Though there are tangible scientific facts, the practice of science is not free from biases. Recent cultural

and political movements reflect a resistance to biases protected by ‘scientific objectivity.’ Epigenetics can aid in developing new understandings of conditions from personal experiences with narrative knowledge, in conjunction with areas of study such as health humanities, movements like Mad Studies and so on. The process of challenging and questioning facts in itself is a useful way of destabilizing entrenched biases in scientific methods.

6.1.2 Epigenetics as a Decolonizing Science

While defending this thesis I was asked if one eye is bigger in my application of Albert and Murdena Marshall’s Two-Eyed Seeing. In the case of epigenetics as it is currently, I have to say yes, one eye is indeed bigger. The eye of Western science is the bigger eye. Personally, I do not see this being as particularly problematic. On the contrary, it is exactly this imbalance that is the source of my optimism in epigenetics. As I have already highlighted, those working in epigenetics have recognized its potential to strengthen and grow multicentric perspectives of health and human experience. In this sense the practitioners of epigenetics have acknowledged the imbalance. It is from here that I derive much of the optimism that epigenetics can be applied as a decolonizing science. While it is currently true that the eye of Western knowledge is bigger, it is a science that when harnessed for the purpose of decolonization seeks to expand the eye of Indigenous knowledge.

So, yes, one eye is bigger. In the perspective of epigenetics being a decolonizing science that I have presented through this thesis, this imbalance is embraced. The true decolonizing potential of this epigenetics perspective is that it not only acknowledges the imbalance, it does so in a manner that makes room. In creating space, I am optimistic this perspective of epigenetics can be used to develop a greater balance between the two eyes. Through this perspective it’s not problematic that one is larger because it is out measured by its potential to aid the other in growing, thus moving towards a more balanced state.

Indigenous perspectives of health and well-being are multi-generational in the way that like a braid, they weave tight connections between generations. This generational connection is fully displayed in Indigenous conceptions of multi-generational trauma and

traditional teachings linking seven generations in either direction. Epigenetics also recognizes a deep and enmeshed connection between generations. Through this commonality it becomes possible to build an understanding of health and well-being that is coherent with Indigenous perspectives as well as Western ones.

An example of a project that could have benefited from such an approach is the work of anthropologist Christianne Stephens with the Anishinaabeg community of Walpole Island. Located on the banks of the St. Clair River, the community of Walpole Island First Nation (WIFN), is roughly an hour's drive south of Sarnia Ontario. Throughout the years, there have been numerous oil and chemical spills into the St Clair River from Sarnia's Chemical Valley (local term for a section of the town that is and was the location of numerous oil and chemical refineries). As a result of these numerous spills, the community has sought out research interventions.

Like the growing trend of moving towards community based participatory research models, research in WIFN has been community-oriented and -driven. Stephens is one researcher who has been welcomed by the community.

Stephens was welcomed to conduct research and "…explore perceptions of environmental health risks on the part of the Anishinaabeg (Ojibwe) living at the Walpole Island First Nation" (2009:1). Stephens's research attempts to gauge the level of divergence between Western and Indigenous perspectives on environmental health risk caused due to the spills. Approaching the community as an outsider trained in Western perspectives, yet in her attempt to understand the Indigenous perspective in this context, Stephens's approach is emblematic of two-eyed seeing.

A significant conclusion that Stephens arrives at through her critique of medical terms such as chemophobia and risk perception is that they are "…problematic due to theoretical, methodological and ethical considerations. The Western assumptions that are built into these terms make them inappropriate descriptors and frameworks for understanding Anishinaabeg experiences" (Stephens 2009:197)

This conclusion is on the cusp of an epigenetics approach, which I have advocated throughout my thesis, because it acknowledges how the particularity of the Indigenous experience on Walpole Island does not fit these Western medical terms. This implies a need to develop Indigenous terms, understandings and perspectives that are congruent with Indigenous experience. This is where an epigenetic approach can help bridge the divide. Studies like this could benefit from an epigenetic approach by providing the tools to apply quantitative measures to social experience. Quantitative measures and the like here can be used to help formulate Indigenous experiences and perspectives in a way that translates Indigenous concepts to those from Western ideologies to bridge those gaps. This can bring the social sciences and sciences, as well as Indigenous and Western perspectives to the same meeting space, giving everybody an equal voice.

6.2 Concluding Remarks

I choose to conclude this thesis with the image which will follow these closing remarks. In chapter five, I shared an example of a medicine wheel. I explain that the medicine wheel is used as a teaching tool to illustrate the wholistic nature of health and well-being. I conclude by sharing a second medicine wheel, which I call the epigenetic medicine wheel. I view this epigenetic medicine wheel as the visual representation of the argument I have made through this thesis.

To summarize bridging the divide between the natural and social sciences, when applied as a decolonizing science, epigenetics can be used to develop integrated perspectives and methodologies. From the perspective of a natural scientist, an epigenetic approach embraces multiplicity and loosening the rigor of the scientific method. From the perspective of a social scientist, social experiences are made available to quantifiable measures and analytical research.

When applied as a decolonizing methodology for Two-Eyed seeing, an epigenetic approach integrates the strengths of both Western and Indigenous knowledges to create a hybridized perspective, as I have attempted to do in my project. This allows for the natural and social sciences, as well as Indigenous and Western perspectives to co-exist in the same meeting space, giving everybody an equal voice. Approaching specific issues

from a diversity of perspectives that are cross-disciplinary and cross cultural, epigenetics makes them mutually accessible for all parties.

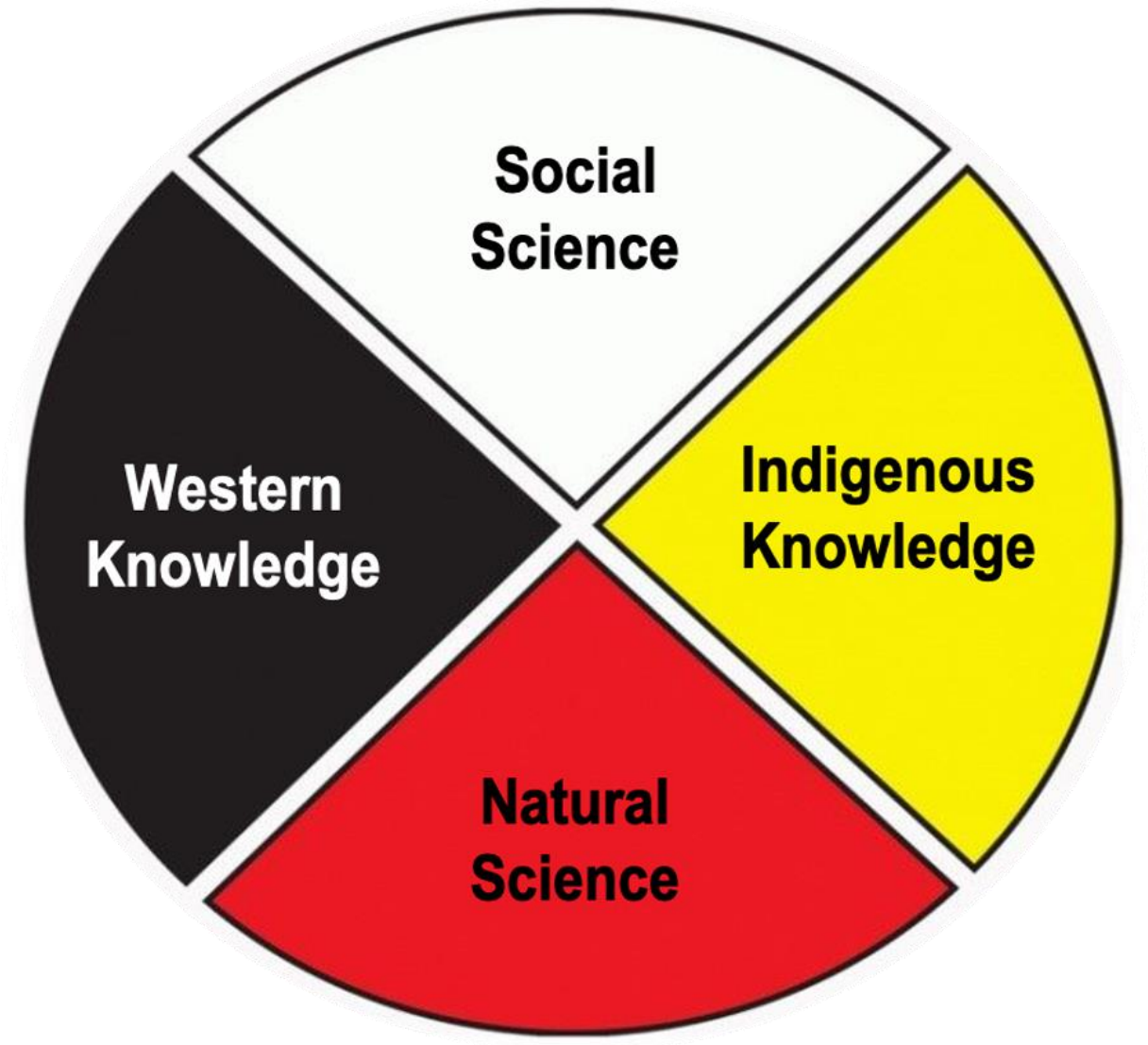

Figure 4: Epigenetic Medicine Wheel (Source: Wade Paul).

References

Adelson, Naomi 2000 'Being Alive Well' : Health and the Politics of Cree Well-Being. Toronto: University of Toronto Press.

Barker, D.J.P., and C. Osmond 1986 INFANT MORTALITY, CHILDHOOD NUTRITION, AND ISCHAEMIC HEART DISEASE IN ENGLAND AND WALES. The Lancet 327(8489). Elsevier: 1077–1081. https://www.sciencedirect.com/science/article/pii/S0140673686913401, accessed July 8, 2019.

Barker, D J, K. M. Godfrey, P. D. Gluckman, et al. 1993 Fetal Nutrition and Cardiovascular Disease in Adult Life. The Lancet 341(8850): 938–941.

Bhullar, Bhart Anjan S., Zachary S. Morris, Elizabeth M. Sefton, et al. 2015 A Molecular Mechanism for the Origin of a Key Evolutionary Innovation, the Bird Beak and Palate, Revealed by an Integrative Approach to Major Transitions in Vertebrate History. Evolution 69(7): 1665–1677.

Blossey, Ralf 2017 Chromatin: Structure, Dynamics, Regulation. 1st Editio. New York: Chapman and Hall/ CRC Press.

Bombay, Amy, Kimberly Matheson, and Hymie Anisman 2014 The Intergenerational Effects of Indian Residential Schools: Implications for the Concept of Historical Trauma. Transcultural Psychiatry 51(3): 320–338. http://journals.sagepub.com/doi/10.1177/1363461513503380.

Bouchard, Luigi, Stéphanie Thibault, Simon Pierre Guay, et al. 2010 Leptin Gene Epigenetic Adaptation to Impaired Glucose Metabolism during Pregnancy. Diabetes Care 33(11): 2436–2441.

Brasfield, Charles 2001 RESIDENTIAL SCHOOL SYNDROME. BC Medical Journal 43(2): 78–81.

Chung, Emma, John Cromby, Dimitris Papadopoulos, and Cristina Tufarelli 2016 Social Epigenetics: A Science of Social Science? The Sociological Review Monographs 64(1): 168–185. http://journals.sagepub.com/doi/full/10.1111/2059-7932.12019.

Cobb, Matthew 2006 Heredity before Genetics: A History. Nature Reviews Genetics 7(12): 953–958.

Collins, Francis S. 1999 Medical and Societal Consequences of the Human Genome Project. New England Journal of Medicine 341(1): 28–37. http://www.nejm.org/doi/abs/10.1056/NEJM199907013410106.

Cook, Deborah 2018 Adorno, Foucault and the Critique of the West. Brooklyn, NY: Verso.

Darnell, Regna 2018 Franz Boas's Legacy of "Useful Knowledge": The APS Archives and the Future of Americanist Anthropology. Proceedings of the American Philosophical Society Held at Philadelphia for Promoting Useful Knowledge 162(1): 1–15.

Erler, Jochen, Ruihan Zhang, Loukas Petridis, et al. 2014 The Role of Histone Tails in the Nucleosome: A Computational Study. Biophysical Journal 107(12): 2911–2922.

Falk, Raphael 2009 Genetic Analysis : A History of Genetic Thinking. New York: Cambridge University Press.

Fara, Patricia 2009 Science: A Four Thousand Year History. New York: Oxford University Press.

Gaudry, Adam, and Danielle Lorenz 2018 Indigenization as Inclusion, Reconciliation, and Decolonization: Navigating the Different Visions for Indigenizing the Canadian Academy. AlterNative 14(3): 218–227.

Gillman, Matthew W., David Barker, Dennis Bier, et al. 2007 Meeting Report on the 3 Rd International Congress on Developmental Origins of Health and Disease (DOHaD). Pediatric Research 61(5): 625–629.

Gould, Stephen Jay 1996 The Mismeasure of Man (Revised & Expanded). Penguin Books.

Gustafsson, Karl 2019 Chinese Collective Memory on the Internet : Remembering the Great Famine in Online Encyclopaedias. Memory Studies 12(2): 184–197.

Harris, Matthew P., Sean M. Hasso, Mark W.J. Ferguson, and John F. Fallon 2006 The Development of Archosaurian First-Generation Teeth in a Chicken Mutant. Current Biology 16(4): 371–377.

Harris, Stewart B., Joel Gittelsohn, Anthony Hanley, et al. 1997 The Prevalence of NIDDM and Associated Risk Factors in Native Canadians. Diabetes Care 20(2): 185–187.

Hart, Michael A., Silvia Straka, and Gladys Rowe 2017 Working Across Contexts: Practical Considerations of Doing Indigenist/Anti-Colonial Research. Qualitative Inquiry 23(5): 332–342.

Heijmans, Bastiaan T, Elmar W Tobi, L H Lumey, and P Eline Slagboom 2009 The Epigenome: Archive of the Prenatal Environment. Epigenetics 4(8): 526–531.

Heijmans, Bastiaan T, Elmar W Tobi, Aryeh D Stein, et al. 2008 Persistent Epigenetic Differences Associated with Prenatal Exposure to Famine in Humans. Proceedings of the National Academy of Sciences of the United States of America 105(40): 17046–17049.

Heindel, Jerrold J., John Balbus, Linda Birnbaum, et al. 2015 Developmental Origins of Health and Disease: Integrating Environmental Influences. Endocrinology 156(10): 3416–3421.

Heindel, Jerrold J., and Laura N. Vandenberg 2015 Developmental Origins of Health and Disease: A Paradigm for Understanding Disease Cause and Prevention. Current Opinion in Pediatrics 27(2): 248–253.

Hessmann, Elisabeth, Steven A. Johnsen, Jens T. Siveke, and Volker Ellenrieder 2017 Epigenetic Treatment of Pancreatic Cancer: Is There a Therapeutic

Perspective on the Horizon? Gut 66(1): 168–179.

Hochberg, Z., R. Feil, M. Constancia, et al. 2011 Child Health, Developmental Plasticity, and Epigenetic Programming. Endocrine Reviews 32(2): 159–224.

Holder, Lawrence B., M. Muksitul Haque, and Michael K. Skinner 2017 Machine Learning for Epigenetics and Future Medical Applications. Epigenetics 12(7). Taylor & Francis: 505–514. https://doi.org/10.1080/15592294.2017.1329068.

Horn, Ojistoh, Amanda Bruegl, Heather Jacobs-Whyte, et al. 2007 Incidence and Prevalence of Type 2 Diabetes in the First Nation Community of Kahnawá:Ke, Quebec, Canada, 1986–2003. Canadian Journal of Public Health 98(6): 438–443. https://search-proquest-com.ezproxy.library.uvic.ca/docview/232007904/fulltextPDF/959C5E660A704A94PQ/1?accountid=14846.

Howard, Heather A. 2014 Canadian Residential Schools and Urban Indigenous Knowledge Production about Diabetes. Medical Anthropology 33(6). Routledge: 529–545. http://dx.doi.org/10.1080/01459740.2013.828722.

Jacob, François 1974 The Logic of Life; a History of Heredity. Translated by Betty E. Spillmann. 1st Americ. New York: Pantheon Books.

Kahn, H. S., M. Graff, A. D. Stein, et al. 2008 A Fingerprint Characteristic Associated with the Early Prenatal Environment. American Journal of Human Biology 65(1): 59–65.

Kahn, Henry S, Mariaelisa Graff, Aryeh D Stein, and L H Lumey 2009 A Fingerprint Marker from Early Gestation Associated with Diabetes in Middle Age : The Dutch Hunger Winter Families Study. International Journal of Epidemiology 38(1): 101–109.

Kakekagumick, Kara E., Mariam Naqshbandi Hayward, Stewart B. Harris, et al. 2013 Sandy Lake Health and Diabetes Project: A Community-Based Intervention Targeting Type 2 Diabetes and Its Risk Factors in a First Nations Community. Frontiers in Endocrinology 4(170): 1–9.

Keller, Evelyn Fox 1995 Refiguring Life: Metaphors of Twentieth-Century Biology. The Wellek Library Lectures Series at the University of California, Irvine.

2000 The Century of the Gene. Cambridge, Mass: Harvard University Press.

2005 The Century beyond the Gene. *In* Journal of Biosciences.

2014 From Gene Action to Reactive Genomes. Journal of Physiology 592(11): 2423–2429.

Landecker, Hannah, and Aaron Panofsky 2013 From Social Structure to Gene Regulation, and Back: A Critical Introduction to Environmental Epigenetics for Sociology. Annual Review of Sociology 39(1): 333–357.

Linklater, Renee 2014 Decolonizing Trauma Work Indigenous Stories and Strategies. Fernwood Publishing.

Lock, Margaret, and Vinh-Kim Nguyen 2010 An Anthropology of Biomedicine.

First. Chichester, West Sussex, U.K. ; Malden, MA: Wiley-Blackwell.

Lumey, L H, Aryeh D Stein, Henry S Kahn, et al. 2007 Cohort Profile : The Dutch Hunger Winter Families Study. International Journal of Epidemiology 36(6): 1196–1204.

Lumey, L H, Aryeh D Stein, and Ezra Susser 2011 Prenatal Famine and Adult Health. Annual Review of Public Health 3(1): 237–262.

Martin, D 2012 Two-Eyed Seeing: A Framework for Understanding Indigenous and Non-Indigneous Approches to Indigenous Health Research. Canadian Journal of Nursing Research 44(2): 20–42.

McGowan, Patrick O., Aya Sasaki, Ana C. D'Alessio, et al. 2009 Epigenetic Regulation of the Glucocorticoid Receptor in Human Brain Associates with Childhood Abuse. Nature Neuroscience 12(342–348).

McGowan, Patrick O., and Moshe Szyf 2010 The Epigenetics of Social Adversity in Early Life: Implications for Mental Health Outcomes. Neurobiology of Disease 39(1). Elsevier Inc.: 66–72. http://dx.doi.org/10.1016/j.nbd.2009.12.026.

Meaney, Michael J. 2010 Epigenetics and the Biological Definition of Gene X Environment Interactions. Child Development 81(1): 41–79.

Milloy, John S. 1999 A National Crime: The Canadian Government and the Residential School System, 1879 to 1986. Winnipeg, Manitoba: The Univeristy of Manitoba Press.

Morgentaler, Goldie 2000 Dickens and Heredity : When like Begets Like. Basingstoke, Hampshire England: Macmillan Press LTD.

Mosby, Ian 2013 Administering Colonial Science: Nutrition Research and Human Biomedical Experimentation in Aboriginal Communities and Residential Schools, 1942-1952. Histoire Sociale / Social History 46(91): 145–172.

Murray, Karen Bridget 2018 EPIGENETICS AND POLITICS IN THE COLONIAL PRESENT. Canadian Journal of Sociology (Online); Toronto 43(4): 349–388. https://www-lib-uwo-ca.proxy1.lib.uwo.ca/cgi-bin/ezpauthn.cgi?url=http://search.proquest.com.proxy1.lib.uwo.ca/docview/2169170343?accountid=15115%0A.

Neel, James 1999 Diabetes Mellitus: A “Thrifty” Genotype Rendered Detrimental by “Progress”? Bulletin of the World Health Organizationn 77(8): 694–703.

O’Sullivan, John 2011 Sustainability, Seventh Generation. Green Ethics and Philosophy: An A-to-Z Guide. SAGE Publications, Inc.

Paradis, Gilles, Lucie Levesque, Ann C. Macaulay, et al. 2005 Impact of a Diabetes Prevention Program on Body Size, Physical Activity, and Diet among Kanien’keha:Ka (Mohawk) Children 6 to 11 Years Old: 8-Year Results from the Kahnawake Schools Diabetes Prevention Project. Pediatrics 115(2): 333–339.

Piovesan, Allison, Maria Chiara Pelleri, Francesca Antonaros, et al. 2019 On the Length, Weight and GC Content of the Human Genome. BMC Research Notes

12(1). BioMed Central: 1–7. https://doi.org/10.1186/s13104-019-4137-z.

Radpour, Ramin, Zeinab Barekati, Corina Kohler, et al. 2011 Integrated Epigenetics of Human Breast Cancer: Synoptic Investigation of Targeted Genes, MicroRNAs and Proteins upon Demethylation Treatment. PLoS ONE 6(11).

Ramutsindela, Maano 2018 Environment. *In* Critical Terms For the Study of Africa. Gaurav Desai and Adeline Masquelier, eds. Pp. 102–114. Chicago: The Univeristy of Chicago Press.

Ravelli, Gian-Paolo, Zena A. Stein, and Mervyn W. Susser 1976 Obesity in Young Men after Famine Exposure in Utero and Early Infancy. New England Journal of Medicine 295(7): 349–353. http://www.nejm.org/doi/abs/10.1056/NEJM197608122950701.

Rodríguez-Paredes, Manuel, and Manel Esteller 2011 Cancer Epigenetics Reaches Mainstream Oncology. Nature Medicine 17(3): 330–339.

Rose, Nikolas 2007 He Politics of Life Itself : Biomedicine, Power, and Subjectivity in the Twenty-First Century. Princeton : Princeton University Press.

Roseboom, Tessa J., Jan H.P. Van der Meulen, Anita C.J. Ravelli, et al. 2001 Effects of Prenatal Exposure to the Dutch Famine and Disease in Later Life: An Overview. Molecular and Cellular Endocrinology 185(1): 93–98. http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L40949554%5Cnhttp://dx.doi.org/10.1016/j.reprotox.2005.04.005%5Cnhttp://sfxhosted.exlibrisgroup.com/dal?sid=EMBASE&issn=08906238&id=doi:10.1016/j.reprotox.2005.04.005&atitle=Prenatal.

Roseboom, Tessa J, Rebecca C Painter, Annet F M Van Abeelen, Marjolein V E Veenendaal, and Susanne R De Rooij 2011 Hungry in the Womb : What Are the Consequences ? Lessons from the Dutch Famine. Maturitas 70(2): 141–145.

Roseboom, Tessa, Susanne de Rooij, and Rebecca Painter 2006 The Dutch Famine and Its Long-Term Consequences for Adult Health. Early Human Development 82(8): 485–491.

Sahlins, Marshall, and David Graeber 2017 Stone Age Economics. Stone Age Economics. New York: Routledge.

Saksvig, Brit I., Joel Gittelsohn, Stewart B. Harris, et al. 2005 A Pilot School-Based Healthy Eating and Physical Activity Intervention Improves Diet, Food Knowledge, and Self-Efficacy for Native Canadian Children. The Journal of Nutrition 135(10): 2392–2398.

Scholte, Robert S, Gerard J van den Berg, and Maarten Lindeboom 2010 The Long-Run Effects of Birth in the Dutch Hunger Winter on Income and Hospitalization. University OfMimeo. 39: 17–30.

Schulz, Laura C. 2010 The Dutch Hunger Winter and the Developmental Origins of Health and Disease. Proceedings of the National Academy of Sciences 107(39). National Academy of Sciences: 16757–16758. https://www.pnas.org/content/107/39/16757, accessed June 19, 2019.

Skinner, Michael K. 2011 Environmental Epigenetic Transgenerational Inheritance and Somatic Epigenetic Mitotic Stability. Epigenetics 6(7): 838–842.

2015 Endocrine Disruptors in 2015: Epigenetic Transgenerational Inheritance. Nature Reviews Endocrinology 12(2). Nature Publishing Group: 68–70. http://www.nature.com/doifinder/10.1038/nrendo.2015.206.

Skinner, Michael K 2014 Environmental Stress and Epigenetic Transgenerational Inheritance. BMC Medicine 12(1): 153. http://bmcmedicine.biomedcentral.com/articles/10.1186/s12916-014-0153-y.

Smith, Linda Tuhiwai 1999 Decolonizing Methodologies: Research and Indigenous Peoples. New York. New York: Zed Books Ltd.

Soubry, A., S. K. Murphy, Z. Huang, et al. 2011 The Effects of Depression and Use of Antidepressive Medicines during Pregnancy on the Methylation Status of the IGF2 Imprinted Control Regions in the Offspring. Clinical Epigenetics 3(1).

Stein, Zena, and Mervyn Susser 1975a The Dutch Famine, 1944-1945, and the Reproductive Process. I. Effects on Six Indices at Birth. Pediatric Research 9(2): 70–76.

1975b The Dutch Famine, 1944-1945, and the Reproductive Process. II. Interrelations of Caloric Rations and Six Indices at Birth. Pediatric Research 9(2): 76–83.

Stein, Zena, Mervyn Susser, Gerhart Saenger, and Francis Marolla 1972 Nutrition and Mental Performance. Science 178(4062): 708–713.

Susser, Ezra, Richard Neugebauer, Hans W Hoek, et al. 1996 Schizophrenia after Prenatal Famine. Further Evidence. Arch. Gen. Psychiatry 53(1): 25–31.

The Survivors Speak: A Report of the Truth and Reconciliation Commission of Canada 2015. Ottawa.

Tobi, Elmar W, Jelle J Goeman, Ramin Monajemi, et al. 2014 DNA Methylation Signatures Link Prenatal Famine Exposure to Growth and Metabolism. NATURE COMMUNICATIONS 5(1): 5592.

Tremblay, Marie Claude, Debbie H. Martin, Alex M. McComber, Amelia McGregor, and Ann C. Macaulay 2018 Understanding Community-Based Participatory Research through a Social Movement Framework: A Case Study of the Kahnawake Schools Diabetes Prevention Project. BMC Public Health 18(1). BMC Public Health: 1–17.

Ueberheide, Beatrix M., and Sahana Mollah 2007 Deciphering the Histone Code Using Mass Spectrometry. International Journal of Mass Spectrometry 259(1–3): 46–56.

Wadhwa, Pathik D., Claudia Buss, Sonja Entringer, and James M. Swanson 2009 Developmental Origins of Health and Disease: Brief History of the Approach and Current Focus on Epigenetic Mechanisms. Semin Reprod Med. 27(5): 358–368. http://dialnet.unirioja.es/servlet/articulo?codigo=4947641&info=resumen&idioma=ENG.

Weaver, Ian C G, Nadia Cervoni, Frances A. Champagne, et al. 2004 Epigenetic Programming by Maternal Behavior. Nature Neuroscience 7(8): 847–854.

Wolfe, Patrick 2006 Settler Colonialism and the Elimination of the Native.

Journal of Genocide Research 8(4): 387–409.

Young, T. Kue, Patricia J Martens, Shayne P Taback, et al. 2002 Type 2 Diabetes Mellitus in Children: Prenatal and Early Infancy Risk Factors Among Native Canadians. Archives of Pediatrics & Adolescent Medicine 156(7): 651–655.

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